



CHILDHOOD TRAUMA AND MENTAL HEALTH OUTCOMES IN ADULTHOOD: A LITERATURE REVIEW OF NEUROBIOLOGICAL AND PSYCHOLOGICAL MECHANISMS

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ABSTRACT

Childhood trauma represents one of the most significant public health challenges of our time, with far-reaching consequences for mental health across the lifespan. This literature review examines the neurobiological and psychological mechanisms through which adverse childhood experiences (ACEs) contribute to mental health disorders in adulthood, including depression, anxiety, post-traumatic stress disorder (PTSD), and psychosis. Drawing on recent systematic reviews and meta-analyses from interdisciplinary fields including psychology, neuroscience, and psychiatry, this paper explores how early adversity disrupts hypothalamic-pituitary-adrenal (HPA) axis functioning, alters brain structure and neurochemistry, and establishes maladaptive psychological patterns. The review also considers moderating factors such as social support and resilience, and concludes with implications for prevention and treatment.

1. Introduction

Adverse childhood experiences (ACEs) — broadly defined as potentially traumatic events occurring before the age of 18, including physical, emotional, and sexual abuse, neglect, and household dysfunction — are among the most consequential risk factors for poor mental health across the lifespan [1]. Epidemiological data consistently indicate that such experiences are far from rare: more than half of adults report at least one ACE, and approximately a quarter report two or more [2]. The cumulative burden of these experiences has been linked to a striking range of mental health disorders in adulthood, including major depressive disorder, anxiety disorders, PTSD, psychotic disorders, borderline personality disorder, and substance use disorders.

The foundational ACE Study, conducted by Felitti and colleagues in the late 1990s, established the dose-response relationship between cumulative childhood adversity and negative health outcomes: the greater the number of ACEs experienced, the greater the risk of

adverse mental and physical health consequences in adulthood [3]. Subsequent decades of research have substantially expanded our understanding of the specific pathways — neurobiological, psychological, and social — through which these effects operate. This literature review aims to synthesize current knowledge on the mechanisms linking childhood trauma to adult mental health outcomes, with attention to both the biological embedding of early adversity and the psychological processes it sets in motion.

2. Defining Childhood Trauma and Its Scope

Childhood trauma encompasses a heterogeneous range of experiences that share the common feature of overwhelming the child's capacity for adaptive coping. The ACE framework, which remains the most widely used classificatory system in this field, identifies three overarching categories: child abuse (emotional, physical, and sexual), child neglect (emotional and physical), and household dysfunction (including parental substance abuse, domestic violence, incarcerated household members, and parental separation or divorce) [1]. More recent scholarship has expanded this framework to include community-level adversities such as exposure to violence, poverty, and discrimination, recognizing that the original ACE categories, while important, do not capture the full spectrum of potentially traumatic childhood experiences [4].

Importantly, research distinguishes between different types of maltreatment in terms of their specific mental health consequences. A systematic review and meta-analysis by Xiao and colleagues (2023), encompassing 90 studies, found that both childhood emotional abuse (CEA) and childhood emotional neglect (CEN) were significantly associated with depression, anxiety, PTSD, borderline personality disorder, and suicidal ideation in adulthood, even after controlling for other forms of maltreatment [5]. These findings underscore the particular importance of emotional dimensions of early adversity, which have historically received less research attention than physical or sexual abuse, despite being at least as prevalent and consequential.

3. Neurobiological Mechanisms

3.1 HPA Axis Dysregulation

The hypothalamic-pituitary-adrenal (HPA) axis is the body's primary neuroendocrine stress-response system. When a threat is perceived, the hypothalamus signals the pituitary gland, which in turn stimulates the adrenal cortex to release cortisol. In typically developing individuals, this system activates in response to genuine stressors and returns to baseline once the threat has passed. Childhood trauma, however, disrupts this regulatory architecture in lasting ways [6]. A comprehensive systematic review of 27 meta-analyses by Kuzminskaite and colleagues (2022) found that individuals with ACE histories demonstrate blunted cortisol reactivity to psychosocial stressors, low-grade systemic inflammation indicated by elevated C-reactive protein levels, and structural alterations in stress-sensitive brain regions — findings that were present irrespective of whether or not participants had a formal psychiatric diagnosis [7].

Longitudinal research provides important evidence for the mediating role of HPA dysregulation in translating ACEs into depressive psychopathology. A genetically-informed twin study published in *Translational Psychiatry* demonstrated that lower daytime cortisol levels explained between 10 and 20 percent of the total association between ACE exposure

and depressive symptoms in young adulthood, even after accounting for genetic liability [8]. Research using animal models has further clarified the cellular and molecular mechanisms involved: adverse early experiences, such as maternal separation, produce lasting alterations in the expression of glucocorticoid receptor genes through epigenetic mechanisms, reducing the sensitivity of the HPA axis feedback loop and creating a chronic state of dysregulated stress reactivity [6].

3.2 Brain Structure and Neural Circuit Alterations

Neuroimaging research has consistently demonstrated that childhood adversity is associated with structural and functional alterations in brain regions critical for emotional regulation, memory, and threat processing. The hippocampus — essential for contextual memory and the downregulation of HPA activity — shows reduced grey matter volume in individuals with ACE histories, while the amygdala, the brain's primary threat-detection structure, exhibits heightened reactivity to threatening stimuli [7]. A large clinical cohort study published in *Frontiers in Psychiatry* (2024) found that specific brain functional abnormalities mediated the relationship between ACE scores and the probability of psychiatric diagnosis, with dose-dependent effects: higher ACE scores were associated with progressively more pronounced neurological alterations [2].

The prefrontal cortex (PFC), which normally exerts regulatory control over limbic structures including the amygdala, also shows adverse structural changes following early trauma, with reduced volume and impaired connectivity associated with deficits in emotion regulation and impulse control. Disruptions of the amygdala-prefrontal cortex circuit may play a crucial role in the neurobiological etiology of emotional dysregulation in individuals with ACE histories [9]. The circuits responsible for the secretion of key neurotransmitters — including serotonin, dopamine, and norepinephrine — are organized in the early years of life in the context of caregiver relationships, and their disruption by adverse experiences lays the groundwork for mood and motivational dysregulation throughout the lifespan.

4. Psychological Mechanisms

Beyond the neurobiological pathways, childhood trauma shapes mental health through a range of psychological mechanisms. Disrupted attachment relationships — arising from inconsistent, frightening, or emotionally unavailable caregiving — establish internal working models of the self as unworthy and others as unreliable, creating a template for relational and emotional functioning that persists into adulthood. Research consistently identifies impaired emotional regulation as a central psychological consequence of early adversity: individuals with ACE histories demonstrate reduced capacity to modulate negative affect, increased emotional reactivity, and greater use of maladaptive coping strategies such as avoidance and dissociation [10].

Cognitive processes are also significantly affected. Early adversity shapes threat-appraisal systems, creating a bias toward interpreting ambiguous stimuli as dangerous — a pattern that is adaptive in genuinely threatening childhood environments but maladaptive in safer adult contexts. A study published in *PLOS ONE* (2025) examining stress appraisal as a mediating mechanism found that childhood trauma was associated with heightened perceived stress in response to daily life events, which in turn increased risk for depression, anxiety, and

suicidal ideation in adulthood [3]. These findings highlight the importance of cognitive-emotional pathways as targets for therapeutic intervention.

A meta-analysis and systematic review by McKay and colleagues (2021) in *Acta Psychiatrica Scandinavica*, which synthesized data from longitudinal cohort studies, confirmed robust prospective associations between childhood trauma and affective disorders (depression and bipolar disorder) as well as psychotic disorders in adulthood [11]. The study noted that associations were strongest for cumulative or severe trauma and for adversities occurring during sensitive periods of brain development. Importantly, these effects remained significant even after controlling for genetic confounders, supporting a direct developmental impact of adverse early experiences on mental health trajectories.

5. Moderating and Protective Factors

While the evidence for the deleterious effects of childhood trauma is compelling, it is equally important to recognize that not all individuals exposed to adversity develop mental health disorders. Individual differences in biological stress sensitivity, genetic factors, temperament, and the availability of social support all moderate the relationship between ACEs and mental health outcomes [12]. Social support — whether from peers, family members outside the abusive context, teachers, or community members — has been identified as one of the most robust protective factors against the mental health consequences of childhood adversity. A cross-sectional study from Bangladesh (2025) found that perceived social support and adaptive coping strategies significantly mediated the relationship between ACEs and stress-related symptoms among adolescents, even in a low-resource, high-adversity context [10].

Demographic factors also moderate the ACE-mental health relationship in important ways. A large population-based study published in *Global Mental Health* (2024) found that female sex, lower socioeconomic status, family disharmony, poor parental relationships, and unhealthy lifestyle factors each independently increased psychiatric risk among individuals with ACE histories [4]. Conversely, higher self-esteem, self-reliance, and social connectedness served as protective factors. These findings have important implications for the design of targeted prevention and intervention programs, particularly in populations with high rates of cumulative adversity.

6. Clinical and Public Health Implications

The convergent evidence reviewed here carries significant implications for clinical practice and public health policy. Trauma-informed care — an approach that recognizes the pervasive impact of trauma and integrates knowledge about trauma into all aspects of service delivery — is increasingly recognized as an essential framework for mental health treatment. Evidence-based therapeutic approaches such as trauma-focused cognitive behavioral therapy (TF-CBT), eye movement desensitization and reprocessing (EMDR), and dialectical behavior therapy (DBT) have demonstrated efficacy in addressing the psychological sequelae of childhood trauma [5]. At the biological level, emerging research on the reversibility of epigenetic modifications associated with early adversity opens promising avenues for pharmacological and behavioral interventions aimed at restoring HPA axis regulation.

At the public health level, the ACE framework underscores the importance of primary prevention — addressing the social determinants that give rise to childhood adversity in the

first place, including poverty, domestic violence, parental mental illness, and social isolation. A systematic review and meta-analysis by Thurston and colleagues (2025) in Trauma, Violence, & Abuse, which prospectively examined associations between ACEs and adult anxiety, depression, PTSD, psychotic experiences, self-harm, and suicidality across 62 studies from 15 countries, concluded that the evidence base strongly supports the integration of ACE screening into routine health care and the development of universal early intervention programs [1].

Conclusion

Childhood trauma exerts profound and enduring effects on mental health through multiple, interconnected neurobiological and psychological mechanisms. The disruption of HPA axis functioning, structural and functional alterations in stress-sensitive brain regions, impaired emotional regulation, maladaptive cognitive schemas, and disrupted attachment all contribute to elevated risk for depression, anxiety, PTSD, and other psychiatric conditions in adulthood. The dose-response relationship between cumulative adversity and mental health outcomes underscores the importance of preventing exposure to multiple ACEs and of providing early, trauma-informed support to those who have been exposed.

Equally important is the recognition that adversity does not determine destiny: social support, resilience, and effective therapeutic intervention can significantly moderate the mental health consequences of early trauma. Future research should continue to clarify the specific biological and psychological pathways linking particular types of adversity to particular mental health outcomes, with attention to culturally diverse populations and to the identification of modifiable protective factors. The integration of this growing evidence base into clinical practice, education, and social policy represents one of the most promising avenues for reducing the global burden of mental illness

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