

Review Article

The role of childhood trauma in the development of psychopathology via neurocognitive pathways

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ABSTRACT

Childhood trauma is a potent risk factor for a wide range of psychopathological outcomes. This review synthesizes evidence on how trauma experienced in childhood (before age 12) impacts neurocognitive development and in turn elevates risk for later psychopathology. A comprehensive literature search was conducted focusing on cognitive, behavioral, and neurobiological effects of early trauma, with special emphasis on Indian research alongside international findings. Childhood trauma is associated with broad cognitive difficulties, including attentional bias toward threat, memory processing impairments (e.g., fragmented or overgeneral memories), executive function deficits, and emotion regulation problems. These trauma-induced neurocognitive alterations often mediate the link between early adversity and diverse psychopathological outcomes such as posttraumatic stress, anxiety, depression, and dissociation. Neurobiologically, trauma-exposed children show dysregulation of stress response systems (e.g., HPA axis) and structural and functional brain changes in regions subserving emotion and cognition (e.g., amygdala, hippocampus, prefrontal cortex). Indian studies mirror global findings, with high prevalence of child trauma and evidence of similar neurocognitive sequelae. Childhood trauma can derail neurocognitive development in ways that confer transdiagnostic psychopathology risk. These findings underscore the need for early identification and intervention for trauma-exposed children, culturally informed research (particularly in India), and dimensional frameworks (e.g., RDoC) to understand trauma-related cognitive profiles. Early interventions targeting cognitive and emotional regulation skills may mitigate the progression from childhood trauma to later mental health disorders.

Keywords: Childhood trauma, Neurocognitive pathways, Psychopathology, Emotion regulation, Executive function, India

INTRODUCTION

Childhood trauma-encompassing abuse, neglect, violence, and other adverse experiences before age 12-is a widespread public health concern with far-reaching developmental consequences. A substantial body of research links early trauma exposure to increased risk for virtually all forms of psychopathology, including posttraumatic stress disorder (PTSD), anxiety disorders, depression, behavior disorders, and dissociative disorders. Not all children who experience trauma develop mental illness; however, trauma markedly elevates the likelihood

of maladaptive outcomes via its impact on developing brain and cognitive systems. It is now recognized that childhood trauma has transdiagnostic effects, meaning it confers vulnerability across many diagnostic categories rather than a single disorder. This review focuses on the neurocognitive pathways by which early trauma may lead to later psychopathology, synthesizing cognitive, behavioral, and neurobiological evidence. We explore how trauma-related cognitive dysfunctions-such as attentional biases, memory processing abnormalities, executive function deficits, and emotion dysregulation-mediate or modulate the association between childhood trauma and psychopathological outcomes.¹

Importantly, this review centers on children in pre-adolescent stages (approximately up to age 12), a period of rapid neurocognitive development and heightened sensitivity to environmental influences. We integrate findings from international research with data from India to highlight cultural contexts and regional research progress. India has one of the world's largest child populations, with studies indicating a high prevalence of childhood adversities. Yet, child trauma remains under-recognized in many settings, and research on neuropsychological impacts in Indian contexts is still emerging. By bringing together evidence from Western and Indian literatures, we aim to provide a comprehensive and culturally relevant understanding of how childhood trauma can alter neurocognitive development and thereby shape mental health trajectories.^{2,3}

LITERATURE REVIEW

We conducted a narrative literature review to capture a broad range of studies on childhood trauma, cognition, and psychopathology. Sources were identified through searches of databases including PubMed, PsycINFO, and Google Scholar for articles published up to 2025. Key search terms included combinations of "childhood trauma," "maltreatment," "child abuse," "cognitive function," "attention bias," "memory," "executive function," "emotion regulation," "psychopathology," "PTSD," "depression," "anxiety," "dissociation," "neuroimaging," "HPA axis," and "RDoC." Particular effort was made to include Indian research: we reviewed publications from Indian journals and reports from Indian institutions. Inclusion criteria emphasized studies of children (approximately 0–12 years old) or findings relevant to that age range, as well as review articles and theoretical papers on developmental trauma. Given the ethical and practical constraints of experimental trauma research in children, most sources were observational (cross-sectional or longitudinal studies) and clinical studies, complemented by some experimental paradigms (e.g., cognitive task performance) and neurobiological assessments.

Our goal was not to perform a formal meta-analysis but rather to integrate findings across methodologies (behavioral testing, neuropsychological assessments, neuroimaging, psychophysiology, etc.) and across disciplines (psychology, psychiatry, neuroscience, developmental psychopathology). We paid special attention to evidence of mediation, where a cognitive or neural variable explains how trauma leads to psychopathology, and moderation, such as the timing or type of trauma influencing outcomes. The review also incorporates conceptual frameworks like the RDoC to interpret the findings dimensionally. Throughout, we highlight points of convergence in the literature, note inconsistencies or gaps (especially where child-specific or India-specific data are lacking), and identify directions for future research.

CHILDHOOD TRAUMA AND PSYCHOPATHOLOGY: SCOPE OF THE PROBLEM

Traumatic experiences in childhood are unfortunately common worldwide. Globally, about 70% of people report at least one traumatic event over the lifespan, and a significant portion of these occur in childhood. In the United States, roughly 60% of adults recall at least one childhood trauma, with about one in four reporting multiple adverse events. In India, estimates suggest extremely high rates of child maltreatment. A landmark national survey found that approximately half of Indian children experienced some form of abuse—for example, 53% reported sexual abuse or assault, and a similar percentage reported physical abuse by caregivers or authority figures (Ministry of women and child development [MWCD], 2007). Children in socioeconomically disadvantaged settings (e.g., urban slums) face compounding adversities, though abuse also occurs across higher-income families. These sobering statistics underscore that childhood trauma is a prevalent public health issue globally and in India.³

Globally, nearly 70% of individuals report at least one traumatic event, with many occurring in childhood. In the U.S., about 60% of adults recall childhood trauma, while India reports even higher prevalence—69% of children experienced physical abuse and 53% sexual abuse. These rates highlight trauma as a major public health issue. Trauma predicts a wide spectrum of outcomes. PTSD is a well-established consequence, with children at higher risk than adults. Early adversity also predicts depression, marked by earlier onset and treatment resistance, as well as anxiety disorders, behavioral dysregulation, and dissociation. Importantly, trauma often leads to multifinality (one cause, multiple outcomes) and comorbidity, where children present with overlapping disorders. Attachment disruption is also common when trauma involves caregivers. Children may develop insecure attachment, interpersonal difficulties, and relational distrust. Yet, resilience is possible, especially with protective factors like supportive caregivers, therapy, and community resources.^{1,3}

NEUROCOGNITIVE CONSEQUENCES OF CHILDHOOD TRAUMA

Trauma during early childhood coincides with critical periods of brain maturation and cognitive development. Severe or chronic stress can disrupt the maturation of neural circuits, leading to measurable deficits in cognitive functions. Research has documented that children with histories of maltreatment or complex trauma often perform worse on a variety of cognitive tasks than non-traumatized peers in one meta-analytic review of longitudinal studies, children exposed to complex interpersonal trauma showed significantly poorer overall cognitive functioning (a global composite of abilities) compared to non-traumatized children. Notably, trauma that began earlier in childhood

and more recent trauma exposure were associated with the greatest cognitive impairments. This suggests that the timing and chronicity of trauma matter: the developing brain is especially vulnerable to insults in early life, and the effects of trauma on cognition can appear quickly after exposure. Below, we break down the neurocognitive sequelae of childhood trauma into specific domains.⁴

ATTENTION AND THREAT PERCEPTION

Trauma-exposed children often exhibit alterations in attentional processing, especially in how they direct attention to threatening or emotional information. A frequently observed pattern is an attention bias toward threat. This means that children with trauma histories are hypervigilant and quick to notice potential dangers in their environment. For example, studies using the dot-probe or visual-search tasks have found that maltreated children disproportionately direct their gaze toward angry or fearful faces compared to non-maltreated children. Such children may perceive neutral social cues as threatening and have difficulty disengaging from negative stimuli. This attentional hypervigilance is thought to be an adaptive response in dangerous environments-by remaining on high alert, the child may better detect and avoid threats. However, in safe environments (like a classroom), this bias can become maladaptive, contributing to anxiety and distraction.⁵

MEMORY AND COGNITIVE PROCESSING OF TRAUMA

Memory is another cognitive domain profoundly affected by early trauma. Hippocampal-dependent memory processes in particular are susceptible to stress hormones and trauma-related neurobiological changes. Children who experience trauma often exhibit problems with both autobiographical memory and working memory. One well-documented phenomenon is overgeneral autobiographical memory-a tendency to recall past events in broad, nonspecific terms rather than with precise details. Research indeed shows that individuals with childhood abuse or neglect often have impaired autobiographical memory specificity, retrieving fewer detailed personal memories, and this correlates with emotional-regulation problems. In one study, childhood trauma was linked to blunted basal cortisol levels and autobiographical memory deficits in the form of overgeneral memories-suggesting a biological correlation (hypocortisolism) of this cognitive symptom.^{4,6}

EXECUTIVE FUNCTION AND COGNITIVE CONTROL

Executive functions (EF) are high-level cognitive skills that include inhibitory control, cognitive flexibility, planning, and working memory (often considered part of EF). These functions depend heavily on the prefrontal cortex, which undergoes protracted development in childhood. Traumatic stress, especially when prolonged,

can disrupt the maturation of prefrontal circuits, resulting in executive dysfunction. A growing literature indicates that trauma-exposed youth have measurable deficits in EF compared to their peers.

Specific executive domains affected by trauma include

EF-including inhibitory control, cognitive flexibility, planning, and working memory-rely on the prefrontal cortex, which develops throughout childhood. Traumatic stress can disrupt prefrontal maturation, resulting in measurable deficits. Trauma-exposed children often struggle with inhibitory control, showing increased impulsive errors on tasks like go/no-go or Stroop tests. Cognitive flexibility and planning are impaired, affecting task-switching, adapting to new rules, and multi-step problem solving. Working memory deficits are common; an Indian study of over 500 adults found higher ACE scores predicted poorer working memory and global cognition. Attention regulation is also compromised, with children easily distractible and unable to sustain focus due to hyperarousal.^{3,7}

These executive deficits contribute to academic underachievement, difficulty following rules, poor decision-making, and risk-taking behavior. Neuroimaging shows trauma-exposed youth often fail to engage prefrontal regions during tasks requiring emotional regulation or conflict resolution, explaining difficulties in managing cognitive control alongside emotional processing.^{3,8}

EMOTION REGULATION AND DYSREGULATION

Emotion regulation refers to the processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions. Childhood trauma can disrupt emotion regulation, leading to maladaptive strategies, reduced adaptive strategies, and emotion impulsivity. Children may show hyperarousal (linked to anxiety/PTSD) or hypoarousal (linked to depression/dissociation). In RDoC terms, this affects Negative Valence and Arousal/Regulatory Systems. These transdiagnostic difficulties make targeting emotion regulation central to treatment.^{3,8}

NEUROBIOLOGICAL MECHANISMS UNDERLYING COGNITIVE CHANGES

The cognitive and emotional difficulties described above do not arise in a vacuum; they reflect underlying neurobiological changes induced by trauma. Early life stress can become biologically embedded, affecting brain structure, connectivity, neurochemistry, and the endocrine and immune systems. Here we outline some key neurobiological findings that link childhood trauma to neurocognitive outcomes.

Stress response systems (HPA axis)

Childhood trauma often dysregulates the hypothalamic-pituitary-adrenal (HPA) axis, the body's central stress system. Initially, trauma can cause hypercortisolism (elevated cortisol) and overactive stress responses; over time, some children develop hypocortisolism (blunted cortisol) due to system exhaustion. Both extremes negatively affect brain development. Dysregulated cortisol impacts cognitive and emotional functioning: hypercortisolism can impair working memory and executive attention, while affecting the amygdala (heightened threat sensitivity) and prefrontal cortex (possible dendritic atrophy). Hypocortisolism may lead to emotional numbness or underarousal, influencing motivation and reward processing, often seen in trauma-related depression. Thus, HPA axis changes link trauma to deficits in memory, learning, arousal, and anxiety regulation.⁴

Brain structure and connectivity

Neuroimaging studies reveal that trauma-exposed children often show structural differences in key brain regions. The hippocampus, critical for memory, is frequently smaller in adults with childhood trauma, particularly those with PTSD. The amygdala, central to threat detection, can be overactive, with some studies reporting larger volumes in children with persistent anxiety, reflecting repeated activation. Prefrontal cortex (PFC) regions, including the ventromedial PFC and anterior cingulate cortex, may be thinner or underdeveloped, affecting impulse control and emotion regulation. The cerebellum, implicated in motor coordination and cognitive timing, may also be reduced in volume following extreme deprivation, as seen in orphanage studies. These differences are group-level trends and not deterministic; some children show no structural alterations, possibly due to genetic resilience or enriched environments.³

Neuroendocrine and neurochemical factors

Beyond cortisol, trauma affects multiple neurochemical systems. Catecholamines such as adrenaline and noradrenaline surge under stress, and chronic elevation can produce hyperarousal and disrupt PFC-mediated EF, contributing to attention and impulse-control problems. Oxytocin, vital for social bonding, may be dysregulated in children who experienced interpersonal trauma, potentially impairing attachment and social cognition. Neuroimmune mechanisms are also implicated; childhood trauma can provoke chronic inflammation, with elevated cytokines linked to slower processing speed and increased risk for depression. Together, these neurobiological alterations illustrate the systemic and multifaceted impact of early trauma.^{9,11}

CHILDHOOD TRAUMA IN INDIA: PREVALENCE, IMPACT AND CULTURAL CONSIDERATIONS

While neurocognitive consequences of childhood trauma appear to be broadly universal, cultural context can influence prevalence, types of trauma and resources available to children. In India, several socio-cultural factors shape landscape of childhood trauma. These include high rates of poverty, child labor, social inequalities (e. g. caste, gender) and at times normalization of harsh physical punishment in some communities.

Clinical observations in India find that trauma-exposed children often present with anxiety, depression, and somatic complaints. Culturally, children (and parents) might express distress in physical terms (headaches, stomachaches) rather than emotional terms, which can complicate recognition of trauma effects.

There is evidence from an adolescent sample in India that parental factors (like parental substance abuse or domestic violence) mediate trauma effects: 1 study found that having a parent with alcoholism significantly increased the risk of child developing anxiety and depression after maltreatment. This aligns with global research on how caregiver mental health can compound/buffer trauma impacts.¹²

TRANSDIAGNOSTIC AND DIMENSIONAL PERSPECTIVES

Childhood trauma often affects multiple disorders, with transdiagnostic factors-such as threat bias, memory problems, executive dysfunction, emotion dysregulation-underlying PTSD, anxiety, depression, and more. This explains high comorbidity and suggests that interventions targeting these core dysfunctions can alleviate multiple problems simultaneously. The RDoC framework highlights trauma impacts across domains: negative valence systems (hypervigilance, anxiety, blunted reward), cognitive systems (attention and executive deficits), social processes (attachment disruptions), and arousal/regulatory systems (hyper/ hypoarousal). Trauma creates latent vulnerabilities-stable neural and cognitive alterations increasing risk across development (e.g., anxiety in childhood, depression in adolescence, substance use in adulthood). Interventions such as attention bias modification, executive function training, and emotion regulation therapies target these core dysfunctions. Considering developmental stage, dysregulation manifests differently from toddlers to adolescents, and dimensional assessment can identify resilience and strengths to guide therapy. This approach also supports preventive psychiatry, enabling early intervention before full disorders emerge.¹³

DISCUSSION

Childhood trauma leaves an indelible mark on the developing mind and brain. This review has highlighted

that trauma in the first decade of life can initiate a cascade of neurocognitive changes-altering how a child pays attention, remembers events, thinks, and regulates emotions-which in turn elevate risk for a spectrum of psychopathologies. These findings carry several important implications.

Integration of findings

Early trauma embeds itself in cognitive-affective systems, shaping survival-oriented adaptations. Children exposed to violence or abuse develop heightened threat detection, stress reactivity, and short-term coping at the cost of learning and exploration. While protective in dangerous environments, these adaptations become maladaptive in safe contexts, leading to hypervigilance, impulsivity, or dissociation that impair functioning and fuel psychiatric symptoms. This model aligns with theories of shattered assumptions, attachment disruptions, and toxic stress neurobiology.¹⁸

Critical analysis of research

Findings in this field are not uniform. Not all maltreated children show cognitive deficits, and effect sizes in meta-analyses are often small, highlighting resilience and compensatory strategies. Causality is difficult to establish, as most evidence is correlational, though natural experiments (e.g., adoption from neglectful orphanages) support environmental influence. Genetic and prenatal factors also interact with trauma, as seen in diathesis-stress models (e.g., 5-HTTLPR polymorphism increasing depression risk).^{14,15}

Moreover, trauma-related cognitive deficits can create a vicious cycle-difficulties with learning or attention may lead to failure, bullying, and added stress-undermining the need for educational support and therapy.^{4,16}

Indian context in discussion

In the Indian context, while the general principles hold, we must consider resource limitations. Many trauma-affected children in India do not receive any mental health intervention. The burden often falls on family (if the family was not the source of trauma) or on the child themselves to cope. There is a scarcity of child psychologists and psychiatrists relative to the need. Culturally adapted screening tools for trauma effects (like simple checklists for teachers or pediatricians to identify cognitive/behavioral red flags) are needed. Encouragingly, awareness is rising, and trauma-informed practices are being slowly introduced in some schools and childcare systems.^{2,17}

Clinical and policy implications

Treatment for trauma-exposed children must be holistic, addressing not only PTSD but also cognitive and emotional difficulties. Effective care often combines

trauma-focused therapies (e.g., TF-CBT, EMDR), cognitive rehabilitation (e.g., working memory and attention training), and emotion regulation skills training (e.g., DBT-based strategies). Parent/caregiver involvement is critical, as supportive, consistent caregiving helps recalibrate stress responses and improve outcomes. At the policy level, prevention is key-child-protection laws, anti-abuse campaigns, and poverty reduction can reduce exposure and protect long-term development, ultimately preventing educational failure, justice involvement, and adult psychiatric disorders.^{7,18}

Future directions

Future research should prioritize longitudinal studies to track how trauma-related deficits evolve over time and to identify sensitive windows for intervention. Biomarkers such as brain imaging, cortisol, and inflammatory profiles may help flag children at high risk, while emerging tools like neurofeedback or brain stimulation could offer targeted treatments. Culturally sensitive studies, especially in India and other non-Western contexts, are crucial for understanding how trauma interacts with factors such as poverty, malnutrition, and community coping strategies. Dimension-based interventions that target core mechanisms like hyperarousal, threat bias, or working-memory deficits may prove more effective than diagnosis-specific approaches.

At the systems level, integrating psychotherapy, cognitive training, and school-based programs can enhance resilience, while trauma-informed curricula that strengthen both emotional and academic skills are especially promising. Technology offers new opportunities through mobile apps, digital tools, and virtual reality, making support more accessible in resource-limited settings. Finally, prevention requires strong policy frameworks-investments in early intervention, training of teachers and health workers, and nationwide trauma-informed initiatives could reduce long-term social and economic costs. Ultimately, the goal is to ensure that trauma does not dictate life trajectories, but is met with early, effective, and culturally relevant support.

CONCLUSION

Childhood trauma disrupts the developmental trajectory of critical neurocognitive processes, including attention, memory, executive function, and emotion regulation. These disruptions increase the risk for a range of psychopathologies across the lifespan. This review integrates evidence from behavioural, cognitive, and neurobiological domains, highlighting the transdiagnostic nature of trauma's impact and emphasizing the importance of culturally sensitive, early interventions. By focusing on neurocognitive mechanisms, this article advances understanding of how trauma becomes biologically embedded and offers pathways for targeted, developmental-stage-appropriate prevention and treatment strategies.

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REFERENCES

1. Boyer SM, Caplan JE, Edwards LK. Trauma-Related Dissociation and the Dissociative Disorders: Neglected Symptoms with Severe Public Health Consequences. *Delaware J Publ Heal*. 2022;8(2):78-84.
2. McCrory E, Gerin MI, Viding E. Annual Research Review: Childhood maltreatment, latent vulnerability and the shift to preventative psychiatry-the contribution of functional brain imaging. *J Child Psychol Psychiat*. 2017;58(4):338-57.
3. McLaughlin KA, Colich NL, Rodman AM. Mechanisms linking childhood trauma exposure and psychopathology: a transdiagnostic model of risk and resilience. *BMC Med*. 2020;18:96.
4. Matte-Landry A, Grisé Bolduc MÈ, Tanguay-Garneau L, Collin-Vézina D, Ouellet-Morin I. Cognitive Outcomes of Children with Complex Trauma: A Systematic Review and Meta-Analyses of Longitudinal Studies. *Trauma Violence Abuse*. 2023;24(4):2743-57.
5. Briggs-Gowan MJ, Grasso D, Bar-Haim Y, Voss J, McCarthy KJ, Pine DS, et al. Attention bias in the developmental unfolding of post-traumatic stress symptoms in young children at risk. *J Child Psychol Psychiat All Disc*. 2016;57(9):1083-91.
6. Williams JMG, Barnhofer T, Crane C, Herman D, Raes F, Watkins E, et al. Autobiographical memory specificity and emotional disorder. *Psychological Bull*. 2007;133(1):122-48.
7. Lakkireddy SP, Balachander S, Dayalamurthy P. Neurocognition and its association with adverse childhood experiences and familial risk of mental illness. *Progress Neuro-Psychopharmacol Biol Psychiat*. 2022;119:110620.
8. Marusak HA, Martin KR, Etkin A, Thomason ME. Childhood trauma exposure disrupts the automatic regulation of emotional processing. *Neuropsychopharmacology*. 2015;40(5):1250-8.
9. Feldman R. Sensitive periods in human social development: New insights from research on oxytocin, synchrony, and high-risk parenting. *Devel Psychopathol*. 2015;27(2):369-95.
10. Stover CS, Keeshin B. Research domain criteria and the study of trauma in children: Implications for assessment and treatment research. *Clin Psychol rev*. 2018;64:77-86.
11. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behavior*. 2012;106(1):29-39.
12. Bhopal S, Verma D, Roy R, Soremekun S, Kumar D, Bristow M, et al. The contribution of childhood adversity to cortisol measures of early life stress amongst infants in rural India: Findings from the early life stress sub-study of the SPRING cluster randomised controlled trial (SPRING-ELS). *Psychoneuroendocrinol*. 2019;107:241-50.
13. Caspi A, Sugden K, Moffitt TE, Taylor A, Craig IW, Harrington H, et al. Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*. 2003;301(5631):386-9.
14. Cook A, Spinazzola J, Ford J, Lanktree C, Blaustein M, Cloitre M, et al. Complex trauma. *Psychiatric Ann*. 2005;35(5), 390-8.
15. Nelson CA 3rd, Bos K, Gunnar MR, Sonuga-Barke EJ. The Neurobiological Toll of Early Human Deprivation. *Monogr Soc Res Child Dev*. 2011;76(4):127-46.
16. Maheshwari P, Jith A, Methala SP, Mathew KA. Prevalence of childhood trauma in patients with psychiatric disorders and its association with perceived social support and suicide attempts: A cross-sectional observational study in a tertiary hospital in South India. *Industrial Psychiat J*. 2024;33(1):88-93.
17. Felitti VJ, Anda RF, Nordenberg D, Williamson DF, Spitz AM, Edwards V, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *Am J Prevent Med*. 1998;14(4):245-58.
18. Arnsten AFT. Stress signalling pathways that impair prefrontal cortex structure and function. *Nature Rev Neurosci*. 2009;10(6):410-22.
19. Bar-Haim Y, Lamy D, Pergamin L, Bakermans-Kranenburg MJ, van IJzendoorn MH. Threat-related attentional bias in anxious and nonanxious individuals: a meta-analytic study. *Psychological Bull*. 2007;133(1):1-24.
20. Ehlers A, Clark DM. A cognitive model of posttraumatic stress disorder. *Behaviour Res Therapy*. 2000;38(4):319-45.
21. Ion A, Bîlc MI, Pițur S. Childhood maltreatment and emotion regulation in everyday life: an experience sampling study. *Sci Rep*. 2023;13:7214.
22. Lindauer RJL, Olff M, van Meijel EP, Ramón JLL, Geertjan O. Executive functions in trauma-exposed youth: A meta-analysis. *Europ J Psychotraumatol*. 2023;9(1):1450595.
23. Tottenham N, Sheridan MA. A review of adversity, the amygdala and the hippocampus: a consideration of developmental timing. *Front Hum Neurosc*. 2010;3:68.
24. Williams JMG, Broadbent K. Autobiographical memory in suicide attempters. *J Abn Psychol*. 1986;95(2):144-9.

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