

Neurobiological effects of childhood trauma: A literature review

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Abstract

This article is a comprehensive review of past literature focusing on neurobiological effects of childhood trauma. Chronic interpersonal violence in children is common worldwide (De Bellis & Zisk, 2014). Exposure to continuous physical and emotional trauma in childhood undoubtedly has long lasting effects like developing post-traumatic stress disorder (PTSD), dysregulation of the hypothalamic–pituitary–adrenal axis (HPA) axis leading to hormonal issues, among other things, in an individual. A systematic review of these effects can prove beneficial in the field of psychiatry.

Introduction

Analyzing prevalence and incidence of traumatic experiences among specified groups (such as children) is a significant way of understanding epidemiologic information. In America, a victimization survey of a nationally representative sample of 4008 adult women, 339 of the women indicated they had experienced at least one completed rape before the age of 18 years, a childhood rape prevalence rate of 85 per 1000 women (Saunders et al., 1999). In another instance, upon conducting searches for studies that were peer-reviewed, published in English, had a representative sample, unweighted estimates, and were published between January 2010 and December 2022 in Criminal Justice Abstracts, Embase, Scopus, PubMed, PsychInfo, and Google Scholar, proportional meta-analysis was conducted to calculate the pooled prevalence for each exposure. The global pooled prevalence of childhood exposure to physical domestic and family violence as a victim or witness was 17.3% and 16.5%, respectively (Whitten et al., 2024). As the events of adverse childhood experiences (ACEs) increase, the risk of negative outcomes and behavior continue to

increase. Childhood trauma not only poses psychological threats but also biological threats.

Methods

This article reviews past literature about psychological and biological effects of childhood trauma. Sources like PubMed, PMC, Google Scholar were used. ChatGPT, an AI tool was also used to gather relevant information to the topic, all of which was corroborated by studies found through the searches. Keywords inputted into the search engines included neurobiological damage, HPA axis dysregulation, PTSD, childhood trauma.

Discussion

Sensitization is defined as enhanced neuroendocrine, autonomic, and behavioral responsiveness to stress as well as limbic-hypothalamic–pituitary–adrenal axis (LHPA) axis dysregulation, particularly corticotropin-releasing factor (CRF) systems, as a result to prolonged exposure to trauma or early life stress. Persistent CRF(1) receptor hypersignaling when a

threatening or traumatic situation is no longer present is maladaptive (Hauger et al., 2012). Such increased stress sensitivity would result in lowering an individual's threshold for developing depression in relation to further stress (De Bellis & Zisk, 2014; Heim et al., 2008). Lower levels of cortisol are seen at the beginning of sleep, while there was a rise towards the end of sleep, with the highest level reached at the moment the individual wakes up (the cortisol awakening response [CAR]) (Azmi et al., 2021). In a study, one hundred and forty-two participants were categorized according to their suicidal history into three groups: suicide attempt, suicidal ideation, or control group. Participants completed questionnaires before commencing a seven-day study. Cortisol samples were provided immediately upon waking, at 15 min, 30 min, 45 min, 3 hr, 6 hr, 9 hr, and 12 hours on seven consecutive days. Childhood trauma was found to be associated with significantly lower CAR associated with HPA-axis dysregulation (O'Connor et al., 2020).

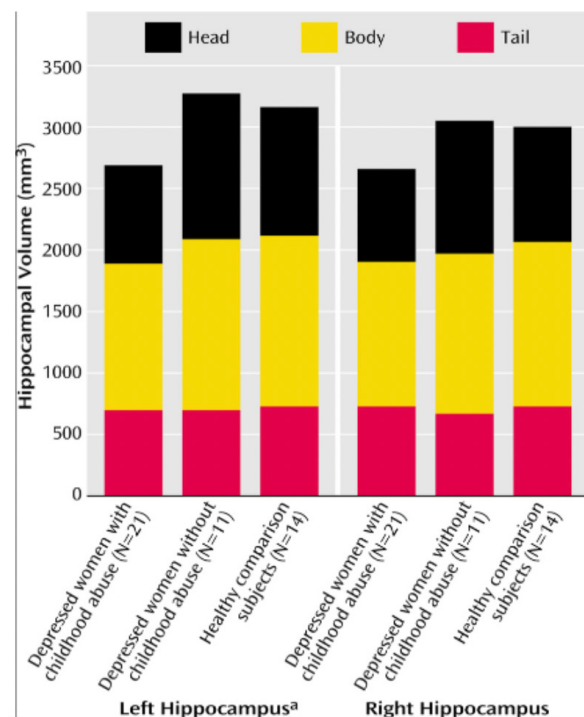
Specific brain areas that play an important role in a variety of types of memory are also preferentially affected by stress, including hippocampus, amygdala, medial prefrontal cortex, and cingulate, having potential to cause neurobiological alterations in post-traumatic stress disorder (PTSD) patients (Vermetten & Bremner, 2002). Exposure to repeated emotional stressors, even in the absence of PTSD diagnoses, has been shown to produce increased synapse formation and dendritic growth in basolateral amygdala, dendritic retraction in the hippocampus (Giotakos, 2020), causing heightened sensitivity to such stressors.

A study showed smaller hippocampal volume as measured with magnetic resonance imaging (MRI) in patients with a history of childhood abuse and the diagnoses of PTSD (Bremner et al., 1995). In another instance, a study aimed at studying the volumes of the hippocampus and of

control brain regions in depressed women with and without childhood abuse and in healthy non-abused comparison subjects concluded women with early childhood sexual abuse and depression had smaller hippocampal volume compared to women with depression without early abuse, and non-abused non-depressed women (Vythilingam et al., 2002).

Figure 1

Results from Vythilingam et al., 2002



Conclusion

Childhood trauma or early life stress has significant impacts and has the potential to create neurobiological alterations. Longitudinal studies over a period are required to determine effective causal relationships of these impacts. Understanding its impacts is important for maintaining healthy functioning in adulthood. Such research also contributes to the effectiveness of treatment for victims.

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