Neuroscience and Biobehavioral Reviews 72 (2017) 68–86

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Review article

Neuropsychological functioning of childhood trauma and post-traumatic stress disorder: A meta-analysis

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ARTICLE INFO

Article history:
Received 1 March 2016
Received in revised form 17 October 2016
Accepted 6 November 2016
Available online 13 November 2016

Keywords:
Children
PTSD
Trauma
Physiological stress response
Maltreatment
Cognition
Neurodevelopment

ABSTRACT

This study reviewed evidence for cognitive impairments in trauma-exposed children with and without PTSD. Twenty-seven studies were eligible for meta-analysis, totalling 1526 participants, including 412 trauma-exposed children (PTSD unknown), 300 children with PTSD (PTSD+), 323 children without PTSD (PTSD-), and 491 trauma-naive controls. Eligible studies mostly investigated familial-maltreatment trauma ($k = 22$). Trauma-exposed children (PTSD unknown) performed more poorly overall than controls ($d = -0.57$). Cognitive deficits were seen in PTSD+ compared to controls, including a large effect size (ES) for general intelligence ($d = -0.88$), moderate ESs for language/verbal ($d = -0.65$), visuospatial ($d = -0.53$), information processing ($d = -0.62$), learning and memory ($d = -0.67$), and executive skills ($d = -0.52$). PTSD+ showed poorer general intelligence ($d = -0.28$) and visuospatial skills ($d = -0.42$) compared to PTSD-, whilst PTSD- showed poorer executive function ($d = -0.23$) and learning and memory ($d = -0.61$) compared to controls. In conclusion, trauma-exposed children showed cognitive deficits compared to controls, although greatest deficits were associated with PTSD diagnosis.

Contents

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http://dx.doi.org/10.1016/j.neubiorev.2016.11.004
0149-7634/

1. Introduction

Research suggests that trauma exposure during childhood has implications for neurodevelopmental outcomes. The physiological stress response is triggered through exposure to a traumatic event, and can have detrimental effects on the brain if prolonged. Activation of the physiological stress response results in the release of glucocorticoids, among other neurochemicals, in various brain regions (Gunnar and Quevedo, 2007), with suppressed or elevated levels being associated with impaired brain development and functioning (Lupien et al., 2009). Supporting this notion, prolonged trauma exposure in the context of childhood maltreatment or deprivation is consistently associated with dysregulated glucocorticoid and catecholamine activity in the developing brain (Weber and Reynolds, 2004; Wilson et al., 2011).

Exposure to extreme stress or deprivation (i.e., trauma) has the potential to disrupt experience-dependent neuroactivity during critical periods of development, which may adversely affect neural structure and function, and disrupt the overall course of neurodevelopment (Gunnar and Quevedo, 2007; Perry et al., 1995). The brain develops in a hierarchical manner starting with primitive structures and functions (i.e., the brainstem and breathing) and ending with complex cortical structures and functions (i.e., frontal lobes and executive functioning; Gogtay and Thompson, 2010). At the neuronal level, this process involves dendritic arborisation, myelination, and synaptogenesis (Anderson et al., 2014; Perry et al., 1995). Given different brain regions develop, organise and become fully functional at different critical developmental periods, age at trauma exposure probably plays a significant role in neurodevelopmental outcomes (Perry et al., 1995; Teicher et al., 2003; van der Kolk, 2003). Supporting this notion, findings from neuroimaging and psychophysiological studies suggest that early life stress occurring during critical developmental periods may affect those brain regions undergoing specific growth spurts at that time, and may also disrupt the development of higher-order association cortices that have prolonged developmental trajectories (e.g., the prefrontal cortex; for review see Pechtel and Pizzagalli, 2011).

In addition to the impact of the trauma exposure itself, the development of post-traumatic stress disorder (PTSD) probably places a child at increased risk of adverse neurodevelopmental outcomes. Post-traumatic stress disorder is a psychiatric disorder that may result from a single-traumatic event, although a dose-response relationship or 'building-block effect' is noted in the literature, with cumulative trauma exposure rendering an individual at increased risk of developing the disorder (Breslau et al., 1999; Brewin et al., 2000; Copeland et al., 2007; Wilker et al., 2015). It is characterized by the onset of trauma-related symptoms including intrusive memories, flashbacks, or nightmares of the trauma; avoidance of trauma-related stimuli; marked alterations in arousal and reactivity post-trauma; and negative alterations in cognitions and mood post-trauma (American Psychiatric Association, 2013).

1.1. The impact of PTSD on childhood neurodevelopment

A widely accepted neurobiological model of PTSD hypothesizes core symptoms of the disorder are associated with a maladaptive stress response that remains disrupted in the absence of the traumatic event (Rauch et al., 1998; Yehuda et al., 2015). There is an abundance of studies investigating the neuroendocrinology, psychopathophysiology, neurostructural and neurofunctional correlates of PTSD in view of this notion. This literature is beyond the scope of this paper, but has been comprehensively reviewed elsewhere (see Pitman et al., 2012; Yehuda et al., 2015). In brief, PTSD symptoms are thought to be associated with structural and functional abnormalities across interacting frontolimbic brain regions that are implicated in the physiological stress response, emotion processing and fear extinction. Core brain regions of interest include the amygdala, hippocampus and prefrontal cortex (Pitman et al., 2012; Yehuda et al., 2015).

The neurocircuitry model of PTSD posits the amygdala response remains exaggerated or hyperactive in the absence of the traumatic event, while the prefrontal cortex remains hyporesponsive, failing to regulate the amygdala response and inhibit associated emotions and irrelevant cognitions (Pitman et al., 2012; Yehuda and LeDoux, 2007; Yehuda et al., 2015). Together these dysfunctions result in deficits in emotion regulation, attentional biases toward perceived threats, increased fear responses, and impaired extinction of trauma-related memories (Pitman et al., 2012). The hyperactive amygdala is also believed to inhibit hippocampal function, leading to impairments in the consolidation of hippocampal-dependent features (i.e., autobiographical memories) of the traumatic event (Goodman et al., 2012). The hyperactive amygdala encodes and consolidates its own version of the traumatic event, with sensory-based representations of the trauma in the amygdala, insula and dorsal visual stream thought to be involved in vivid flashbacks (Brewin et al., 2010; Goodman et al., 2012). Inhibition of the hippocampus is likely associated with the individual's inability to voluntarily recall important aspects of the traumatic event and fragmented or disorganised recall of the traumatic memory (Goodman et al., 2012). Disruption in the hippocampus is also probably associated with deficits in contextual processing post-trauma, whereby the individual fails to distinguish safe from dangerous contexts, resulting in an exaggerated response to trauma-related stimuli (Yehuda and LeDoux, 2007).

Findings from adult structural and functional neuroimaging research show abnormalities in these key frontolimbic regions in individuals with PTSD, providing support for the neurocircuitry model of PTSD (Garfinkel and Liberzon, 2009; Hayes et al., 2012; Patel et al., 2012; Rauch et al., 1998). With regard to the paediatric literature, however, neuroimaging studies on PTSD are relatively scarce. It is therefore difficult to determine whether this neurobiological model is an accurate depiction of childhood PTSD. Current findings are variable and are not entirely consistent with the documented neuropathological process involved in the adult traumatic stress response and PTSD. A detailed review is beyond the scope of this article but has been comprehensively covered in previous literature (see Rinne-Albers et al., 2013).