



Two Hits, but the First Matters Most: Childhood Trauma Predisposes to Adult PTSD and Influences Adult Interpersonal Dynamics



Itamar Shapira, MS-3*; Shirley Zhang, MS-3*; Cooper Elkins, MS-3*; Pamela Parker, MD+

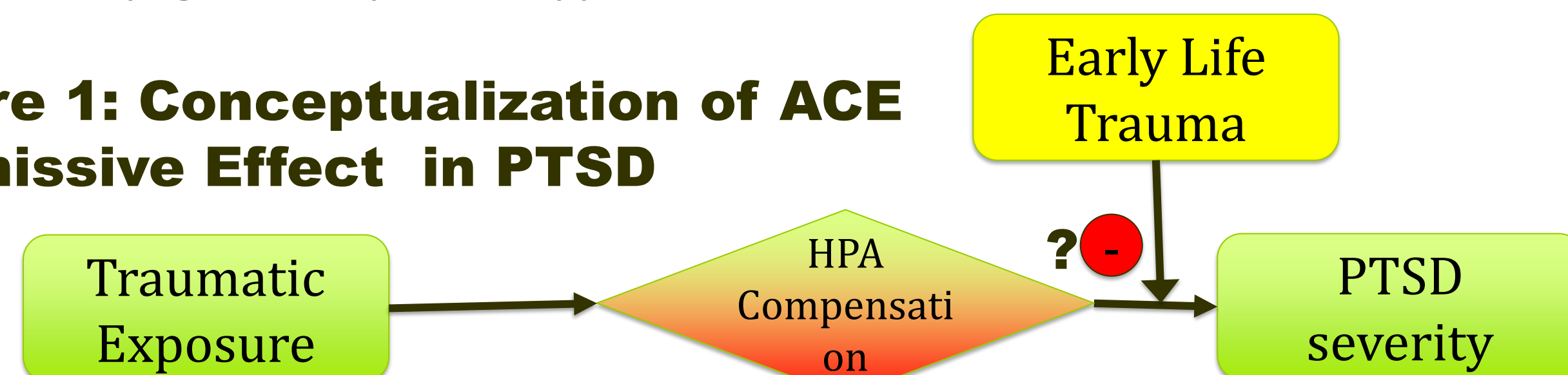
*University of Alabama at Birmingham School of Medicine

+University of Alabama at Birmingham Department of Psychiatry and Behavioral Neurobiology

Introduction

Despite traumatic events being as old as human life itself, our understanding of post-traumatic stress disorder (PTSD) is recent, formally dating to 1980 in the DSM (Scott, 2014). New, phenotypically distinct entities have been recognized as different from the post-traumatic disorders described by survivors of 20th century warzones, such as complex post-traumatic stress disorder (CPTSD), now recognized in the International Classification of Diseases, version 11 (ICD-11). Whereas classic PTSD was understood solely through the lens of reexperiencing, avoidance, and hyperarousal, entrained by a well-defined traumatic event such as combat or motor vehicle collision, ICD-11 CPTSD includes these same criteria and further adds affect dysregulation, alterations of consciousness, disturbed self-perception, disturbed perceptions of offender(s), relational dysfunction, and an altered values system developing progressively in the context of chronic, developmentally adverse interpersonal trauma (Maercker, 2021). There is a possible link between developmentally entrained CPTSD and adult PTSD, with the former having a permissive effect for the development of the latter. In a Dutch study of 85 veterans examined pre- and post-deployment to Afghanistan during Operation Enduring Freedom, methylation of SKA-2, a biomarker correlate of suicidality and cortisol dysregulation (often seen in PTSD), was predictive of PTSD symptoms; strikingly, while greater combat related trauma exposure predicted increasing methylation and cortisol blunting, this appeared to be a pro-compensatory response. Severity of PTSD symptoms was predicted by a longitudinal decrease in SKA-2 methylation, and pre-deployment SKA-2 levels together with childhood trauma exposure significantly predicted severity of PTSD symptoms independently of wartime trauma exposure (Boks et al., 2016). While it is not clear if childhood trauma directly mediates these epigenetic changes, its effect modification with biomarkers raises its likelihood of being part of a causal mechanism. While recent studies have shown that gene methylation is associated with severity of Adverse Childhood Experiences (ACEs), no distinct pattern of methylation was discovered in PTSD cohorts, despite such patterns of methylation being seen with military and combat trauma inventories (Hossack et al., 2020). This may be because of a heterogeneity of epigenetic phenotypes in those with high ACEs or because the remoteness of the temporal association results in an indistinct epigenetic phenotype.

Figure 1: Conceptualization of ACE Permissive Effect in PTSD



Case Summary

Patient is a 44 year old female who was hospitalized for treatment of lacerations and orthopedic injuries sustained during an accident while riding an all-terrain vehicle with her child. She was seen by psychiatry team for symptoms concerning for PTSD. Her history was significant for motor vehicle collision (MVC) approximately ten years ago in which her other child had also been in the vehicle with her. Immediately subsequent to the most recent accident, she reported reexperiencing of the prior MVC, with her first thought after impact being who would take care of her child if she did not survive. Current symptoms includes muscle tension, greatly increased frequency of nightmares (increasing from approximately three yearly to multiple nightly), flashbacks about first MVC and intrusive thoughts about it. She reports having avoided “crazy drivers” subsequent to the first MVC, but feels able to drive because she remains in control of her own vehicle. Her social history was significant for childhood parental physical abuse and subsequent abandonment, death of friends and family members during adolescence and early adulthood, and a prolonged abusive relationship with a substance abusing partner who physically and sexually abused her during adolescence. Patient reports recent oral-maxillofacial surgery performed for cosmetic reasons (related to perceived need to improve attractiveness) and recently terminated a relationship with a different partner who was also substance abusing. Patient is involved in caregiving for others and has adopted parentless children, despite multiparity. She has mainly self-managed PTSD hyperarousal by developing calming breathing practices and nightmares through imaginative exercises to plan non-distressing dreams before falling asleep.

Table 1: Comparison of PTSD and CPTSD criteria

Traumatic Stress Disorders	
PTSD	Complex PTSD
Reexperiencing	Reexperiencing
Avoidance	Avoidance
Sense of threat	Sense of threat
	Affect dysreg
	Negative Self Concept
	Interpersonal disturbances

Discussion

This patient’s presentation is interesting because it demonstrates both classic PTSD (symptoms within the reexperiencing, avoidance, and hyperarousal clusters) and CPTSD (disturbed self-perception resulting in surgery, disturbed perception of abusers leading to renewed harmful relationships, and relational dysfunction in which patient is a caregiver to many people despite personal history of abandonment). Her PTSD development and exacerbation subsequent to both accidents were expressed through concerns regarding interpersonal attachments (her concern for injury or death was mainly for caregiving for children), further connecting PTSD symptoms to a background of interpersonal abandonment and trauma. Though she demonstrates adaptive responses to trauma, such as self-calming techniques and altruism, this second MVC demonstrates that she is at risk of PTSD relapse, with childhood traumatic exposures being a significant risk factor. This case report should encourage clinicians to always screen patients with classic PTSD symptom clusters for childhood adverse experiences and history of physical, sexual, emotional, or other abuse in childhood, because a growing body of evidence indicates the existence of multiple traumatic phenotypes and potential mechanistic effects of early life trauma on post-traumatic pathogenesis and development of DSM-5 PTSD.

References

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