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Permalink
https://escholarship.org/uc/item/17s625xs

Journal
European Journal of Psychotraumatology, 3(0)

ISSN
2000-8198 2000-8066

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Publication Date
2012-09-10

DOI
10.3402/ejpt.v3i0.19492

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Peer reviewed
Effects of Traumatic Stress
Molecular and Hormonal Mechanism
Abstracts from 42nd Annual Conference
New York, September 11–14, 2012
42nd ANNUAL CONFERENCE
ON EFFECTS OF
TRAUMATIC STRESS

MOLECULAR AND
HORMONAL MECHANISMS

Program chair: Tom Hildebrandt
President: Rachel Yehuda
Editor: Miranda Olff

New York
September 11–14, 2012

ISPNE ABSTRACT BOOK
Exposure to childhood trauma among pregnant women is associated with increased placental CRH production over gestation

**Rationale:** Exposure to traumatic events, particularly during sensitive periods in childhood, is known to have persisting effects on health and disease risk in adult life. A few studies that have examined the course and outcome of later pregnancies in women with early trauma history bring up the intriguing possibility of transgenerational transmission of the effect of maternal childhood trauma on her developing fetus. However, the mechanism(s) underlying this effect have yet to be clarified. In humans and other higher primates, a stress-related system that is particularly relevant for key gestational processes, fetal development, and birth outcomes is placental corticotropin-releasing hormone (pCRH). In this study, we address the hypothesis that history of early life trauma is associated with variation in the level and trajectory of pCRH production over the course of human gestation.

**Methods:** A study population of sociodemographically and ethnically diverse women with singleton pregnancies ($N = 333$) provided information about childhood abuse and neglect (Childhood Trauma Questionnaire, or CTQ). Placental CRH levels were assessed prospectively at 1–5 time points over gestation (T1: mean $= 15.0$ weeks, SD $=.72$ until T5: mean $= 36.5$ weeks, SD $=.78$). Because of the expected exponential increase of pCRH production over gestation, pCRH values were log-transformed and Generalized Estimating Equation modeling was employed.

**Results:** One hundred thirty-seven women (41.1%) reported having experienced at least one type of trauma during childhood, and 75 (22.5%) reported exposure to multiple traumas. A higher childhood trauma score was significantly associated with higher pCRH levels over the entire period of gestation ($\chi^2(1) = 4.68, p = .030, \beta = .005$). With the exception of physical and sexual abuse, this relationship was observed for all trauma subscales. The effect was dose dependent, with a higher number of different types of traumas being related to higher concentrations of pCRH.

**Conclusion:** To the best of our knowledge, this is the first report linking exposure to traumatic events in childhood with subsequent placental physiology, thus identifying a possible mechanism of transgenerational transmission. Given the importance of placental CRH in primate pregnancy, this finding also may have appreciable clinical significance.

Keywords: childhood trauma; placental CRH; pregnancy; maternal life course history

Citation: European Journal of Psychotraumatology Supplement 1, 2012, 3 - http://dx.doi.org/10.3402/ejpt.v3i0.19492

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Child abuse moderates cortisol’s relationship to memory

**Background:** Early life stress restructures the nervous system. In rodents, the level of maternal care causes lifelong differences in central glucocorticoid (GC) sensitivity and memory. Furthermore, human adults with a history of child abuse have decreased hippocampal GC receptor gene expression and lower cortisol responses to stress. As GCs modulate memory, hypothalamic–pituitary–adrenal (HPA) axis functions altered by atypical care may influence memory.

**Methods:** Participants were women ($N = 55$) reporting no-to-minimal abuse (no maltreatment group) on the Child Trauma Questionnaire and those reporting clinically significant sexual or physical abuse (abuse group). Participants completed the Beck Depression Inventory (BDI), as depression is more prevalent in child abuse survivors and is known to impact cortisol levels. Participants then completed the Emotional Picture Memory Task. During encoding, participants viewed pictures composed of negative or neutral emotionally valenced objects and backgrounds. In a surprise retrieval test, participants indicated if objects and backgrounds presented separately were the same, similar, or new to those viewed earlier. Saliva samples were collected to measure basal (unstressed) cortisol levels. Analyses focused on “gist” memory, and the percentage of responses when an object viewed previously was classified as similar or the same. Higher cortisol has been tied to better gist memory.

**Results:** Abuse history moderated cortisol’s effect on gist memory, $\beta = −0.557$, SE $= 0.212$, $p < 0.01$. When controlling BDI, cortisol negatively correlated with memory in the abuse group and positively correlated with memory in the no abuse group.