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Psychobiology of Cumulative Trauma: Hair Cortisol as a Risk Marker for Stress Exposure in Women

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Abstract

Childhood trauma (CT) is associated with long-lasting alterations of the hypothalamic-pituitary-adrenal (HPA) axis and elevated risk for stress exposure in adulthood. Although HPA alterations are present in the early aftermath of trauma, it remains unclear how initial HPA activity is associated with subsequent stress exposure and whether CT exposure influences the strength and direction of this association. The present study examined prospective associations between hair cortisol content (HCC) and stress exposure from baseline to 3-month follow-up in young adult women with recent (i.e., past 3 months) exposure to interpersonal violence (IPV; i.e., physical or sexual assault) and non-traumatized controls. History of significant CT (abuse or neglect was determined based on clinical cutoffs for a self-report childhood trauma measure: 12 women had abuse or neglect and recent IPV exposure (CT+IPV); 7 women had abuse or neglect but no IPV exposure (CT); 15 women had no history of trauma (NTC). HCC was computed for 3cm sections reflecting cortisol secretion during the 3 months preceding the baseline assessment. The interaction of cumulative trauma and HCC predicted stress exposure over 3-month follow-up, controlling for baseline stress exposure and depressive symptoms. Simple slopes analyses revealed that lower baseline HCC predicted greater stress exposure in the CT+IPV group compared to the CT group; HCC was not associated with stress exposure in the NTC group. The present findings highlight the potential utility of HCC as a predictor of stress exposure for women with a history of childhood abuse or neglect, particularly in the context of recent IPV.

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Keywords

interpersonal violence; childhood trauma; hair cortisol; stress; abuse; neglect

1. Introduction

Childhood maltreatment is highly prevalent in the United States, affecting as many as 1 in 4 children and resulting in an estimated 3.6 million referrals for suspected abuse or neglect in 2014 (U.S. DHHS., 2016). Exposure to maltreatment can trigger long-lasting changes in hypothalamic-pituitary-adrenal (HPA) function, which, in turn, can have a devastating impact on brain development during critical vulnerability periods (De Bellis, Spratt, & Hooper, 2011). Moreover, maltreatment increases risk for developing posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) following trauma exposure in adulthood, particularly for women (Heim, Newport, Mletzko, Miller, & Nemeroff, 2008).

One potential mechanism contributing to elevated risk for trauma-related psychopathology in adults with maltreatment history is increased exposure to traumatic and non-traumatic stressors. Adults with a history of maltreatment are more likely to experience stressful life events (‘stress generation’; Hammen, Henry & Daley, 2000) and traumatic stressors (‘revictimization’; Coid et al., 2001). Stress generation models emphasize the role individuals play in contributing directly or indirectly to the occurrence of stressful life events. These generated stressors are often interpersonal in nature (e.g., relationship difficulties) and associated with behaviors frequently observed in trauma survivors, including conflict avoidance and restricted emotional expression (Davila & Beck, 2002).

The context processing model of PTSD pathophysiology emphasizes dysregulation in neural circuits recruited in the processing of contextual information to modulate emotional responses (Liberzon & Abelson, 2016). One important implication of this model is that individuals with vulnerability to PTSD may fail to recognize contextual indicators of potential risk despite hypervigilance for threat (Liberzon & Abelson, 2015). Prospective studies suggest that victims of childhood abuse and neglect – particularly women -are at high risk for exposure to interpersonal violence (IPV; Widom, Czaja & Dutton, 2008).

Meta-analytic findings show that diminished cortisol secretion is associated with higher subsequent PTSD symptoms in adults (Morris, Hellman, Abelson & Rao, 2016). Animal studies reveal the importance of HPA activity for context processing: glucocorticoid signaling may be critical for both pattern separation and mediation of contextual fear (Liberzon & Abelson, 2016). Hair cortisol content (HCC) provides a window into long-term HPA secretion patterns, and is closely linked to risk for negative health outcomes (De Bellis et al., 2011). Lower HCC predicted more severe PTSD symptoms in combat-exposed soldiers (Steudte-Schmiedgen et al., 2015). To our knowledge, no studies have yet assessed the impact of HCC on subsequent stress exposure. The present study focused on women, who are at greater risk than men for exposure to specific trauma subtypes and for developing trauma-related psychopathology (Heim et al., 2008; Widom et al., 2008). We hypothesized that lower HCC would predict greater stress exposure in women with greater cumulative trauma exposure.

2. Materials and Methods

We investigated the association between HCC and stress exposure in women with a history of childhood maltreatment/trauma (CT) and recent IPV exposure, women with CT but no IPV exposure, and women with no CT or IPV exposure. HCC was used to gauge cumulative cortisol production over the 3 months preceding the baseline assessment. For women with recent IPV, baseline assessments were conducted within 3 months of an IPV incident; for women without recent IPV, baseline assessments reflected their first visit to PI's laboratory.

2.1. Participants

The sample was drawn from a larger, ongoing longitudinal study of PTSD risk markers and included all women who agreed to provide hair samples for cortisol analysis. Participants were screened for IPV exposure prior to baseline using the Life Events Checklist (Gray, Litz, Hsu & Lombardo, 2005); they were deemed eligible if they either (a) experienced an incident involving physical and/or sexual assault within 3 months of the baseline assessment, or (b) had no history of exposure to physical or sexual assault. Participants were 12 women with CT (abuse or neglect) and recent IPV exposure (CT+IPV), 7 women with CT but no history of IPV exposure (CT), and 15 women had no history of CT or IPV (non-trauma controls, NTC). They were recruited through online advertisements, local agencies providing services to domestic violence and sexual assault survivors, and through a team of nurse practitioners providing medical legal exams to rape survivors. Follow-up assessments were conducted 1- and 3-months after baseline.

Participants with current or past psychiatric disorders (i.e., bipolar disorder, current PTSD, current MDD) or health conditions (Cushing's Disease, Addison's Disease, hyperthyroidism, severe kidney or liver disease, pregnancy, and hypoglycemia) known to exert an influence on stress hormones, or current use (i.e., any use in the past month) of prescription (corticosteroids, amphetamines, antianxiety or antidepressant medication, marijuana) and non-prescription (marijuana) drugs that might affect HPA activity were excluded. The diagnosis of lifetime psychiatric disorders was based on the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; First, Spitzer, Gibbon & Williams, 1997) and the Clinician-Administered PTSD Scale (Blake et al., 1995). IPV participants with prior episodes of MDD or PTSD, or those using oral contraceptives, were included due to concerns about recruitment feasibility and generalizability of the findings. NTC reported no lifetime history of physical or sexual assault and no traumatic events occurring in the year prior to their first assessment. Participants provided written informed consent and all procedures were approved by the institutional review board.

2.2. Measures

Childhood trauma—The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994) is a 28-item self-report assessing the frequency of different types of abuse experienced as a child. Participants rated each item on a 5-point scale from “never true” to “very often true.” The CTQ has five subscales: emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Clinical cutoffs for scores on each scale were used to determine the presence or absence of significant abuse or neglect (Walker et al., 1999). The

CTQ has good reliability and validity (Bernstein et al., 1994); coefficient alpha in this sample was .90.

Prolonged HPA activation—Participants provided hair samples at baseline to determine HCC. Hair samples were cut as close to the scalp as possible from a posterior vertex position. Each sample yielded a 3cm segment reflecting cortisol secretion over the previous 3 months. HCC was determined using a commercially available immunoassay with chemiluminescence detection (CLIA, IBL-Hamburg, Germany). Samples were analyzed in duplicates. The intra-assay and inter-assay coefficients of variation of this assay were < 12%.

Hair characteristics—Hair-specific characteristics were assessed with a self-report form. They included current hair color, natural hair color, hair structure (straight, curls, waves/wavy, extensions, wig), hair washing frequency (number of washes per week), and hair treatment (none, gel/hair spray, highlights, coloring, dying, chemical/heat straightening).

Depressive symptoms—The Beck Depression Inventory-Second Edition (BDI-II; Beck, Steer, Ball & Ranieri, 1996) is a 21-item, self-report measure assessing severity of depressive symptoms. The BDI-II has good reliability and validity (Beck et al., 1996); coefficient alpha in this sample was .94.

Stress exposure—The short form (90 items) young adult version of the Perceived Events Scale (PES; Compas, Davis, Forsythe & Wagner, 1987) was used to measure the number of negative life events experienced by participants six months prior to the baseline assessment. Participants were asked to indicate whether each event occurred during this time, and to rate the valence of those events on a 9-point scale (-4 = Extremely Bad; 0 = Neither Good or Bad; +4 = Extremely Good). A stress exposure score was calculated by summing the number of negative events (i.e., those rated -1 to -4 on desirability).

2.3. Follow-up Measures

Stress levels—The PES was repeated at one and 3 months, instructing participants to report the events during the follow-up interval. A stress exposure score was calculated by summing the number of negative events from baseline to 3-month follow-up. The PES has adequate test-retest reliability for event occurrence and valence (Compas et al., 1987); test-retest reliability for stress exposures at baseline and 3-month follow-up in this sample was .71.

2.4. Data-Analytic Strategy

Given positive skewness in raw HCC data, HCC was log-transformed. Multiple linear regression was used to test the interaction of HCC (estimated 3 months prior to baseline) and cumulative trauma (group: CT+IPV, CT, NTC) predicting stress exposure over 3-month follow-up; baseline stress exposure and depressive symptoms were included as covariates. HCC was centered before including in the interaction; significant interactions were probed with simple slope analysis.

3. Results

Demographic and clinical characteristics for the CT+IPV, CT and NTC groups are presented in Table 1. No significant group differences were observed for the distribution of race, hair structure, hair treatment or frequency of hair washing. Groups differed significantly in the distribution of current and natural hair color. The CT+IPV group reported higher scores on depressive symptoms than CT and NTC groups. Contrary to expectation, with the exception of emotional neglect, the CT+IPV group reported significantly higher scores on childhood trauma subtypes compared to the CT group.

There were no between-group differences in HCC; moreover, HCC was not significantly associated with current hair color, natural hair color, hair structure, hair treatment, or frequency of hair washing (p's > .2). Baseline stress exposure was significantly higher in the CT+IPV and CT groups compared to the NTC group but no significant between-group differences emerged during 3-month follow-up. Across groups, HCC did not correlate with baseline (r = .07, p = .68) or follow-up (r = -.001, p = .99) stress exposure. However, within groups (see Table 2): HCC was positively correlated with baseline (r = .67, p = .007) and follow-up (r = .65, p = .009) stress exposure in the NTC group; HCC was negatively correlated with follow-up stress exposure (r = -.68, p = .015), but not with baseline stress exposure (r = -.24, p = .45), in the CT+IPV group; HCC did not correlate with baseline (r = -.12, p = .80) or follow-up (r = -.08, p = .87) stress exposure in the CT group.

3.1 HCC and Cumulative Trauma Predict Stress Exposure during Prospective Follow-up

Multiple regression analysis indicated that the interaction of cumulative trauma and HCC predicted stress exposure over 3-months (β = -.52, t = 4.09, p < .001), over and above the influence of baseline stress exposure and depressive symptoms (Figure 1). Simple slopes analyses revealed that lower baseline HCC predicted greater stress exposure (i.e., more negative life events) in the CT+IPV group (β = -.92, t = 3.91, p = .001) compared to the CT group (β = -.34, t = 2.69, p = .012). There was a non-significant trend for higher HCC to be associated with higher stress exposure in the NTC group (β = .23, t = 1.77, p = .088).

4. Discussion

The potential role of stress response systems as contributors to stress exposure in trauma survivors has been relatively neglected. The present findings suggest that lower HCC predicts greater stress exposure in women with a history of significant childhood abuse or neglect (CT) compared to women with no history of maltreatment - particularly when this maltreatment-related diathesis is primed by recent exposure to IPV. There is mounting evidence that HPA hypoactivity is a marker of risk for PTSD (Morris et al., 2016; Steudte-Schmiedgen et al., 2015). This study adds to that literature by demonstrating a potential role for HPA hypoactivity as a prospective risk marker for stress exposure in women with a history of childhood maltreatment, particularly in the context of recent trauma experience.

The negative impact of childhood trauma exposure on mental and physical health has long been recognized (De Bellis et al., 2011; Heim et al., 2008). In the present study, childhood maltreatment in the context of lower HCC predicted greater stress exposure. Higher rates of

peer rejection, bullying, relationship difficulties, and social withdrawal in maltreated individuals could be explained, in part, by maladaptive interpersonal behaviors. Context-processing deficits in individuals at risk for PTSD (Liberzon & Abelson, 2016) may contribute to these maladaptive behaviors by increasing the likelihood of revictimization (via interference with social threat detection) and social withdrawal (via interference with safe context detection). Animal studies underscore the importance of glucocorticoid signaling within neural circuits that subserve contextual information processing (Liberzon & Abelson, 2016). The context-processing model posits that accumulating trauma leads to reduced HPA axis reactivity to subsequent exposure and alterations in cortisol-sensitive neural circuits involving hippocampus and pre-frontal cortex, and thus proposes an HPA-based disruption of context-processing capability that can contribute to repeated trauma exposure. Future studies should investigate the possibility that HPA hypoactivity may increase risk for stress exposure by disrupting cognitive processes (such as contextual information processing) that are central to interpersonal functioning.

Hair samples provide estimates of long-term integrated free cortisol production. Our finding that lower HCC predicted greater stress exposure among women with both childhood and recent trauma exposure is consistent with one study showing lower HCC in high-stress compared to low-stress mothers (Ouellette et al., 2015). The association between HCC and perceived stress may be curvilinear. Consistent with this hypothesis, one study found a positive association between HCC and perceived stress at low-to-moderate stress levels but diminishing HCC at more extreme stress levels (Wells et al., 2014). The present findings add to this literature by showing a *positive* correlation between HCC and stress exposure over follow-up for the NTC group but a *negative* correlation between HCC and stress exposure over follow-up for the CT+IPV group.

Strengths of the present study included the use of an integrated measure of long-term cortisol output in women with recent IPV exposure, careful assessment of hair characteristics, and prospective ‘post-post’ design. The following limitations provide directions for future research. First, the sample size was modest but comparable to other studies examining HCC in trauma-exposed individuals. Second, exposure to childhood maltreatment (CT) was determined by a retrospective self-report measure. The CTQ is widely-accepted and well-validated (Bernstein et al., 1994) and cutoff scores were utilized to determine significant abuse/neglect exposure (Walker et al., 1999). However, it is possible that higher baseline depressive symptoms in the CT+IPV group relative to the CT and NTC groups influenced reporting of childhood trauma, which could explain higher trauma subtype scores in the CT+IPV compared to the CT group. Third, this study was conducted in women only, which limits generalizability and precludes examination of potential sex differences. Fourth, this study did not include a comparison group of women who recently experienced an incident of IPV but did not report a history of significant childhood abuse or neglect.

The present findings highlight the potential role for low HCC as a risk marker for stress exposure that may be particularly salient during the acute aftermath of physical or sexual assault in women a history of childhood maltreatment. Prolonged HPA hyposecretion may interfere with information processing relevant to interpersonal encounters which, in turn,

could account for both stress generation effects and revictimization (Coid et al., 2001). One possible route for stress prevention suggested by the present study is to increase HCC in vulnerable women. Interventions designed to reduce stress levels in healthy individuals through training in cognitive-behavioral stress management skills have reported significant decreases in stress reactivity measured by HCC (Iglesias et al., 2015). Whether interventions could be used to produce the opposite pattern – an increase in HCC – in women with cumulative trauma exposure remains unclear. Future studies should examine the benefits of IPV-relevant interventions that enhance HPA secretion for reducing the risk for additional stress exposure.

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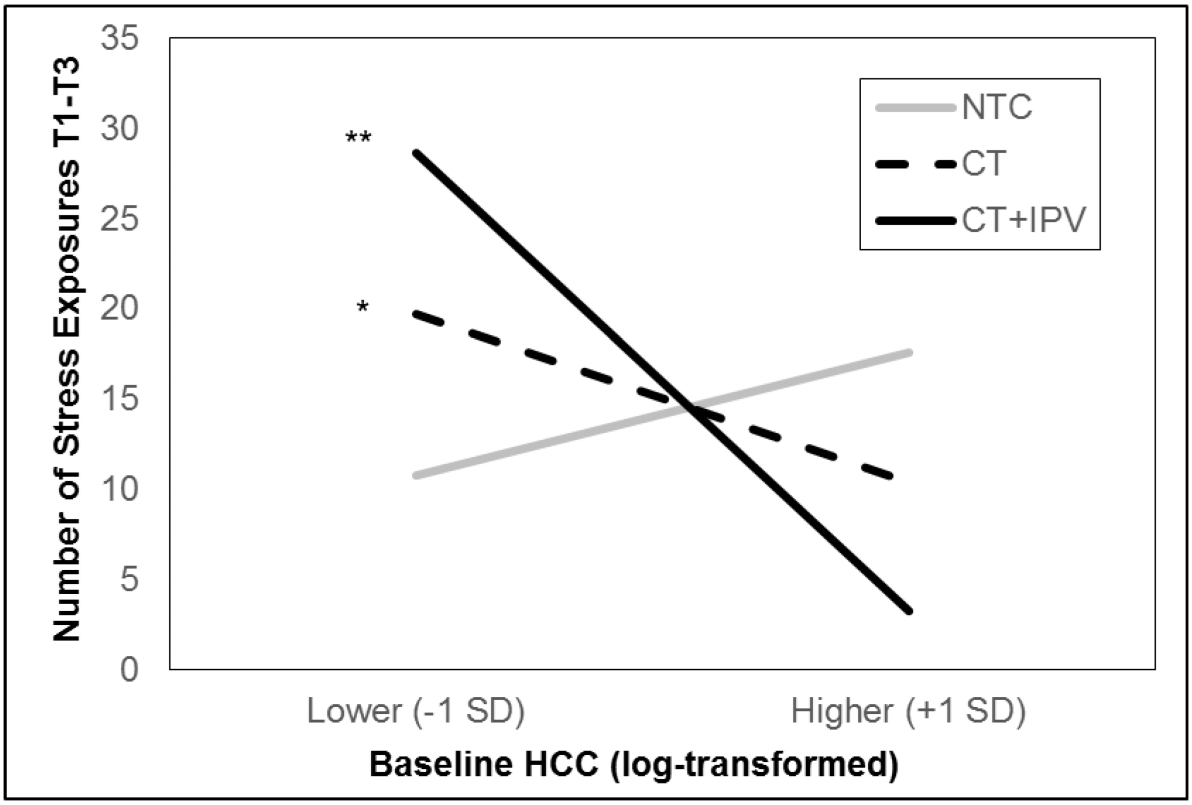

Figure 1.

Interaction of HCC and Cumulative Trauma Predicting Stress Exposure from Baseline (T1) to 3-month follow-up (T3). Simple slopes groups represent: NTC = non-trauma controls; CT = childhood abuse or neglect without recent interpersonal violence exposure; and CT+IPV = childhood abuse or neglect with recent interpersonal violence exposure. HCC = hair cortisol content. *$p < .05$; **$p < .01$.

Table 1

Demographic and Clinical Characteristics of the Sample.

	CT+IPV (*n* = 12)	CT (*n* = 7)	NTC (*n* = 15)	Group Differences
	***N* (%)**	***N* (%)**	***N* (%)**	X^2
Race				5.14
Caucasian	6 (50)	5 (71)	13 (87)	
African-American	5 (42)	2 (29)	2 (13)	
Asian	1 (8)	-	-	
Hair color (current)				14.00 *
Brown	7 (58)	1 (14)	9 (60)	
Black	1 (8)	1 (14)	2 (13)	
Blond	1 (8)	5 (71)	4 (27)	
Red	3 (25)	-	-	
Hair color (natural)				15.56 *
Brown	9 (75)	1 (14)	10 (67)	
Black	1 (8)	1 (14)	1 (7)	
Blond	-	5 (71)	4 (27)	
Red	2 (17)	-	-	
Hair structure				6.35
Straight	2 (17)	2 (29)	9 (60)	
Curls	5 (42)	2 (29)	4 (27)	
Waves/wavy	5 (42)	3 (43)	2 (13)	
Hair treatment				8.97
None	4 (33)	1 (14)	7 (47)	
Gel/hair spray	-	1 (14)	2 (13)	
Highlights	1 (8)	1 (14)	1 (7)	
Hair coloring	3 (25)	-	2 (13)	
Hair dying	1 (8)	2 (29)	1 (7)	
Chemical/heat	3 (25)	2 (29)	2 (13)	
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*F*
Age	23.6 (3.6)	22.6 (2.3)	25.7 (3.4)	2.72
Depressive symptoms	20.5 (14.8)[a]	7.0 (5.4)[b]	3.7 (6.0)[b]	9.90 ***
Trauma subtypes				
Emotional abuse	13.5 (6.7)[a]	7.6 (3.2)[b]	6.1 (1.4)[b]	10.40 ***
Physical abuse	10.3 (6.5)[a]	6.1 (1.2)[b]	5.4 (0.7)[b]	5.39 *
Sexual abuse	10.9 (7.4)[a]	5.7 (1.9)[b]	5.1 (0.3)[b]	6.20 **
Emotional neglect	10.3 (5.3)[a]	10.6 (5.3)[a]	5.9 (1.3)[b]	5.46 **
Physical neglect	10.8 (4.5)[a]	7.7 (2.3)[b]	5.3 (0.6)[b]	11.62 ***
Hair wash per week	3.0 (2.4)	4.1 (2.5)	5.2 (2.4)	2.89

	CT+IPV (*n* = 12)	CT (*n* = 7)	NTC (*n* = 15)	Group Differences
	N (%)	*N* (%)	*N* (%)	X^2
HCC (pg/mg; log)	1.3 (0.4)	1.2 (0.4)	1.5 (0.7)	0.48
Stress Exposure				
Baseline	18.2 (9.7)[a]	13.6 (12.5)[a]	9.3 (5.6)[b]	3.42 *
Follow-up (3 months)	23.9 (19.0)	12.4 (8.5)	11.9 (10.6)	2.83

* $p < .05$;

** $p < .01$;

*** $p < .001$.

Note: Within rows, values with different superscripts ([a] and [b]) differ significantly at $p < .05$; CT+IPV = childhood abuse or neglect with recent interpersonal violence exposure; CT = childhood abuse or neglect without recent interpersonal violence exposure; NTC = non-trauma controls; HCC = hair cortisol content (log-transformed to reduce skewness); group differences were examined with chi-square tests for categorical variables and analysis of variance for continuous variables.

Table 2

Correlations among Hair Cortisol and Stress Exposure.

	CT+IPV (*n* = 12)	CT (*n* = 7)	NTC (*n* = 15)
Stress exposure	**HCC (baseline)**	**HCC (baseline)**	**HCC (baseline)**
Baseline	-.24	-.12	.67**
Follow-up (3 months)	-.68*	-.08	.65**

* $p < .05$;

** $p < .01$.

Note: CT+IPV = childhood abuse or neglect with recent interpersonal violence exposure; CT = childhood abuse or neglect without recent interpersonal violence exposure; NTC = non-trauma controls; HCC = hair cortisol content (log-transformed to reduce skewness).