

# Intimate partner violence is associated with increased maternal hair cortisol in mother–child dyads

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## Abstract

**Background:** The chronic consequences of intimate partner violence (IPV) on HPA activation are a topic of debate. The current study investigated hair cortisol concentrations in female victims of IPV and their children.

**Methods:** A total of 52 mother–child dyads were divided into two groups depending on exposure to IPV: IPV group ( $n = 27$  dyads) and control group ( $n = 25$  dyads). Hair cortisol concentration was measured in 1-cm-long hair strands, representing 30 days of exposure before assessment. PTSD and depression symptoms were assessed in the mother and child.

**Results:** Women reporting IPV presented with higher hair cortisol levels, depression and PTSD symptoms severity in comparison to control women. Children who witnessed IPV reported more severe PTSD symptoms, but depressive symptoms and hair cortisol were not statistically different than those in control children. Correlation analyses revealed a positive association between the number of injury events and the level of hair cortisol in children. No associations between the hair cortisol levels in mothers and those in their children were found.

**Conclusion:** Higher hair cortisol levels detected in women exposed to IPV reflected long-lasting changes in HPA axis functioning associated with chronic stress exposure. Children whose parents recurrently engage in violent conflicts with intimate partners may often feel threatened and consequently reporting more PTSD-related symptoms. Given that experiencing and witnessing violence during childhood and adolescence are predictive of intimate partner violence in adulthood, the need of early interventions is crucial.

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## 1. Introduction

Violence against women inflicted by intimate partners is an important worldwide public health problem that significantly affects women's mental and physical health [1]. Globally, an estimated 30% of women are exposed to intimate partner violence during their lifetimes [2,3]. In most cases, this chronic social traumatic stressor involves repeated episodes of physical, psychological, and sexual violence that dramatically disturb the victim's sense of security [4]. Specifically, sexual abuse occurs in approximately 40% of all cases of intimate partner violence. The mental health consequences are greater in women who are sexually abused

than in those who are not [5–7]. Intimate partner violence (IPV) refers to physical violence, sexual violence, or psychological aggression (including coercive acts) perpetrated by a current or former intimate partner [8]. Despite the relevance of IPV, most research to date have focused on assessing the consequences of IPV on mental health [9–11], while its biological correlates have been relatively under-explored [5,12,13].

As in other stressful conditions, exposure to IPV might trigger a series of harmful biological consequences in victims [14–17]. For example, chronic violence or sexual abuse is usually associated with short- and long-term alterations in the functioning of the hypothalamic-pituitary-adrenal (HPA) axis and with automatic nervous system dysregulation [18]. Although HPA axis activation acts primarily as a protective response against stress [19], chronic and recurrent exposure to stressful experiences increases metabolism. Resources are consumed without sufficient recovery, promoting long-term

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changes in the functioning of stress-related biological systems. Consequently, the risk for physical and mental health problems increases [14,19]. This process, named “allostatic load”, can inhibit neurogenesis, block neuronal plasticity, and induce neurotoxicity [14]. Brain regions such as the hippocampus, amygdala, and prefrontal cortex undergo structural and functional remodeling in response to acute and chronic stress and chronic HPA axis activation [16].

Measurement of cortisol, a steroid hormone released from the adrenal gland, is often used to investigate the association between chronic stress exposure and HPA axis reactivity [20]. Previous investigations have yielded mixed results regarding cortisol levels in women exposed to IPV, with cortisol levels both higher and lower than those in control women [17,21]. Psychiatric conditions such as the severity of depressive and posttraumatic stress disorder (PTSD) symptoms may be important mediators of the relationship between IPV and cortisol levels [4,22]. For example, a systematic review [23] found that patients with major depression presented with increased cortisol levels, while patients with anxiety disorders (generalized anxiety, panic, and PTSD) presented with lower levels, suggesting that distinct pathways contribute to the dysregulation of the HPA axis in the development and/or maintenance of psychiatric conditions.

In addition, some studies have also shown higher cortisol levels and increased physiological arousal in children who witness marital violence [24]. Children and adolescents in families characterized by higher levels of marital functioning showed lower wake-up cortisol levels [25]. However, children whose parents recurrently engage in violent conflicts with intimate partners may often feel threatened and consequently stressed, leading to chronic elevation of cortisol levels. Furthermore, some authors highlight associations between hormone levels in parents and their children [25,26]. In children with higher cortisol levels, one or both parents also exhibit elevated cortisol concentrations [25]. Specifically, for victims of intimate partner violence, maternal and infant salivary cortisol concentrations were positively correlated [26]. Therefore, the mother–child bond might deeply influence and regulate behavioral and physiological responses to stress [26,27].

Although some studies have investigated the relationship between cortisol levels and intimate partner violence in women [23,28–30], the majority have used plasma, serum, or saliva samples to assess cortisol. These measurement methods are subject to major physiological daily fluctuations, making the assessment of overall long-term systemic cortisol exposure difficult and leading to inconsistent results. On the other hand, in recent years, hair cortisol measurement has emerged as a promising way to measure chronic stress and capture systemic cortisol exposure over longer periods of time [28,30,31]. Hair grows at a median rate of 1 cm/month; therefore, the first centimeter of hair at the scalp follicle indicates the past month’s cortisol production, while the second centimeter section indicates the cortisol production of

the month before, and so on [30]. In addition, evidence has shown that hair cortisol concentrations are not affected by sex, hair treatments, or pharmacological interventions, indicating that hair cortisol could be a highly important biomarker in stress studies [30,32]. On the other hand, a recent study found that hair cortisol was higher in boys than in girls [33].

Given that hair cortisol assessment is a promising approach with which to investigate chronic stress load in children and adults [34–38], the present study compared hair cortisol levels in women exposed to IPV and their children to hair cortisol levels in women without such exposure and their children. To our knowledge, this study is the first to test the hypothesis that both women exposed to IPV and their children will present with higher hair cortisol levels. We also hypothesized that PTSD or/and depressive symptoms would be associated with variations in cortisol levels.

## 2. Materials and methods

### 2.1. Participants

A total of 118 participants were included in this study. Thirty-five women exposed to IPV and their biological children were recruited through the Center for Helping Women Victims of Intimate Partner Violence in Southern Brazil; three withdrew from the study ( $n = 32$ ). This service is designed to protect women from contact with their partner in such a way that no participant had ongoing partner violence when assessed. For each woman, one child aged 6–12 years who had witnessed IPV in any form was included in the study. If a woman had more than one child in this age range, she was asked to select the child she thought had the most exposure to the consequences of intimate partner violence. Thirty-five control participants were recruited from Community Family Associations. We invited potential research volunteers by direct approach. When these volunteers agreed to join the research, we interviewed them. After screening for the absence of psychiatric symptoms and any violence exposure, we retained 27 mothers and 27 children in this group. For all participants, the inclusion criteria were: at least 1 cm of hair length, lower income according to Brazilian parameters (US\$450 per capita per month, according to Brazilian parameters – Social quotas’ law n° 12.711, August 29th, 2012), no severe medical illness, no severe cognitive impairment, no psychotic symptoms, no substance abuse in the past 30 days, no illiteracy, and no corticosteroid use. The ethical committee of institutions approved this study, and all participants provided written informed consent.

### 2.2. Demographics and clinical assessments

Trained researchers conducted face-to-face interviews just after collecting hair samples. Each mother and child was

interviewed separately in a two-hour session: while one interviewer was with the mother, another was with the child in a separate room. Information about the age and educational level of the mother and the child, the marital status, income, number of children per woman, and the violence perpetrated by the woman's partner against the child was obtained from the mothers during the clinical interview.

All women were assessed using the Revised Conflict Tactics Scale (CTS2), a 78-item scale that assesses whether a couple engages in psychological and physical attacks on each other, as well as their use of reasoning or negotiation to deal with conflicts throughout the life of the relationship [8,39]. The CTS2 has five scales: Physical Assault, Sexual Coercion, Injury, Psychological Aggression, and Negotiation. The Physical Assault scale assesses direct partner-inflicted physical aggression toward the partner. The Sexual Coercion scale indicates whether the respondent was exposed to coerced or forced sex with a partner, covering a range of coercive acts, from verbal insistence to physical force. The Injury scale measures partner-inflicted physical injury, as indicated by bone or tissue damage, need for medical attention, or pain continuing for a day or more. The Psychological Aggression scale investigates coercive or aversive acts intended to produce emotional harm or threat of harm. The Negotiation scale evaluates actions taken to settle a disagreement through discussion. In this study, positive exposure to IPV was determined if respondents reported being exposed to any situation of sexual coercion, physical assault, severe psychological aggression or injury inflicted by their intimate partner in the last 12 months [8]. In addition, if IPV is detected, CTS2 gives a chronicity score by counting the mean number of times the acts or events occurred in the previous 12 months. The Cronbach's coefficient alpha in the current study was 0.90 for CTS2. The history of violence against children was collected during the clinical interview with the mothers.

The Beck Depression Inventory-II (BDI-II) [40] and the Children's Depression Inventory (CDI) [41] were used to assess depressive symptoms in women and their children, respectively. Cronbach's coefficient alphas were 0.90 for total BDI and 0.80 for total CDI. The Child PTSD Symptom Scale (CPSS) [42] and the PTSD Symptom Scale – Self-Report Version (PSS-SR) [43] were used to assess PTSD symptoms in women and their children, respectively. Cronbach's coefficient alphas were 0.95 for total PSS and 0.88 for total CPSS.

### 2.3. Hair cortisol assessment

Hair cortisol concentrations were measured in 10 mg of 1-cm-long hair strands carefully collected with a surgical scissor from the vertex posterior of a participant's head, as close to the scalp as possible, following our group's standardized protocol [34]. One-cm hair sections proximal to the scalp represented a 1-month period of cortisol

exposure [44,45]. The hair follicle was not included [45]. This scalp area was chosen because it shows the lowest variation among individuals and does not result in any aesthetic damage. Dyed hair color does not affect cortisol concentrations [30]. After collection, samples were stored at room temperature for up to 6 months.

Hair steroid extraction procedures were performed according to the protocol described previously [29,34]. At least 10 mg of powdered hair per 1-cm section was weighed and separated into different glass vials. Methanol (1 mL, 50 °C) was added, and the vials were sealed. The samples were sonicated for 30 min and incubated overnight at 52 °C for 16 h. After incubation, 0.75 mL of the supernatant methanol was removed, placed into disposable glass tubes, and evaporated under 50 °C. The residues were dissolved in 250  $\mu$ L phosphate-buffered saline (pH 8.0) and vortexed for 1 min. For a double-blinded measurement of cortisol in the extracts, a commercially available, high-sensitivity salivary cortisol enzyme-linked immunosorbent assay (ELISA) (Salimetrics LLC, State College, PA, USA) was used, according to the manufacturer's instructions. All samples were run in duplicate.

### 2.4. Statistical analyses

Hair cortisol data presented a non-normal distribution. Therefore, between-group comparisons were carried out using the Kruskal–Wallis test with Dunn's test for post-hoc analysis. Because of abnormal and extreme values of hair cortisol, we excluded two outliers from the IPV group and one outlier from the control group from cortisol analyses since they presented aberrant values. We corrected all analyses for multiple comparisons using the Bonferroni adjustment. The independent two-tailed t-test, Mann–Whitney U test, and chi-square test were used to compare clinical and sociodemographic data when appropriate. Spearman correlations were performed to investigate the association between violence exposure, clinical and demographics variables, and hair cortisol.

## 3. Results

The clinical and sociodemographic profiles of the groups are presented in Table 1. No differences were found regarding the age and sex of the child. We found that IPV mothers had a median of three children while control women had two. All women reported themselves as married or living in a stable relationship with their partner, with the exception of two divorced IPV participants. We identified lower education among IPV women. As expected, women exposed to IPV reported higher rates of physical and sexual violence, as well as greater psychological aggression and injuries inflicted by their partners. Similarly, the rates of the violence perpetrated against children within the IPV group were higher (Table 1).

Table 1  
Sociodemographics, clinical and violence exposure characteristics of dyads.

Variables	IPV women (n = 32)	Control women (n = 27)	Statistics	<i>p</i>	IPV children (n = 32)	Control children (n = 27)	Statistics	<i>p</i>
Age (years)	34.15 (6.52)	36.03 (8.31)	t(57) = 0.97	.33	8.96 (2.00)	8.92 (1.61)	t(57) = −.08	.33
Sex								
Female (%)	100	100			50.0	55.6	×2(1) = .18	.67
Male (%)	-	-			50.0	44.4		
Number of children (mdn, min–max)	3.00 (1–8)	2.00 (1–8)	U = 298.00	.03	-	-		
Education (years)	6.96 (3.25)	8.88 (3.02)	t(57) = 2.32	.02	3.33 (1.77)	3.48 (1.61)	t(50) = .31	.75
PTSD severity (PSS-SR/CPSS)	20.53 (11.56)	2.37 (2.60)	t(29.41) = −9.45	<.001	15.37 (9.71)	3.18 (5.49)	t(50.31) = −6.04	<.001
Depression severity (BDI-II/CDI)	16.43 (9.99)	4.96 (3.33)	t(31.70) = −5.81	<.001	11.15 (6.57)	8.51 (6.64)	t(57) = −1.52	.13
Intimate partner violence (CTS2)								
Physical assault								
Prevalence (%)	100	0	×2(1) = 59.00	<.001	-	-		
Chronicity	27.56 (13.01)	-						
Sexual coercion								
Prevalence (%)	65.6	0	×2(1) = 27.51	<.001	-	-		
Chronicity	6.03 (6.99)	-						
Severe psychological aggression								
Prevalence (%)	93.8	0	×2(1) = 51.49	<.001	-	-		
Chronicity	6.93 (4.02)	-						
Severe injury								
Prevalence (%)	46.9	0	×2(1) = 16.97	<.001	-	-		
Chronicity	9.53 (5.26)	-						
Rate of direct violence against children (%)					45.20	0	×2(1) = 16.07	<.001

Values exhibited as mean (standard deviation), or proportion (%), or median (minimum–maximum).

### 3.1. Hair cortisol levels between IPV-dyads and control-dyads

A Kruskal–Wallis H test showed that there was a statistically significant difference in hair cortisol levels between groups ( $H(4) = 23.62, p < 0.0001$ ), with a mean rank hair cortisol of 81.9 for IPV women, 56.98 for control women, 50.07 for IPV children, and 41.81 for control children. Dunn's test for post-hoc analysis revealed that IPV women had higher hair cortisol levels than did control women ( $p = 0.005, adj p = 0.003$ ), IPV children ( $p < 0.0001, adj p = 0.001$ ), and control children ( $p < 0.0001, adj p < 0.0001$ ) (Fig. 1).

We did not find a correlation between age and hair cortisol among women or children. The correlation between hair cortisol levels in mothers and those in their children failed to reach significance ( $r = 0.262, p = 0.051$ ) however if we considered this a type II error, this marginal significance could suggest a transgenerational effect. In addition, we did not find a gender effect on hair cortisol among children ( $U = 438.50, p = 0.945$ ).

### 3.2. Differences in psychopathology and hair cortisol between IPV mothers and control mothers

Depression and PTSD symptoms severity was higher among IPV women in comparison with control women in the control group (Table 1). The number of sexual coercion events reported in the past year positively correlated with

depression symptoms among women ( $r = 0.483, p = 0.005, adj p = 0.01$ ). No correlations between hair cortisol levels, depression, PTSD and CTS2 chronicity scores were found within IPV mothers.

### 3.3. Differences in psychopathology and hair cortisol between IPV children and control children

Children who witnessed IPV reported more severe PTSD symptoms, but depressive symptoms were not statistically different than those in control children (Table 1). Moreover, the number of injury events positively correlated with the severity of PTSD ( $r = 0.388, p = 0.028, adj p = 0.056$ ) and depression ( $r = 0.350, p = 0.049, adj p = 0.098$ ) symptoms among IPV children. The chronicity of severe psychological aggression also correlated with child depression symptoms ( $r = 0.382, p = 0.0062, adj p = 0.024$ ). No correlations between hair cortisol levels, depression and PTSD were found within IPV children but the number of injury events reported by the mother and the level of hair cortisol among IPV children were associated ( $r = 0.388, p = 0.028$ ).

## 4. Discussion

The results of the current study highlight an association between IPV and higher levels of chronic cortisol in women exposed to such violence, but not in their children. An increase

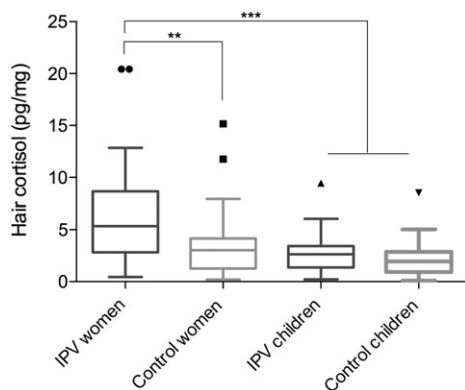


Fig. 1. Hair cortisol of intimate partner violence (IPV)–exposed women and their children. Bars represent median (mdn) with interquartile range (IQR). Women reporting IPV presented higher hair cortisol levels (mdn = 5.34 pg/mg, IQR = 5.84) within the last month before assessment compared to control women (mdn = 3.03 pg/mg, IQR = 2.9), IPV children (mdn = 2.64 pg/mg, IQR = 2.07) and control children (mdn = 1.94 pg/mg, IQR = 1.94) while there were no significant differences between both children groups ( $H = 26.24$ ;  $p < .001$ ). \*\*  $p = .003$ ; \*\*\*  $p < .001$ .

in cortisol related to intimate partner violence exposure was previously demonstrated [4,17,22]. However, to our knowledge, this is the first time that chronic HPA axis activity has been investigated in this specific population using hair cortisol assessment. This approach avoids the major limitations of traditional methods because it does not require baseline cortisol data and because circadian variations in cortisol levels do not affect hair cortisol concentration. In addition, hair cortisol measurement is a reliable method for retrospective longitudinal assessment of stress-related cortisol secretion (1-month assessment in 1-cm hair sample). Therefore, this study is innovative and has merit because it also investigated hair cortisol concentrations in mother–child dyads in the context of chronic stress exposure.

As previously mentioned, some studies have reported that women exposed to intimate partner violence present with higher cortisol levels (salivary, plasma) than non-exposed women, reflecting long-lasting changes in HPA axis functioning associated with stress exposure [22,46,47]. However, some evidence indicates that higher cortisol levels are found only in women who develop PTSD after chronic intimate partner violence exposure [22]. We found that women exposed to IPV and their children exhibited higher PTSD symptoms in comparison with controls. Hair cortisol levels had only a modest positive correlation with PTSD severity, in contrast with the expectation that PTSD would be associated with hypocortisolism [48,49]. These findings should be considered with caution because some studies did not find an association between PTSD symptoms and peripheral cortisol levels [18,21], while others corroborate our findings of increased cortisol within the context of intimate partner violence [17,50].

The absence of significant results when comparing hair cortisol levels in the children’s groups could be related to developmental periods of vulnerability or opportunities to

reverse negative effects [51,52]. Furthermore, we investigated children exposed to IPV from 6 to 12 years old, but a recent study showed a positive relationship between methylation of the children’s glucocorticoid receptor (GR) promoter and maternal exposure to IPV only if such exposure occurred prenatally, suggesting that HPA signaling could be affected differently across developmental stages [53].

In addition, we did not find an association between the hair cortisol levels of mothers and those of their children. This is in contrast to findings that showed a positive association between maternal and infant salivary cortisol levels in the context of domestic violence exposure [26]. However, in the previous study, positive salivary cortisol correlations in mother–infant dyads reporting intimate partner violence were only evident after a stress challenge task, while no associations were found when baseline cortisol levels were analyzed [26]. A previous study reported that intimate partner violence against women correlated positively with behavior problems and emotional distress in children [54], consistent with the results of our analysis of IPV.

Our results should be interpreted in light of some limitations. First, considering the relatively small sample size, we cannot exclude the lack of power to detect associations between some variables or the effects of false positive in some correlations that did not survive after Bonferroni correction. However, it is important to note that participant recruitment was very difficult because of study design (mother and child) and inclusion criteria. Our sample size is similar to that of other studies of this topic [48]. Second, we did not assess categorical psychiatric diagnoses. Therefore, we cannot exclude the effects of psychiatric comorbidities in our analysis. Third, the controls were selected based on not having psychiatric symptoms or any violence exposure. Fourth, we only assessed hair 1 cm from the scalp. Thus, our conclusions pertain to cortisol exposure within the 30 days before assessment. Fifth, we did not investigate the history of prenatal stress. However, some evidence shows that stress during pregnancy, particularly during the third trimester, can influence fetal programming of the HPA axis [53,55]. Hence, we cannot rule out potential transgenerational effects on the children’s HPA axis. Sixth, although hair cortisol is reportedly influenced by cumulative trauma [34,56], we did not investigate the history of stressful life events. This limitation should be considered when our data are interpreted. Seventh, we did not control for the time since the last IPV exposure or the duration of such exposure. However, all women reported having been exposed at least once in the 6 months before assessment.

## 5. Conclusions

The results of the present study indicate a pattern of increased levels of hair cortisol among women exposed to IPV, when compared to levels in women without such exposure. Although we did not find any hair cortisol differences among children from IPV-exposed women and controls, the number of injury events was related to hair cortisol levels, and these

children reported more PTSD symptoms than controls. Hence, given that experiencing and witnessing violence during childhood and adolescence are predictive of intimate partner violence in adulthood [57,58], and prospectively with child externalizing, internalizing, and total adjustment problems [59], interventions with couples, especially ones teaching conflict resolution, better coordination between services for women and for children, and parenting programs [60], are a crucial topic that needs to be considered with regard to domestic violence.

### Conflict of interest

The authors report no conflict of interest.

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### References

- [1] Pico-Alfonso MA, Garcia-Linares I, Celda-Navarro N, Blasco-Ros C, Echeburúa E, Martínez M. The impact of physical, psychological, and sexual intimate male partner violence on women's mental health: depressive symptoms, posttraumatic stress disorder, state anxiety, and suicide. *J Women's Health* 2006;15:599-611.
- [2] Devries KM, Mak JYT, García-Moreno C, Petzold M, Child JC, Falder G, et al. The global prevalence of intimate partner violence against women. *Science* 2013;27-8, <http://dx.doi.org/10.1126/science.1240937>.
- [3] Garcia-Moreno C, Jansen HAFM, Ellsberg M, Heise L, Watts CH. Prevalence of intimate partner violence: findings from the WHO multi-country study on women's health and domestic violence. *Lancet* 2006;368:1260-9, [http://dx.doi.org/10.1016/S0140-6736\(06\)69523-8](http://dx.doi.org/10.1016/S0140-6736(06)69523-8).
- [4] Johnson DM, Delahanty DL, Pinna K. The cortisol awakening response as a function of PTSD severity and abuse chronicity in sheltered battered women. *J Anxiety Disord* 2008;22:793-800, <http://dx.doi.org/10.1016/j.janxdis.2007.08.006>.
- [5] Martínez M, Garcia-Linares MI, Pico-Alfonso MA. Women victims of domestic violence: consequences for their health and the role of the health system. In: Renate CA, Klein RCA, & Wallner B, editors. *Gender, conflict, and violence*. Vienna: Studien-Verlag; 2003. p. 53-71.
- [6] Ellsberg M, Jansen HAFM, Heise L, Watts CH, García-moreno C, Study WHOM. Intimate partner violence and women's physical and mental health in the WHO multi-country study on women's health and domestic violence: an observational study. *Lancet* 2008;371:1165-72.
- [7] Campbell JC. Health consequences of intimate partner violence. *Lancet* 2002;359:1331-6, [http://dx.doi.org/10.1016/S0140-6736\(02\)08336-8](http://dx.doi.org/10.1016/S0140-6736(02)08336-8).
- [8] Straus MA, Hamby SL, Boney-McCoy S, Sugarman DB. The revised conflict tactics scales (CTS2) development and preliminary psychometric data. *Journal of family issues* 1996;17(3):283-316, <http://dx.doi.org/10.1177/019251396017003001>.
- [9] Dillon G, Hussain R, Loxton D, Rahman S. Mental and physical health and intimate partner violence against women: a review of the literature. *Int J Family Med* 2013;2013:313909, <http://dx.doi.org/10.1155/2013/313909>.
- [10] La Flair LN, Bradshaw CP, Campbell JC. Intimate partner violence/abuse and depressive symptoms among female health care workers: longitudinal findings. *Womens Health Issues* 2012;22:e53-9, <http://dx.doi.org/10.1016/j.whi.2011.07.001>.
- [11] Vives-Cases C, Ruiz-Cantero MT, Escribà-Agüir V, Miralles JJ. The effect of intimate partner violence and other forms of violence against women on health. *J Public Health (Oxf)* 2011;33:15-21, <http://dx.doi.org/10.1093/pubmed/fdq101>.
- [12] Sanchez-Lorente S, Blasco-Ros C, Martínez M. Factors that contribute or impede the physical health recovery of women exposed to intimate partner violence: a longitudinal study. *Womens Health Issues* 2012;22:491-500, <http://dx.doi.org/10.1016/j.whi.2012.07.003>.
- [13] Stewart DE, Vigod S, Riazantseva E. New developments in intimate partner violence and management of its mental health sequelae. *Current psychiatry reports* 2016;18(1):1-7, <http://dx.doi.org/10.1007/s11920-015-0644-3>.
- [14] Gunnar M, Quevedo K. The neurobiology of stress and development. *Annu Rev Psychol* 2007;58:145-73, <http://dx.doi.org/10.1146/annurev.psych.58.110405.085605>.
- [15] McEwen BS. The neurobiology of stress: from serendipity to clinical relevance. *Brain Res* 2000;886:172-89.
- [16] McEwen BS. Protective and damaging effects of stress mediators: central role of the brain. *Dialogues Clin Neurosci* 2006;8:367-81.
- [17] Pico-Alfonso MA, Garcia-Linares MI, Celda-Navarro N, Herbert J, Martínez M. Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence. *Biol Psychiatry* 2004;56:233-40, <http://dx.doi.org/10.1016/j.biopsych.2004.06.001>.
- [18] Seedat S, Stein MB, Kennedy CM, Hauger RL. Plasma cortisol and neuropeptide Y in female victims of intimate partner violence. *Psychoneuroendocrinology* 2003;28:796-808, [http://dx.doi.org/10.1016/S0306-4530\(02\)00086-0](http://dx.doi.org/10.1016/S0306-4530(02)00086-0).
- [19] McEwen BS. Understanding the potency of stressful early life experiences on brain and body function. *Metabolism* 2008;57(Suppl 2):S11-5, <http://dx.doi.org/10.1016/j.metabol.2008.07.006>.
- [20] Levine A, Zagoory-Sharon O, Feldman R, Lewis JG, Weller A. Measuring cortisol in human psychobiological studies. *Physiol Behav* 2007;90:43-53, <http://dx.doi.org/10.1016/j.physbeh.2006.08.025>.
- [21] Basu A, Levedosky AA, Lonstien JS. Trauma sequelae and cortisol levels in women exposed to intimate partner violence. *Psychodyn Psychiatry* 2013;41:247-75, <http://dx.doi.org/10.1521/pdps.2013.41.2.247>.
- [22] Inslicht SS, Marmar CR, Neylan TC, Metzler TJ, Hart SL, Otte C, et al. Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Y Acad Sci* 2006;1071:428-9, <http://dx.doi.org/10.1016/j.psyneuen.2006.03.007>.
- [23] Staufenbiel SM, Penninx BWJH, Spijker AT, Elzinga BM, van Rossum EFC. Hair cortisol, stress exposure, and mental health in humans: a systematic review. *Psychoneuroendocrinology* 2012, <http://dx.doi.org/10.1016/j.psyneuen.2012.11.015>.
- [24] Saltzman KM, Holden GW, Holahan CJ. The psychobiology of children exposed to marital violence. *J Clin Child Adolesc Psychol* 2005;34:129-39, [http://dx.doi.org/10.1207/s15374424jccp3401\\_12](http://dx.doi.org/10.1207/s15374424jccp3401_12).
- [25] Pendry P, Adam EK. Associations between parents' marital functioning, maternal parenting quality, maternal emotion and child cortisol levels. *Behav Dev* 2007;31:218-31, <http://dx.doi.org/10.1177/0165025407074634>.
- [26] Hibel LC, Granger DA, Blair C, Cox MJ. Intimate partner violence moderates the association between mother-infant adrenocortical activity across an emotional challenge. *J Fam Psychol* 2009;23:615-25, <http://dx.doi.org/10.1037/a0016323>.
- [27] Coria-Avila GA, Manzo J, Garcia LI, Carrillo P, Miquel M, Pfaus JG. Neurobiology of social attachments. *Neuroscience & Biobehavioral Reviews* 2014;43:173-82, <http://dx.doi.org/10.1016/j.neubiorev.2014.04.004>.
- [28] Stalder T, Steudte S, Alexander N, Miller R, Gao W, Dettenborn L, et al. Cortisol in hair, body mass index and stress-related measures. *Biol Psychol* 2012;90:218-23, <http://dx.doi.org/10.1016/j.biopsycho.2012.03.010>.
- [29] Van Uum SH, Sauv e B, Fraser LA, Morley-Forster P, Paul TL, Koren G. Elevated content of cortisol in hair of patients with severe chronic pain: a novel biomarker for stress. *Stress* 2008;11:483-8.

- [30] Stalder T, Kirschbaum C. Analysis of cortisol in hair – state of the art and future directions. *Brain Behav Immun* 2012;26:1019–29, <http://dx.doi.org/10.1016/j.bbi.2012.02.002>.
- [31] Kirschbaum C, Tietze A, Skoluda N, Dettenborn L. Hair as a retrospective calendar of cortisol production – increased cortisol incorporation into hair in the third trimester of pregnancy. *Psychoneuroendocrinology* 2009;34:32–7, <http://dx.doi.org/10.1016/j.psyneuen.2008.08.024>.
- [32] Karlén J, Ludvigsson J, Frostell A, Theodorsson E, Faresjö T. Cortisol in hair measured in young adults – a biomarker of major life stressors? *BMC Clin Pathol* 2011;11:12, <http://dx.doi.org/10.1186/1472-6890-11-12>.
- [33] Simmons JG, Badcock PB, Whittle SL, Byrne ML, Mundy L, Patton GC, et al. The lifetime experience of traumatic events is associated with hair cortisol concentrations in community-based children. *Psychoneuroendocrinology* 2016;63:276–81, <http://dx.doi.org/10.1016/j.psyneuen.2015.10.004>.
- [34] Grassi-Oliveira R, Pezzi JC, Daruy-Filho L, Viola TW, Francke IDA, Leite CE, et al. Hair cortisol and stressful life events retrospective assessment in crack cocaine users. *Am J Drug Alcohol Abuse* 2012;38:535–8, <http://dx.doi.org/10.3109/00952990.2012.694538>.
- [35] Groer MW, Kane B, Williams SN, Duffy A. Relationship of PTSD symptoms with combat exposure, stress, and inflammation in American soldiers. *Biol Res Nurs* 2015;17:303–10, <http://dx.doi.org/10.1177/1099800414544949>.
- [36] Dettenborn L, Tietze A, Kirschbaum C, Stalder T. The assessment of cortisol in human hair: associations with sociodemographic variables and potential confounders. *Stress* 2012;15:578–88, <http://dx.doi.org/10.3109/10253890.2012.654479>.
- [37] Vaghri Z, Guhn M, Weinberg J, Grunau RE, Yu W, Hertzman C. Hair cortisol reflects socio-economic factors and hair zinc in preschoolers. *Psychoneuroendocrinology* 2013;38:331–40, <http://dx.doi.org/10.1016/j.psyneuen.2012.06.009>.
- [38] Veldhorst MAB, Noppe G, Jongejan MHTM, Kok CBM, Mekic S, Koper JW, et al. Increased scalp hair cortisol concentrations in obese children. *J Clin Endocrinol Metab* 2014;99:285–90, <http://dx.doi.org/10.1210/jc.2013-2924>.
- [39] Moraes CL, Hasselmann MH, Reichenheim ME. Adaptação transcultural para o português do instrumento “Revised Conflict Tactics violência entre casais Portuguese-language cross-cultural adaptation of the Revised Conflict Tactics Scales (CTS2), an instrument used to identify violence in couples. *Cad Saude Publica* 2002;18:163–76.
- [40] Beck AT, Steer RA, Brown GK. *Manual for the Beck Depression Inventory-II*. San Antonio, Texas: Psychological Corporation; 1996.
- [41] Kovacs M. *Children’s depression inventory*. New York: Multi-Health System; 1992.
- [42] Foa EB, Johnson KM, Treadwell NCH, Kimberli RF. The Child PTSD Symptom Scale: a preliminary examination of its psychometric properties. *J Clin Child Adolesc Psychol* 2001;30:376–84.
- [43] Foa EB, Riggs DS, Dancu CV, Rothbaum BO. Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *J Trauma Stress* 1993;6:459–73.
- [44] Sauvé B, Koren G, Walsh G, Tokmakejian S, Van Uum SHM. Measurement of cortisol in human hair as a biomarker of systemic exposure. *Clin Invest Med* 2007;30:E183–91.
- [45] Ito N, Ito T, Kromminga A, Bettermann A, Takigawa M, Kees F, et al. Human hair follicles display a functional equivalent of the hypothalamic-pituitary-adrenal axis and synthesize cortisol. *FASEB J* 2005;19:1332–4, <http://dx.doi.org/10.1096/fj.04-1968fje>.
- [46] Bair-Merritt MH, Voegtline K, Ghazarian SR, Granger DA, Blair C, Johnson SB. Maternal intimate partner violence exposure, child cortisol reactivity and child asthma. *Child Abuse Negl* 2015;48:50–7, <http://dx.doi.org/10.1016/j.chiabu.2014.11.003>.
- [47] Bair-Merritt MH, Johnson SB, Okelo S, Page G. Intimate partner violence exposure, salivary cortisol, and childhood asthma. *Child Abuse Negl* 2012;36:596–601, <http://dx.doi.org/10.1016/j.chiabu.2011.12.002>.
- [48] Luo H, Hu X, Liu X, Ma X, Guo W, Qiu C, et al. Hair cortisol level as a biomarker for altered hypothalamic-pituitary-adrenal activity in female adolescents with posttraumatic stress disorder after the 2008 Wenchuan earthquake. *Biol Psychiatry* 2012;72:65–9, <http://dx.doi.org/10.1016/j.biopsych.2011.12.020>.
- [49] Steudte S, Kirschbaum C, Gao W, Alexander N, Schönfeld S, Hoyer J, et al. Hair cortisol as a biomarker of traumatization in healthy individuals and posttraumatic stress disorder patients. *Biol Psychiatry* 2013;74:639–46, <http://dx.doi.org/10.1016/j.biopsych.2013.03.011>.
- [50] Inslicht SS, Marmar CR, Neylan TC, Metzler TJ, Hart SL, Otte C, et al. Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Ann N Y Acad Sci* 2006;1071:428–9, <http://dx.doi.org/10.1196/annals.1364.035>.
- [51] Teicher MH, Andersen SL, Polcari A, Anderson CM, Navalta CP. Developmental neurobiology of childhood stress and trauma. *Psychiatr Clin North Am* 2002;25:397–426, [http://dx.doi.org/10.1016/S0193-953X\(01\)00003-X](http://dx.doi.org/10.1016/S0193-953X(01)00003-X).
- [52] Teicher MH, Samson JA. Childhood maltreatment and psychopathology: a case for ecophenotypic variants as clinically and neurobiologically distinct subtypes. *Am J Psychiatry* 2013;170:1114–33, <http://dx.doi.org/10.1176/appi.ajp.2013.12070957>.
- [53] Radtke KM, Ruf M, Gunter HM, Dohrmann K, Schauer M, Meyer A, et al. Transgenerational impact of intimate partner violence on methylation in the promoter of the glucocorticoid receptor. *Transl Psychiatry* 2011;1:e21, <http://dx.doi.org/10.1038/tp.2011.21>.
- [54] Spiller LC, Jouriles EN, McDonald R, Skopp NA. Physically abused women’s experiences of sexual victimization and their children’s disruptive behavior problems. *Psychol Violence* 2012;2:401–10, <http://dx.doi.org/10.1037/a0028912>.
- [55] Yehuda R, Engel SM, Brand SR, Seckl J, Marcus SM, Berkowitz GS. Transgenerational effects of posttraumatic stress disorder in babies of mothers exposed to the World Trade Center attacks during pregnancy. *J Clin Endocrinol Metab* 2005;90:4115–8, <http://dx.doi.org/10.1210/jc.2005-0550>.
- [56] Steudte S, Kolassa IT, Stalder T, Pfeiffer A, Kirschbaum C, Elbert T. Increased cortisol concentrations in hair of severely traumatized Ugandan individuals with PTSD. *Psychoneuroendocrinology* 2011;36:1193–200, <http://dx.doi.org/10.1016/j.psyneuen.2011.02.012>.
- [57] Smith-Marek EN, Cafferky B, Dharmidharka P, Mallory AB, Dominguez M, High J, Stith SM, Mendez M. Effects of Childhood Experiences of Family Violence on Adult Partner Violence: A Meta-Analytic Review. *Journal of Family Theory & Review* 2015;7(4):498–519.
- [58] Gomez AM. Testing the cycle of violence hypothesis: child abuse and adolescent dating violence as predictors of intimate partner violence in young adulthood. *Youth Soc* 2010;43:171–92, <http://dx.doi.org/10.1177/0044118X09358313>.
- [59] Vu NL, Jouriles EN, McDonald R, Rosenfield D. Children’s exposure to intimate partner violence: a meta-analysis of longitudinal associations with child adjustment problems. *Clinical psychology review* 2016;46:25–33, <http://dx.doi.org/10.3402/gha.v9.31516>.
- [60] Guedes A, Bott S, Garcia-Moreno C, Colombini M. Bridging the gaps: a global review of intersections of violence against women and violence against children. *Global health action* 2016;9, <http://dx.doi.org/10.3402/gha.v9.31516>.