Psychoneuroendocrinology 74 (2016) 57–64

Contents lists available at ScienceDirect

Psychoneuroendocrinology

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

Cortisol awakening response among women exposed to intimate partner violence

Ricardo J. Pinto [a,*], Patrícia Correia-Santos [a], Joana Costa-Leite [a], Alytia A. Levendosky [b], Inês Jongenelen [a]

[a] Faculty of Psychology, Education and Sports, University Lusófona of Porto, Rua Augusto Rosa, n° 24, Porto 4000-098, Portugal
[b] Psychology Department, Michigan State University, US, Psychology Building 316 Physics Rm 262 East Lansing, MI 48824, United States

ARTICLE INFO

Article history:
Received 21 March 2016
Received in revised form 20 August 2016
Accepted 24 August 2016

Keywords:
Cortisol awakening response
Intimate partner violence
Social support

ABSTRACT

The studies of the effects of intimate partner violence (IPV) on the cortisol awakening response (CAR) are scarce and contradictory. While some of the studies suggested that female victims of IPV showed high CAR, other studies found low CAR. Mixed results may be related to differences in sample characteristics as well as other potential covariates associated with the cortisol, as femaleís history of abuse, chronicity, severity and type of IPV, psychological distress, posttraumatic stress disorder, and social support. The study examined individual differences in CAR among 149 female victims of severe IPV reported to authorities, including 76 (51%) living in shelter and 73 (49%) living with the abusive partners. Results revealed several individual differences in CAR that may contribute to understanding the mixed results found in literature, including women with cortisol that decreased between the baseline and 30 min later, women with no increase of cortisol, and women whose cortisol increased above baseline. Additionally, women without CAR experienced more chronic and severe violence, more psychological distress and PTSD symptoms. However, hierarchical multiple regression indicated that chronic severe violence was the only independent variable that significantly explained 13% of the variance in CAR, even after including all covariates in the model, and adjusting for sociodemographic variables. In conclusion, this study suggests that the HPA axis dysregulation is influenced by chronic severe violence among women victims of IPV.

1. Introduction

The hypothalamic–pituitary–adrenal (HPA) axis functioning is an important stress response system, in which a cascade of physiological reactions in response to a stressful event leads to an increase in the secretion of cortisol, a steroid hormone with widespread effects on both body and brain (Sapolsky et al., 2000). Increases in cortisol levels mobilize energy and physiological resources towards addressing the stressor (Miller et al., 2007). Due to feedback loops in this system, cortisol peaks about 20–30 min after the stressor and then recovers to pre-stress levels by 41–60 min (Dickerson and Kemeny, 2004). While an acute stressor typically leads to a normal increase in cortisol activity (Dickerson and Kemeny, 2004), exposure to severe and repeated stressful events may lead to alterations of the normal HPA-axis (Miller et al., 2007; Yehuda, 2002). Dysregulation can be manifested in hyper or hyporeactivity but the findings are mixed regarding HPA axis functioning in individuals exposed to chronic distress or patients experiencing stress-related diseases (Chida and Steptoe, 2009; Fries et al., 2009; Kudielka et al., 2012; Miller et al., 2007). In the same way, research investigating cortisol alterations in victims of intimate partner violence (IPV) is also mixed. One possibility that may explain the mixed findings in literature is that chronic stress both increases and decreases HPA activity. Shortly after the stress has begun, the HPA axis may become activated, resulting in elevated cortisol output, but with the passage of time, when a state of exhaustion is reached, the cortisol output rebounds below normal due to the negative-feedback system of the HPA axis (Chida and Steptoe, 2009; Fries et al., 2005; Miller et al., 2007; Yehuda, 2002).

One way to assess the functioning of the HPA axis is through the cortisol awakening response (CAR). The cortisol awakening response is a marker of HPA axis function and for a well-regulated functioning is expected an increase in cortisol levels immediately following awakening, peaking approximately 30 min after awakening (Elder et al., 2014; Hucklebridge et al., 1998; Pruessner et al., 1997; Wüst et al., 2000b). In healthy adults salivary free cortisol

* Corresponding author.
E-mail address: ricardo.pinto@ulp.pt (R.J. Pinto).

http://dx.doi.org/10.1016/j.psyneuen.2016.08.024
0306-4530/

concentrations increase by between 50 and 160% in the first 30 min immediately post-awakening (Clow et al., 2004; Wüst et al., 2000b). A cortisol response can be defined as an increase of salivary cortisol levels of at least 2.5 nmol/l above individual baseline (Federenko et al., 2004; Huber et al., 2006; Oskis et al., 2009; Petrowski et al., 2010; Roberts et al., 2004; Rosmalen et al., 2005; Weitzman et al., 1971; Westenberg et al., 2009; Wüst et al., 2000b). CAR is believed to be a robust phenomenon and is used by most large-scale studies of stress and the HPA axis (Adam and Kumari, 2009), and is particularly appropriate for assessing HPA activation in relation to psychosocial factors (Chida and Steptoe, 2009), because it is not significantly impacted by many of the confounding variables (e.g., age, oral contraceptive use, habitual smoking, time of awakening, sleep duration, sleep quality, physical activity, or morning routines) that may impact other indexes of HPA axis functioning, such as diurnal or basal cortisol (Pruessner et al., 1997; Schmidt-Reinwald et al., 1999; Wüst et al., 2000a; Johnson et al., 2008).

There are currently five published studies of the effects of IPV on CAR. While, overall these studies suggested that severity and chronicity of the IPV, and PTSD are associated with the CAR, the results are mixed. For instance, Kim et al. (2015), with a community sample of 122 couples, found that women with higher levels of physical IPV, compared to women with lower levels, had significantly lower CAR. In addition, Suglia et al. (2010) found that cumulative stress was associated with lower level of CAR among pregnant female victims of IPV. Johnson et al. (2008), in a sample of 52 sheltered battered women, showed that PTSD severity was associated with significantly greater CAR, while abuse chronicity was associated with lower CAR. More recently, the same researchers (Pinna et al., 2014), in a sample of 104 abused battered women in shelters, found that women who had experienced IPV with PTSD showed significantly greater CAR compared to those without PTSD. In addition, the authors found that CAR was higher in women with PTSD plus comorbid depression compared to women with neither PTSD nor depression. Conversely, Basu et al. (2013) found that CAR did not distinguish female victims of IPV with and without PTSD and depression.

In addition, a review of the extant research on cortisol and IPV, including studies of other types of cortisol measurement, such as plasma cortisol, and other times of assessment, such as diurnal cortisol suggests that the IPV exposure and PTSD have effects on HPA-axis regulation; however, the results between studies are inconsistent. For example, Seedat et al. (2003), using plasma cortisol collected in the morning, found that women who were victims of physical IPV showed lower mean cortisol levels compared to women who were not victims. Conversely, another study found no differences between female victims of IPV and a matched group in the mean morning levels of cortisol (Pico-Alfonso et al., 2004). Yet another study found that in pregnant women from the community, those who reported IPV presented higher levels of salivary cortisol (Valladares et al., 2009). Furthermore, regarding the studies that included female victims of IPV, with and without PTSD, the results are also contradictory. For instance, while one study found that women with lifetime PTSD had significantly higher cortisol levels across the day compared to abuse-exposed participants without PTSD (Inslicht et al., 2006), another study (Griffin et al., 2005) found that women victims of IPV with PTSD showed lower early morning plasma cortisol levels than battered women without PTSD or normal healthy controls.

There are some important methodological differences between these studies that may explain the mixed findings. For example, considering the five published studies of the effects of IPV on salivary CAR, the samples were quite different: two collected data from community samples with small to moderate levels of violence, and PTSD was not assessed (Kim et al., 2015; Suglia et al., 2010), while two assessed PTSD but they only included women in domestic violence shelters (Johnson et al., 2008; Pinna et al., 2014), and one required women to meet criteria for PTSD and/or depression (Basu et al., 2013). Furthermore, there are methodological issues that were not addressed. None of these studies included women living with the abusive partner. Additionally, CAR was used as a dependent variable rather than examining potential individual differences in CAR and how that may function with PTSD or psychological distress. Finally, social support a known key protective factor of IPV (Carlson et al., 2002; Coker et al., 2003), which also affects the stress response (Cohen and Wills, 1985; Heinrichs et al., 2003; Uchino et al., 1996) was not examined in these studies and it may explain the conflicting findings.

The present study fits within the research on cortisol related to IPV in the attempt to explore reasons for previous conflicting findings by looking at individual differences in CAR among women victims of severe IPV reported to authorities. In general, CAR research often investigates group differences (e.g., between clinical patients and control subjects) (Stalder et al., 2016) instead of explore individual differences within groups. Further, we aimed to examine whether individual differences in CAR were associated with some potential covariates of CAR (Kudielka et al., 2009), including the womanís history of abuse, chronicity and severity of the IPV, psychological distress and PTSD, and social support, after adjusting for sociodemographic variables. Additionally, potential differences in CAR between women living with the abusive partner and women living in shelters were examined. Given the variability of the previous findings, the present study is exploratory and hypotheses were not developed.

2. Material and methods

2.1. Participants

In order to recruit the sample, 260 institutions that provide anonymous assistance to women victims of partner violence, including shelter residences, were contacted. Recruitments of women took place in the Portuguese Association for Victim Support (APAV), Child Protective Services, Domestic Violence Organizations and Shelter Residences from north to south of Portugal. One hundred and seventeen institutions agreed to collaborate in the study. The professionals of these institutions made the first contact with the participants, a general explanation of the study was provided to them, and asked if they agree to participate in the study. In total, 352 women were contacted and 160 women, obtained from 35 institutions, agreed to participate. For the present study, seven women were excluded due to insufficient saliva samples and four have delayed the collection of the first saliva sample over 15 min. The sample of the present study included 149 women, which 73 (49%) were living with the aggressors and 76 (51%) in shelters. The participants' ages ranged from 21 to 54 years old ($M = 36.38$, $SD = 7.57$). In terms of marital status, 31 (20.8%) women were single, 54 (36.2%) married, 35 (23.5%) civil union, 28 (18.8%) divorced or separated, and one (0.7%) widowed. In terms of education, 22 (14.8%) participants had only 4 years, 57 (38.3%) had completed 6 years, 53 (35.6%) had completed 9 years, 13 (8.7%) participants had completed the full compulsory education of 12 years, and 4 (2.5%) participants had obtained university degrees. The majority of women were unemployed ($n = 108$, 72.5%), 36 (24.2%) was employed, and five (3.4%) never worked. The time of the IPV self-reported by participants ranged from five months to 29 years ($M = 10.43$; $SD = 5.74$). For sheltered women, the time in shelter ranged from one to nine months ($M = 2.76$; SD = 2.09).

The inclusion criteria were as follows: over 18 years old (civil majority), have children between four and ten years old, and either to have reported the partner's violence to authorities, such as the

police department and/or other institutions for victim support, or to have entered into shelter residences. Exclusionary criteria were psychotherapeutic support, apparent psychosis, intoxication, pregnancy, or mental retardation to ensure that informed consent could be given.

2.2. Procedure

The present study is part of a larger research project funded by Fundação para a Ciência e Tecnologia (Foundation for Science and Technology – Portuguese and European funding) on the impact of IPV on women and childrenís health carried out in Portugal. The study was approved by the National Commission for Data Protection (NCDP) and the ethics committees of both the University of Porto and the University Lusófona of Porto granted approval for this study. Prior informed written consent was obtained from all participants. Participants received vouchers from a local department store as a courtesy for participating in the study. The initial contact with the institutions was made by email and then followed by telephone, where a face-to-face meeting was scheduled to present the study. The first contact with the participants was made by the professionals of the institutions and a general explanation of the purposes of the study, methods, and procedures was provided to them. After the participants agreed to participate, the researchers scheduled the interviews, in which more detailed information about the study and the informed consent was given. The questionnaires were administered in an interview format by trained female psychologists either in the institutions or in the shelters in a calm and private room. The saliva collection was schedule for the next morning after the interviews and all sample collection procedures were explained verbally as well as in a brochure that was handed to all participants. The first saliva sample was collected as soon as the subject wake up but while still in bed. Participants were instructed to fill in a record sheet their awakening and sampling times. They were also instructed not to brush their teeth before completing saliva sampling to avoid contamination of saliva with blood caused by micro-injuries in the oral cavity. They were also instructed not to eat and smoking during the sampling period. Besides these restrictions, subjects were free to follow their normal daily routines. The salivette devices were delivered to women in identified bags with labels of the time 0 and 30 min to help them to identify the correct order of saliva collection. After saliva collection, subjects stored the salivette devices in their freezers to be collected at the end of the morning by one researcher, including the record sheets. The salivettes were transported in mobile freezer and delivered to the laboratory (ENDOCLAB – Labco Group Portugal) for analysis.

2.3. Measures

2.3.1. Salivary cortisol

Cortisol Awakening Response (CAR). Early morning saliva samples were collected at home or in shelters. We sent two text messages to participants, one in the previous day, and the other in the early morning of the day, to remind the saliva collection. Using Salivette sampling device without citric acid (Sarstedt, Rommelsdorf, Germany), participants obtained saliva samples immediately upon awakening and 30 min later on a single day. We used self-report sampling method of recording awaking and sampling collection times. CAR was measured by subtracting the 30 min post-awakening sample from the wake up sample. To identify inaccurate data, the delay between awakening and initiation of sampling was calculated. The extent of inaccuracy was calculated as the discrepancy between the reported awakening time and the first sample collection after awakening. In the cases of discrepancy have exceeded the margin of 15 min, CAR data for the respective sampling were excluded from subsequent analyses. Delays greater than 15 min between awakening and commencement of saliva sampling reduce CAR magnitude (Smyth et al., 2015, 2013). The time of the first sample collection after awakening ranged from 0 to 15 min ($M = 1$, $SD = 3$). The total sampling error was not significant correlated to the CAR, $F(1, 147) = 1.21$, $p = 0.26$.

2.3.2. Sociodemographics

A questionnaire was used to collect information about age, gender, marital status, occupation, education, number of family members, including children, living in shelter vs. aggressor, time in the shelter, time of abuse exposure, history and current medical problems and medication use, information about alcohol, drugs and smoking habits.

2.3.3. Psychological distress

The Brief Symptom Inventory questionnaire (BSI) (Derogatis, 1982; Portuguese version of Canavarro, 1999) is a short form of the SCL-90-R and includes 53 self-report items that evaluate psychological distress. Subjects describe whether they have experienced any distress symptoms over the past seven days on a 5-point scale (0 = *not at all*; 4 = *extremely*) and, if so, how they were affected by these symptoms. The inventory includes nine symptom dimensions: somatization, obsessive compulsivity, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism. We used the Global Severity Index as a continuous variable which higher scores reflects more distress. The cut-off score for clinical cases in the Portuguese adaptation was 1.7 on the Positive Symptom Distress Index (PSDI). The BSI internal consistency for the present sample was 0.96 for the overall items.

2.3.4. Posttraumatic stress disorder

The PTSD Checklist–Civilian Version (PCL-C) (Weathers et al., 1994; Portuguese version of Marcelino and Gonçalves, 2012) is a Checklist that includes 17 self-report items of symptoms of posttraumatic stress disorder based on DSM-IV B, C, and D criteria. It requires participants to rate the severity of each symptom during the previous 30 days on a Likert-type scale ranging from 1 (*not at all*) to 5 (*extremely*). The DSM-IV criteria for PTSD were met when a participant reported a moderate or higher level of at least one intrusion symptom, three avoidance symptoms, and two hyperarousal symptoms. In this study, participants were instructed to respond while thinking about the exposure to actual or threatened death or serious injury to meet DSM-IV A1 criteria. We also assessed DSM-IV A2 criteria by asking to participants if they recalled feeling terrified or helpless at the time of exposure. We also used a total PTSD score based on the sum of all symptoms. The internal consistency for the present sample was 0.89.

2.3.5. Social support

Social Provisions Scale (Cutrona and Russell, 1987; Portuguese version of Moreira and Canaipa, 2007) is a scale that includes 24 self-report items to assess the degree to which respondent's social relationships provide various dimensions of social support. The respondent indicates on a 4-point scale the extent to which each statement describes her current social network. Responses range from 1 (*strongly disagree*) to 4 (*strongly agree*).The instrument contains six subscales as following: attachment, social integration, reassurance of worth, reliable alliance, guidance, and opportunity for nurturance. After reversal of negatively worded items, a total score may be computed by summing all items. A high score indicates a greater degree of perceived support. The internal consistency for the present sample was 0.95.

2.3.6. Physical and psychological IPV victimization

The Revised Conflict Tactics Scales (CTS2; Straus et al., 1996; Portuguese version by Paiva and Figueiredo, 2006) was used to

measure the chronicity of women's physical assault and psychological aggression on an eight-point scale (0 = *this has never happened* to 7 = *More than 20 times in the past year*). Respondents reported on the frequency of abusive behaviors perpetrated by their current or most recent abusive partner within the previous 12 months. The chronicity was calculated by determining the midpoint of the items as follows: "This has never happened" = 0; "Once in the past year" = 1; "Twice in the past year" = 2; "3–5 times in the past year" = 4; "6–10 times in the past year" = 8; "11–20 times in the past year" = 15; "More than 20 times in the past year" = 25. These mid points were then summed to obtain psychological aggression and physical assault subscales (Straus, 2001). To differentiate the severity level of the violence, chronicity was also calculated by separating the items of minor and severe violence. In the present study, the subscales presented good reliability: psychological aggression ($\alpha = 0.78$) and physical assault ($\alpha = 0.89$) subscales, as well as for the subscales for two levels of severity, as minor ($\alpha = 0.86$) and severity ($\alpha = 0.85$).

2.3.7. Femaleís history of abuse and neglect

Adverse Childhood Experiences Study Questionnaire (ACE: Felitti et al., 1998; Portuguese version of Pinto et al., 2014). The questionnaire included detailed information on 10 adverse childhood experiences, organized into two areas: children's experiences and household dysfunction. The five categories of children's experiences included emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect. Responses range from 0 (*never*) to 5 (*very often*), with the exception of sexual abuse, for which a dichotomous response (*yes or no*) was given. The evaluation of household dysfunction included questions about whether the mother was treated violently, household substance abuse, mental illness or suicide in the family, parental separation or divorce and incarcerated household members. Responses of mother treated violently range from 0 (*never*) to 5 (*very often*), and (*yes or no*) for the other last four categories of household dysfunction. All items for the 10 different examples of childhood adversity were dichotomized based on how often the experiences occurred (see Felitti et al., 1998). We then computed a total score of the adverse experiences exposure for each subject ranged from zero to 10.

2.4. Statistics

Data analyses were carried out using the SPSS version 20 for Windows (United States, New York, IBM Corporation, 2011). We calculated the effect sizes by post hoc power analyses using GPower 3.1 (Faul et al., 2009) and by Lakens (2013). We classified three groups of CAR, including no CAR, low CAR, and CAR increase. The no CAR group (n = 66) contained women with cortisol that decreased between the baseline and 30 min later, and women with no increase of cortisol (showed the same levels of cortisol at baseline and 30 min after awakening). The low CAR group (n = 27) included women whose cortisol increased but not enough to reach the 2.5 nmol/l above baseline. The CAR increase group (n = 60) comprised all women with an increase of cortisol levels of at least 2.5 nmol/l above baseline (Weitzman et al., 1971). Then, we used one-way analysis of variance (ANOVA) to compare these three groups in terms of the study variables: Womenís history of maltreatment, IPV victimization, PTSD total symptoms, psychological distress, and perceived social support. We followed this up by analysis of covariance (ANCOVA) in order to test whether or not these effects remained when covariates were considered (study variables, age and education). We used chi-square analyses to test associations between CAR and women living with the aggressor and living in shelter. We also made this comparison using the independent samples *t*-test with CAR as a continuous variable. Additionally, we used the total score of the ACE to assess the femaleís history of abuse, and the chronicity from the CTS2, a continuous variable, to assess psychological aggression and physical assault exposure. The PTSD symptoms as a continuous variable did not approximate normality, and log transformations were used to bring large scores closer to the center to obtain normal distribution. Hierarchical multiple regression analyses assessed the independent contributions of the womenís history of maltreatment, IPV victimization, PTSD total symptoms, psychological distress, and perceived social support to predicting the linear changes of CAR between the baseline and 30 min later, after adjusting for sociodemographic variables. We used hierarchical regression analysis to handle potential covariates related to CAR (Stalder et al., 2016). Two cases with standardized residuals below 3 and two cases above 3 were removed (Field, 2013). We began with a base model consisting of the sociodemographic variables, including age, education, and location (shelter versus living with the aggressor). In the second block, the total score of womanís history of abuse was entered. The total scores of minor and severe violence were added in the third block. We then entered the total scores of psychological distress, posttraumatic stress symptoms, and perception of social support in the fourth block. We used the total scores of the key variables to reduce the number of variables to be included in the equation and therefore increasing the parsimonious of the regression model. Missing data occurred in one case (0.7%) for the total score of distress symptoms and the total score of womenís history of abuse, and two cases (1.3%) for PTSD as a continuous variable.

Table 1
Means and Standard Deviations of Key Measures.

Variables	*M*	*SD*	*Min*	*Max*
Global psychological distress symptoms	1.54	0.73	0.02	3.02
Total PTSD symptoms	51.42	12.83	17	70
Cortisol	18.76	7.11	5.66	40.28
Adverse childhood experiences	4.61	2.40	0	10
Physical assault	117.56	82.55	0	265
Psychological aggression	126.54	59.91	0	200
Total social support perception	66.72	11.46	30	94

Note: *N* = [147–149]. PTSD = posttraumatic stress disorder. Cortisol = Total post-awakening cortisol levels – nmol/l. Physical and psychological aggression = chronicity of the violence exposure.

3. Results

3.1. Descriptive

Table 1 provides the descriptive statistics of the key variables. Regarding the descriptive statistics of the cortisol at baseline, 30 min later, and CAR (see Table 2), in 60 (40.3%) women, the cortisol decreased between the first sample collection, immediately after awakening, and 30 min later; in 5 (3.4%) women, the cortisol released was the same for both moments; in 27 (18.1%) women, the cortisol increased but was not enough to reach the 2.5 nmol/l above baseline; and finally, in 57 (38.3%) women, the cortisol increased of at least 2.5 nmol/l above individual baseline. According to these results of CAR, we classified three groups, including no CAR, low CAR, and CAR increase. We included in the same group the women with the cortisol decreased after awakening (no CAR) with the five participants with the same post-awakening cortisol because they had no increase in cortisol, compared to the other two groups. Then, we compared these three groups of CAR with individual difference characteristics of the women, including womenís history of abuse, chronicity of the violence exposure (physical and psychological aggression), global psychological distress symptoms, total PTSD symptoms, and social support. We repeated the analyses without the five participants with the same cortisol released for both moments and the results were the same.

Table 2
Descriptive Statistics of Cortisol Awakening Response.

Subgroups	n	%	CAR				Baseline				30 min later			
		Z	*M*	*SD*	*Min*	*Max*	*M*	*SD*	*Min*	*Max*	*M*	*SD*	*Min*	*Max*
Cortisol decreased after awakening	60	40.3	−4.51	4.48	−20.69	−0.28	19.62	7.46	8	38.63	15.11	6.62	3.86	32.56
Same post-awakening cortisol levels	5	3.4	0	0	0	0	13.62	4.03	9.38	19.86	13.46	4.03	9.38	19.86
Low CAR[a]	27[a]	18.1	1.31	0.69	0.28	2.48	16.72	5.27	5.52	28.42	18.03	5.32	5.79	29.25
CAR increase[b]	57[b]	38.3	8.20	4.77	2.76	24	17.25	6.79	4.97	34.76	25.45	9.24	8.83	51.87

Note: CAR = nmol/l.
[a] The cortisol increased but was not enough to reach the 2.5 nmol/l above baseline.
[b] The cortisol increased of at least 2.5 nmol/l above individual baseline.

3.2. The relationship between CAR and study variables

We did not find significant associations between CAR groups and women living with abusive partner and women living in shelter, $\chi(2) = 1.59$, $p = 0.45$. In addition, we did not find significant differences between women living with abusive partner and women living in shelter in CAR means, $t(147) = 0.62$, $p = 0.53$, CI [−1.55, 2.98], $d = 0.10$. A one-way analysis of variance (ANOVA) found no differences between CAR and womenís history of abuse, $F(2,144) = 3.00$, $p = 0.076$, partial $\eta^2 = 0.040$, Cohen's $f = 0.20$. However, there were significant differences between CAR and type of IPV. Physical aggression differed between groups, $F(2,146) = 5.67$, $p = 0.004$, partial $\eta^2 = 0.072$, Cohen's $f = 0.28$. Hochberg post-hoc comparisons revealed that the group no CAR experienced more physical aggression ($M = 138.29$, $SD = 79.47$) than the group CAR increase ($M = 90.19$, $SD = 78.31$). This effect remained significant in an ANCOVA after controlling for the effect of covariates, $F(2,133) = 3.20$, $p = 0.044$, partial $\eta^2 = 0.046$, Cohen's f = 0.22. In addition, psychological aggression differed between groups, $F(2,146) = 6.78$, $p = 0.002$, partial $\eta^2 = 0.085$, Cohen's $f = 0.30$. Hochberg post-hoc comparisons revealed that the group no CAR experienced more psychological aggression ($M = 141.62$, $SD = 53.16$) than the group CAR increase ($M = 104.54$, $SD = 63.99$). This effect remained significant in an ANCOVA adjusting for covariates, $F(2,133) = 3.22$, $p = 0.043$, partial $\eta^2 = 0.046$, Cohen's f = 0.22. There were significant differences between CAR groups and PTSD symptoms, $F(2,140) = 4.12$, $p = 0.018$, partial $\eta^2 = 0.056$, Cohen's $f = 0.24$. Hochberg post-hoc comparisons revealed more PTSD symptoms in the group no CAR ($M = 54.74$, $SD = 12.13$) than the group CAR increase ($M = 48.22$, $SD = 11.80$). This effect remained significant in an ANCOVA adjusting for covariates, $F(2,131) = 5.04$, $p = 0.008$, partial $\eta^2 = 0.071$, Cohen's f = 0.28. There were also significant differences between CAR groups and global distress symptoms, $F(2,145) = 4.44$, $p = 0.013$, partial $\eta^2 = 0.058$, Cohen's $f = 0.25$. Hochberg post-hoc comparisons revealed more psychological distress in the group no CAR ($M = 1.74$, $SD = 0.74$) than the group CAR increase ($M = 1.37$, $SD = 0.73$). This effect remained significant in an ANCOVA adjusting for covariates, $F(2,131) = 4.67$, $p = 0.011$, partial $\eta^2 = 0.066$, Cohen's f = 0.27. Finally, there were differences between CAR and total perception of social support, $F(2,142) = 4.63$, $p = 0.011$, partial $\eta^2 = 0.061$, Cohen's $f = 0.25$. Hochberg post-hoc comparisons revealed more perception of social support in the group CAR increase ($M = 69.81$, $SD = 10.85$) than the group no CAR ($M = 63.71$, $SD = 11.44$). This effect was no longer significant when adjusted for the effects of covariates, $F(2,131) = 0.88$, $p = 0.42$, partial $\eta^2 = 0.013$, Cohen's f = 0.11.

The results of hierarchical multiple regression analysis with CAR as a continuous variable are shown in Table 3. The first block, including age, education, location, did not significantly contribute to the regression model, $F(3, 139) = 0.56$, $p = 0.65$. Adding the womenís history of abuse and neglect in the second block, the model remained statistically no significant, $F(4, 138) = 1.71$, $p = 0.15$. The two levels of severity, minor and severe violence, were entered in the third block, and the model became statistically significant, $F(6, 136) = 4.81$, $p < 0.001$, Cohen's $f^2 = 0.16$, contributing an additional 13% to the explained variance. Here, only severe violence was significantly associated with the outcome variable. Finally, the PTSD total symptoms, psychological distress, and perceived social support were added in the final step, and the model remained significant, $F(9, 133) = 3.41$, $p < 0.01$, but did not add significant additional variance to the model. Again, chronic exposure to severe violence remained as the only significant predictor in the final model.

Table 3
Hierarchical Regression Analyses with CAR as Outcome Continuous Variable.

Model[a]	B	β	*t*
Step 1: $R^2 = 0.01$			
Age	0.03	0.03	0.36
Location (shelter or home)	−1.34	−0.11	−1.28
Education	0.04	0.02	0.21
Step 2: $R^2 = 0.05$; $\Delta R^2 = 0.04^{*}$			
Womenís history of abuse	−0.58	−0.19	−2.27[*]
Step 3: $R^2 = 0.18^{***}$; $\Delta R^2 = 0.13^{***}$			
Chronicity of minor violence	−0.04	−0.03	−0.14
Chronicity of severe violence	−0.55	−0.39	−2.26[***]
Step 4: $R^2 = 0.19^{**}$; $\Delta R^2 = 0.01$			
Constant	7.99		
Age	0.01	0.01	0.01
Location (shelter or home)	−2.09	−0.17	−1.85
Education	0.06	0.03	0.31
Womenís history of abuse	0.11	0.04	0.36
Chronicity of minor violence	−0.03	−0.02	−0.11
Chronicity of severe violence	−0.53	−0.38	−2.12[*]
PTSD total symptoms	0.35	0.05	0.50
Psychological distress	−5.26	−0.12	−1.25
Social support	0.57	0.07	0.65

Note: $N = 154$.
[a] Only Steps 1 and 4 show the complete model for that step. Other steps show only new variables. Womenís history of adversity = Total score of childhood adversity. IPV victimization = Total score of both psychological and physical violence. PTSD = Total score of PTSD symptoms. Psychological distress = Global Severity Index. Social support = Total score of social support perception.
[*] $p < 0.05$.
[**] $p < 0.01$.
[***] $p < 0.001$, two-tailed.

4. Discussion

The present study explored individual differences in CAR among women victims of severe IPV reported to authorities to contribute for the explanation of conflicting findings in literature. In addition, we aimed to examine whether individual differences in CAR were associated with other variables that may act as confounders between the relationship of IPV exposure and CAR, including the woman's history of abuse, chronicity, severity and type of IPV, psychological distress and PTSD, and social support. Finally, we compared women living with the abusive partner and women in IPV shelters in terms of CAR differences.

We found the presence of a wide range of individual differences in CAR among women exposed to IPV, despite all having been exposed to severe violence reported to authorities. This heterogeneity in CAR, suggesting individual differences in CAR in women exposed to IPV may help explain prior mixed findings in the literature. In addition, the heterogeneity in CAR found here supports the idea that the HPA axis is a highly adaptive system characterized by marked variability within and between individuals (Kudielka et al., 2012).

We also found that the CAR group without any morning increase seems the most impaired, showing more levels of physical and psychological aggression, PTSD and psychological distress symptoms. This finding is consistent with the hypothesis that for a well-regulated functioning is expected an increase in cortisol levels immediately following awakening (Clow et al., 2004; Elder et al., 2014; Hucklebridge et al., 1998; Pruessner et al., 1997; Wüst et al., 2000b).

However, we also found that only the chronicity of severe violence independently accounted for the variance in CAR when modelled as a continuous variable. The lack of variance explained by potential covariates between the violence exposure and CAR contrasts with prior findings that chronic stress and PTSD symptoms may have a differential impact on HPA axis functioning (e.g., Johnson et al., 2008). Our findings suggest that in our sample of women identified by authorities, the chronicity of severe violence have an impact on HPA axis functioning that is not influenced by other factors, including the chronicity of minor violence and social support.

High levels of chronic exposure to severe violence were significantly associated to low levels of CAR, which is consistent with previous studies that found lower levels of CAR associated with higher levels of physical violence (Kim et al., 2015), chronicity of abuse (Johnson et al., 2008), and cumulative stress (Suglia et al., 2010), including the studies that used other cortisol measures and found lower CAR levels in female victims of IPV (Griffin et al., 2005; Seedat et al., 2003). These findings are consistent with the hypocortisolism hypothesis that suggests the failure of HPA axis system to respond and recovery after chronic exposure to stress, increasing the sensitivity of the negative-feedback system of the HPA (Fries et al., 2005; Miller et al., 2007; Yehuda, 2002). In normal conditions, under the "flight-or-fight" response to acute stress, a counter-regulatory response occurs to contain the stress response when it is no longer needed, following the onset of a stressor, in which cortisol return to baseline levels. However, chronic exposure to stress can lead to the HPA axis dysregulation, as low cortisol secretion (McEwen 2007; Raison and Miller, 2003), which some researchers believe that this process may signify an adaptive form of dissociation or inhibition of the psychological experience of threat (Sturge-Apple et al., 2012) and to ensure long-term survival by preventing high cortisol levels from negative effects on body and brain (Fries et al., 2005; McEwen 2007).

A few limitations must be considered when interpreting our results. This is a cross-sectional study. On the interpretation of the relationship between CAR and other variables, such as psychological distress, PTSD, and perception of social support, the study's cross-sectional design does not allow determination of causality between variables. In addition, the conclusion that there is no associations between covariates and CAR needs to be tempered with the fact that despite the advantage in looking at the individual differences in both women in IPV shelters as well as those living with the abusive partner, the absence of a control group limits our findings due to the low between-subject variability. The scales for PTSD and psychological distress are symptom checklists, rather than diagnostic measures. Therefore, conclusions about diagnoses of mental disorders and comorbidity cannot be drawn. In addition, the conclusions of this study are limited to CAR and other aspects of the HPA axis may respond differently to chronic and severe stress. Salivary cortisol measurements cannot perfectly reflect the complexity of the entire system, which is one conceptual reason why inconsistent findings are found in literature (Kudielka et al., 2012). Further, although protocols with only two post-awakening samples can provide a general approximation of the CAR, they cannot be sure to capture the CAR peak (Stalder et al., 2016), considering that CAR peak can be extended to the period of 30–45 min post-awakening (Smyth et al., 2015), and thus we may have missed some increases in cortisol that occurred after the second sample collection. Therefore, the recent recommendations for CAR research on adult populations are the use of protocols with a minimum of three sampling points: awakening, 30 min and 45 min (Stalder et al., 2016) over multiple days, and we used two-sample protocol in only a single day. Although the use of a two-sample protocol (0 min and 30 min) may be justifiable, the data may be difficult to interpret as it remains unknown whether potential relationships are seen with CAR magnitude or differential CAR peak timing (Stalder et al., 2016). Finally, we did not use electronic monitoring devices to verify sampling accuracy. Therefore, the verification provided that the sample collection occurred correctly was only based on self-reports, which are subject to human error.

In conclusion, this study contributes to our knowledge on the impact of severe IPV on CAR. The women with no CAR seems the most impaired, since they experienced more physical and psychological aggression, had higher levels of psychological distress and PTSD symptoms. Our findings also suggest the presence of individual differences in CAR among women exposed to severe IPV. The predictor of this differential HPA activity seems to be chronicity of the severe violence. However, our findings need to be replicated in a study with multiple time sampling points of CAR and over multiple days.

Conflict of interest

Authors have no conflicts of interest.

Role of the funding source

This study was funded by the Fundação para a Ciência e Tecnologia (research grant, FCT – EXPL MHC PED 1977/2013). The sponsor had no involvement in study design, data collection, analysis, and interpretation, article preparation, or the decision to submit the article for publication.

Contributors

All authors contributed substantially to the conception and design of the study. J.C.L. and P.C.S. collected the data. R.P., I.J. and A.L. analyzed and interpreted the data. R.J.P. wrote the current version of the manuscript with critical revision of I.J. and A.L. All authors contributed to its critical revision. All authors have approved the final version of the article.

Acknowledgements

The authors express appreciation to the Fundação para a Ciência e Tecnologia (research grant, FCT – EXPL MHC PED 1977/2013) for financing this project. The authors also express appreciation to Portuguese Association for Victim Support (APAV), Child Protective Services (CPCJ), Domestic Violence Organizations and Shelter Residences from Portugal.

References

Adam, E.K., Kumari, M., 2009. Assessing salivary cortisol in large-scale, epidemiological research. Psychoneuroendocrinology 34, 1423–1436, http://dx.doi.org/10.1016/j.psyneuen.2009.06.011.

Basu, A., Levendosky, A.A., Lonstein, J.S., 2013. Trauma sequelae and cortisol levels in women exposed to intimate partner violence. Psychodyn. Psychiatry 41, 247–275, http://dx.doi.org/10.1521/pdps.2013.41.2.247.

Canavarro, C., 1999. Inventário de sintomas psicopatológicos-B.S.I. [Brief Symptom Inventory], in: Simões, M.R., Gonçalves, M.M. (Eds.), Testes e provas psicológicas em Portugal Vol. 2. Braga, Portugal : APPORT/SHO, pp. 95–109.

Carlson, B.E., McNutt, L.A., Choi, D.Y., Rose, I.M., 2002. Intimate partner abuse and mental health the role of social support and other protective factors. Violence Against Women 8, 720–745, http://dx.doi.org/10.1177/10778010222183251.

Chida, Y., Steptoe, A., 2009. Cortisol awakening response and psychosocial factors: a systematic review and meta-analysis. Biol. Psychol. 80, 265–278, http://dx.doi.org/10.1177/10778010222183251.

Clow, A., Thorn, L., Evans, P., Hucklebridge, F., 2004. The awakening cortisol response: methodological issues and significance. Stress 7, 29–37, http://dx.doi.org/10.1080/10253890410001667205.

Cohen, S., Wills, T.A., 1985. Stress, social support, and the buffering hypothesis. Psychol. Bull. 98, 310–357.

Coker, A.L., Watkins, K.W., Smith, P.H., Brandt, H.M., 2003. Social support reduces the impact of partner violence on health: application of structural equation models. Prev. Med. 37, 259–267, http://dx.doi.org/10.1016/S0091-7435(03)00122-1.

Cutrona, C.E., Russell, D.W., 1987. The provisions of social relationships and adaptation to stress. Adv. Personal Relat. 1, 37–67.

Derogatis, L.R., 1982. BSI: Brief Symptom Inventory. National Computers Systems, Minneapolis, MN.

Dickerson, S.S., Kemeny, M.E., 2004. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol. Bull. 130, 355–391, http://dx.doi.org/10.1037/0033-2909.130.3.355.

Elder, G.J., Wetherell, M.A., Barclay, N.L., Ellis, J.G., 2014. The cortisol awakening response – Applications and implications for sleep medicine. Sleep Med. Rev., http://dx.doi.org/10.1016/j.smrv.2013.05.001.

Faul, F., Erdfelder, E., Buchner, A., Lang, A.G., 2009. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. Behav. Res. Methods 41, 1149–1160, http://dx.doi.org/10.3758/BRM.41.4.1149.

Federenko, I., Wüst, S., Hellhammer, D.H., Dechoux, R., Kumsta, R., Kirschbaum, C., 2004. Free cortisol awakening responses are influenced by awakening time. Psychoneuroendocrinology 29, 174–184, http://dx.doi.org/10.1016/S0306-4530(03)00021-0.

Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., Koss, M.K., Marks, J.S., 1998. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. Am. J. Prev. Med. 14, 245–258, http://dx.doi.org/10.1016/S0749-3797(98)00017-8.

Field, A., 2013. Discovering Statistics Using IBM SPSS Statistics, fourth ed. SAGE.

Fries, E., Hesse, J., Hellhammer, J., Hellhammer, D.H., 2005. A new view on hypocortisolism. Psychoneuroendocrinology 30, 1010–1016, http://dx.doi.org/10.1016/j.psyneuen.2005.04.006.

Fries, E., Dettenborn, L., Kirschbaum, C., 2009. The cortisol awakening response (CAR): facts and future directions. Int. J. Psychophysiol. 72, 67–73, http://dx.doi.org/10.1016/j.ijpsycho.2008.03.014.

Griffin, M.G., Resick, P.A., Yehuda, R., 2005. Enhanced cortisol suppression following dexamethasone administration in domestic violence survivors. Am. J. Psychiatry 162, 1192–1199, http://dx.doi.org/10.1176/appi.ajp.162.6.1192.

Heinrichs, M., Baumgartner, T., Kirschbaum, C., Ehlert, U., 2003. Social support and oxytocin interact to suppress cortisol and subjective responses to psychosocial stress. Biol. Psychiatry 54, 1389–1398, http://dx.doi.org/10.1016/S0006-3223(03)00465-7.

Huber, T.J., Issa, K., Schik, G., Wolf, O.T., 2006. The cortisol awakening response is blunted in psychotherapy inpatients suffering from depression. Psychoneuroendocrinology 31, 900–904, http://dx.doi.org/10.1016/j.psyneuen.2006.03.005.

Hucklebridge, F., Sen, S., Evans, P.D., Clow, A., 1998. The relationship between circadian patterns of salivary cortisol and endogenous inhibitor of monoamine oxidase A. Life Sci. 62, 2321–2328, http://dx.doi.org/10.1016/S0024-3205(98)00213-6.

IBM Corp., 2011. IBM SPSS Statistics for Windows (Version 20.0) [Computer Software]. Armonk, NY.

Inslicht, S.S., Marmar, C.R., Neylan, T.C., Metzler, T.J., Hart, S.L., Otte, C., McCaslin, S.E., Larkin, G.L., Hyman, K.B., Baum, A., 2006. Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. Psychoneuroendocrinology 31, 825–838, http://dx.doi.org/10.1016/j.psyneuen.2006.03.007.

Johnson, D.M., Delahanty, D.L., Pinna, K., 2008. The cortisol awakening response as a function of PTSD severity and abuse chronicity in sheltered battered women. J. Anxiety Disord. 22, 793–800, http://dx.doi.org/10.1016/j.janxdis.2007.08.006.

Kim, H.K., Tiberio, S.S., Capaldi, D.M., Shortt, J.W., Squires, E.C., Snodgrass, J.J., 2015. Intimate partner violence and diurnal cortisol patterns in couples. Psychoneuroendocrinology 51, 35–46, http://dx.doi.org/10.1016/j.psyneuen.2014.09.013.

Kudielka, B.M., Hellhammer, D.H., Wüst, S., 2009. Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. Psychoneuroendocrinology 34, 2–18, http://dx.doi.org/10.1016/j.psyneuen.2008.10.004.

Kudielka, B.M., Gierens, A., Hellhammer, D.H., Wüst, S., Schlotz, W., 2012. Salivary cortisol in ambulatory assessment-some dos, some don'ts, and some open questions. Psychosom. Med. 74, 418–431, http://dx.doi.org/10.1097/psy.0b013e31825434c7.

Lakens, D., 2013. Calculating and reporting effect sizes to facilitate cumulative science: a practical primer for *t*-tests and ANOVAs. Front. Psychol. 4, 1–12, http://dx.doi.org/10.3389/fpsyg.2013.00863.

Marcelino, D., Gonçalves, S.P., 2012. Perturbação pós-stress traumático: características psicométricas da versão portuguesa da Posttraumatic Stress Disorder Checklist—Civilian Version (PCL-C). Rev. Port. Saude Publ. 30, 71–75.

McEwen, B.S., 2007. Physiology and neurobiology of stress and adaptation: central role of the brain. Physiol. Rev. 87, 873–904, http://dx.doi.org/10.1152/physrev.00041.2006.

Miller, G.E., Chen, E., Zhou, E.S., 2007. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. Psychol. Bull. 133, 25–45, http://dx.doi.org/10.1037/0033-2909.133.1.25.

Moreira, J.M., Canaipa, R., 2007. A Escala de Provisões Sociais: desenvolvimento e validação da versão portuguesa da Social Provisions Scale. Rev. Iberoam. Diag. Ev. 24, 23–58.

Oskis, A., Loveday, C., Hucklebridge, F., Thorn, L., Clow, A., 2009. Diurnal patterns of salivary cortisol across the adolescent period in healthy females. Psychoneuroendocrinology 34, 307–316, http://dx.doi.org/10.1016/j.psyneuen.2008.09.009.

Paiva, C.A., Figueiredo, B., 2006. Versão portuguesa das Escalas de Táticas de Conflito Revisadas: estudo de validação. Psicol. Teor. Prat. 8, 14–39.

Petrowski, K., Herold, U., Joraschky, P., Wittchen, H.U., Kirschbaum, C., 2010. A striking pattern of cortisol non-responsiveness to psychosocial stress in patients with panic disorder with concurrent normal cortisol awakening responses. Psychoneuroendocrinology 35, 414–421, http://dx.doi.org/10.1016/j.psyneuen.2009.08.003.

Pico-Alfonso, M.A., Garcia-Linares, M.I., Celda-Navarro, N., Herbert, J., Martinez, M., 2004. Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence. Biol. Psychiatry 56, 233–240, http://dx.doi.org/10.1016/j.biopsych.2004.06.001.

Pinna, K.L., Johnson, D.M., Delahanty, D.L., 2014. PTSD, comorbid depression, and the cortisol waking response in victims of intimate partner violence: preliminary evidence. Anxiety Stress Coping 27, 253–269, http://dx.doi.org/10.1080/10615806.2013.852185.

Pinto, R., Correia, L., Maia, Â., 2014. Assessing the reliability of retrospective reports of adverse childhood experiences among adolescents with documented childhood maltreatment. J. Fam. Violence 29, 431–438, http://dx.doi.org/10.1007/s10896-014-9602-9.

Pruessner, J.C., Wolf, O.T., Hellhammer, D.H., Buske-Kirschbaum, A.B., Von Auer, K., Jobst, S., Kaspers, F., Kirschbaum, C., 1997. Free cortisol levels after awakening: a reliable biological marker for the assessment of adrenocortical activity. Life Sci. 61, 2539–2549, http://dx.doi.org/10.1016/S0024-3205(97)01008-4.

Raison, C.L., Miller, A.H., 2003. When not enough is too much: the role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. Am. J. Psychiatry, http://dx.doi.org/10.1176/appi.ajp.160.9.1554.

Roberts, A.D.L., Wessely, S., Chalder, T., Papadopoulos, A., Cleare, A.J., 2004. Salivary cortisol response to awakening in chronic fatigue syndrome. Br. J. Psychiatry 184, 136–141, http://dx.doi.org/10.1192/bjp.184.2.136.

Rosmalen, J.G.M., Oldehinkel, A.J., Ormel, J., De Winter, A.F., Buitelaar, J.K., Verhulst, F.C., 2005. Determinants of salivary cortisol levels in 10–12 year old children; A population-based study of individual differences. Psychoneuroendocrinology 30, 483–495, http://dx.doi.org/10.1016/j.psyneuen.2004.12.007.

Sapolsky, R.M., Romero, M., Munck, A., 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr. Rev. 21, 55–89, http://dx.doi.org/10.1210/edrv.21.1.0389.

Schmidt-Reinwald, A., Pruessner, J.C., Hellhammer, D.H., Federenko, I., Rohleder, N., Schurmeyer, T.H., Kirschbaum, C., 1999. The cortisol response to awakening in relation to different challenge tests and a 12-h cortisol rhythm. Life Sci. 64, 1653–1660, http://dx.doi.org/10.1016/S0024-3205(99)00103-4.

Seedat, S., Stein, M.B., Kennedy, C.M., Hauger, R.L., 2003. Plasma cortisol and neuropeptide Y in female victims of intimate partner violence. Psychoneuroendocrinology 28, 796–808, http://dx.doi.org/10.1016/S0306-4530(02)00086-0.

Smyth, N., Clow, A., Thorn, L., Hucklebridge, F., Evans, P., 2013. Delays of 5–15 min between awakening and the start of saliva sampling matter in assessment of the cortisol awakening response. Psychoneuroendocrinology 38, 1476–1483, http://dx.doi.org/10.1016/j.psyneuen.2012.12.013.

Smyth, N., Thorn, L., Hucklebridge, F., Evans, P., Clow, A., 2015. Detailed time course of the cortisol awakening response in healthy participants. Psychoneuroendocrinology 62, 200–203, http://dx.doi.org/10.1016/j.psyneuen.2015.08.011.

Stalder, T., Kirschbaum, C., Kudielka, B.M., Adam, E.K., Pruessner, J.C., Wüst, S., Dockray, S., Smyth, N., Evans, P., Hellhammer, D.H., Miller, R., Wetherell, M.A., Lupien, S.J., Clow, A., 2016. Invited review: assessment of the cortisol awakening response: expert consensus guidelines. Psychoneuroendocrinology 63, 414–432.

Straus, M.A., Hamby, S.L., Boney-McCoy, S., Sugarman, D.B., 1996. The revised conflict tactics scales (CTS2) development and preliminary psychometric data. J. Fam. Issues 17, 283–316, http://dx.doi.org/10.1177/019251396017003001.

Straus, M.A., 2001. Scoring and norms for the CTS2 and CTSPC. Paper no. CTS28, (available online at murraystraus@unh.edu).
Sturge-Apple, M.L., Davies, P.T., Cicchetti, D., Manning, L.G., 2012. Interparental violence, maternal emotional unavailability and children"s cortisol functioning in family contexts. Dev. Psychol. 48, 237–249, http://dx.doi.org/10.1037/a0025419.
Suglia, S.F., Staudenmayer, J., Cohen, S., Enlow, M.B., Rich-Edwards, J.W., Wright, R.J., 2010. Cumulative stress and cortisol disruption among Black and Hispanic pregnant women in an urban cohort. Psychol. Trauma Theory Res. Pract. Policy 2, 326–334, http://dx.doi.org/10.1037/a0018953.
Uchino, B.N., Cacioppo, J.T., Kiecolt-Glaser, J.K., 1996. The relationship between social support and physiological processes: a review with emphasis on underlying mechanisms and implications for health. Psychol. Bull. 119, 488, http://dx.doi.org/10.1037/0033-2909.119.3.488.
Valladares, E., Peña, R., Ellsberg, M., Persson Å, L., Högberg, U., 2009. Neuroendocrine response to violence during pregnancy–impact on duration of pregnancy and fetal growth. Acta Obstet. Gynecol. Scand. 88, 818–823.
Wüst, S., Federenko, I., Hellhammer, D.H., Kirschbaum, C., 2000a. Genetic factors, perceived chronic stress, and the free cortisol response to awakening. Psychoneuroendocrinology 25, 707–720, http://dx.doi.org/10.1016/S0306-4530(00)00021-4.
Wüst, S., Wolf, J., Hellhammer, D.H., Federenko, I., Schommer, N., Kirschbaum, C., 2000b. The cortisol awakening response-normal values and confounds. Noise Health 2, 79–88.
Weathers, F.W., Litz, B.T., Herman, D., Huska, J., Keane, T., 1994. The PTSD Checklist-civilian Version (PCL-C). National Center for PTSD, Boston, MA.
Weitzman, E.D., Fukushima, D., Nogeire, C., Roffwarg, H., Gallagher, T.F., Hellman, L., 1971. Twenty-four hour pattern of the episodic secretion of cortisol in normal subjects. J. Clin. Endocrinol. Metab. 33, 14–22.
Westenberg, P.M., Bokhorst, C.L., Miers, A.C., Sumter, S.R., Kallen, V.L., van Pelt, J., Blöte, A.W., 2009. A prepared speech in front of a pre-recorded audience: subjective, physiological, and neuroendocrine responses to the Leiden Public Speaking Task. Biol. Psychol. 82, 116–124, http://dx.doi.org/10.1016/j.biopsycho.2009.06.005.
Yehuda, R., 2002. Current status of cortisol findings in post-traumatic stress disorder. Psychiatr. Clin. North Am. 25, 341–368, http://dx.doi.org/10.1056/NEJMra012941.