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Cortisol awakening response among women exposed to intimate partner violence

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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

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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