

# Physiological Stress Responses Predict Sexual Functioning and Satisfaction Differently in Women Who Have and Have Not Been Sexually Abused in Childhood

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Physiological responses to sexual stimuli may contribute to the increased rate of sexual problems seen in women with childhood sexual abuse (CSA) histories. We compared two physiological stress responses as predictors of sexual function and satisfaction (sympathetic nervous system [SNS] activation and cortisol) in women with CSA ( $N = 136$ ) and without CSA histories (NSA,  $N = 102$ ). In CSA survivors, cortisol response to sexual stimuli did not significantly predict sexual functioning; however, in NSA women, cortisol increases were associated with poorer sexual functioning, and cortisol decreases with higher sexual functioning. For women with CSA histories, lower SNS activity was associated with poorer sexual functioning. For CSA survivors with low lifetime trauma, lower SNS activity was associated with higher sexual satisfaction; for women with high lifetime trauma, the reverse was true. Decreased SNS activity during sexual stimuli predicted higher sexual functioning in NSA women with low lifetime exposure to traumatic events, but lower sexual functioning in those with high exposure. Differences between women with and without CSA histories in the association between cortisol and SNS response and sexual functioning and satisfaction suggest that CSA causes disruptions in both short- and long-term stress responses to sexual stimuli that perpetuate into adulthood.

*Keywords:* childhood sexual abuse, sexual functioning, sexual satisfaction, heart rate variability, cortisol

Childhood sexual abuse (CSA) has been linked to sexual problems in adulthood, with prevalence estimates of sexual dysfunction ranging from 85% in community samples (Gorcey, Santiago, & McCall-Perez, 1986) to 94% in clinical samples (Jehu, 1988). Specifically, women with CSA histories are significantly more likely to report sexual aversion or avoidance, hypo- or hyperactive sexual desire, inhibited sexual arousal or orgasm, sexual pain, negative attitudes toward sexuality and intimacy, and difficulty establishing satisfying sexual relationships (for reviews, see Bartoi & Kinder, 1998; DiLillo, 2001; Van Berlo & Ensinck, 2000). Though most of the proposed mechanisms through which CSA may affect sexual functioning and satisfaction have been psychological (Polusny & Follette, 1995), there may also be physiological factors. Specifically, it is possible that, for women with CSA histories, sexual problems are related to disturbances in physiological stress-response systems. Two likely candidates are the sympathetic nervous system (SNS), a short-term stress response, and cortisol, a longer-term response.

## Short-Term Stress Response: Sympathetic Nervous System Activity

During a stressor, the SNS activates systems across the body to increase glucose availability, heart rate, and blood pressure (Bremner, Krystal, Southwick, & Charney, 1996). Shortly after a normal stressor, these physiological functions return to resting equilibrium. After a traumatic stressor, however, baseline SNS activity can become increased (Yehuda, 2003). Among sexually healthy women, increasing SNS activity to a moderate degree facilitates genital sexual arousal, whereas high levels of SNS activation inhibit genital arousal (Lorenz, Harte, Hamilton, & Meston, 2012; Meston & Gorzalka, 1996). Exercise-induced activation of the SNS increases genital arousal in sexually functional women but not among women with a history of CSA (Rellini & Meston, 2006). The authors suggested that as women with CSA histories have high baseline SNS (Shalev & Rogel-Fuchs, 1993), additional SNS activity from exercise pushed the stress system beyond the levels which facilitate sexual arousal. We would expect, based on these findings, that better sexual function may be associated with higher SNS activation to sexual stimuli in women without CSA histories, but lower SNS activity, or even withdrawal, in women with CSA histories.

Some studies in trauma survivors have found blunted SNS response to stressors relative to controls (Tan, Dao, Farmer, Sutherland, & Gevirtz, 2011); others found increased SNS responsivity (Keary, Hughes, & Palmieri, 2009). This may be explained by degree of trauma exposure: Individuals exposed to a single trauma may show exaggerated SNS stress responses, but those with exposure to multiple trauma may respond with

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blunted SNS reactivity (Yehuda & LeDoux, 2007). While this phenomenon is confounded by symptoms of posttraumatic stress disorder (PTSD), there is reason to believe that the impact of early trauma on stress reactivity is similar in survivors with and without PTSD, if controlling for the number of trauma exposures (Kobayashi, 2010; Scheeringa, Zeanah, Myers, & Putnam, 2004).

Most previous research in this area has been limited by indirect measurement of SNS via increases in heart rate (HR), which may be due to increasing SNS activation, or withdrawal of the parasympathetic nervous system (PNS). A more accurate assessment of SNS activity is heart-rate variability (HRV). Both SNS and PNS act on the heart: The PNS sets frequency of heartbeats (i.e., HR), and the SNS signals each individual contraction. During inspiration, PNS cardiac output is briefly disrupted, allowing the lungs and heart to coordinate respiration and circulation. These interruptions of PNS control by respiration cause tiny fluctuations of the interval between heartbeats, a phenomenon known as respiratory sinus arrhythmia. However, when the SNS is dominant, its influence on individual contractions of the heart become more prominent in the HRV signal.

One simple way to analyze HRV is to measure the standard deviation of beat-to-beat intervals (R wave to R wave), also known as the RRSD. As the RRSD (i.e., total HRV) decreases, SNS activity is thought to be increasing, relative to PNS output (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). The RRSD has good validity, and is noninvasive, easy to interpret, and easy to calculate from a 3-lead electrocardiogram (EKG) or heart rate monitor. The RRSD is often used clinically to diagnose or predict heart disease (Tsuji et al., 1996), hypertension (Terathongkum & Pickler, 2004), and depression risk in heart attack survivors (Carney et al., 2001). In the present study, we measured changes in HRV as a precise index of SNS activity in women with and without CSA histories, and with low or high lifetime exposure to trauma.

### Long-Term Stress Response: Cortisol

Cortisol is a hormone that mediates the body's long-term stress response. Cortisol released in response to stressors increases blood sugar levels through suppression of insulin. In normal situations, cortisol allows the body to focus energy on the stressor and returns to baseline after a recovery period of 60 to 90 minutes (Kirschbaum & Hellhammer, 1994); however, following trauma, baseline cortisol levels as well as frequency and amount of release are altered (Southwick, Yehuda, & Morgan, 1995). Women reporting CSA have lower baseline levels of cortisol (Bremner, Vermetten, & Kelley, 2007; King, Mandansky, King, Fletcher, & Brewer, 2001), but higher levels of cortisol released during stress exposure (Elzinga, Schmahl, Vermetten, van Dyck, & Bremner, 2003). As with SNS activity, there is some evidence that number of trauma exposures modulate cortisol responsivity, such that a single traumatic exposure is associated with an exaggerated response, and multiple traumas with a blunted response (Pierrehumbert et al., 2009; Yehuda, Halligan, Golier, Grossman, & Bierer, 2004). This tendency may be exaggerated for sexual trauma (Resnick, Yehuda, Pitman, & Foy, 1995).

In women never sexually abused (NSA), cortisol generally decreases in response to sexual stimuli. The degree of cortisol decrease in response to sexual stimuli is associated with sexual desire (Hamilton, Rellini, & Meston, 2008; Van Anders, Brotto, Farrell, & Yule, 2009). Although genital arousal is associated with decreases in cortisol (Exton et al., 2000; Hamilton & Meston, 2011; Van Anders, et al., 2009), there is limited evidence that *subjective* sexual arousal is tied to cortisol, with most studies reporting either no significant association (Goldey & van Anders, 2010; Heiman, Rowland, Hatch, & Gladue, 1991) or associations only to perceptions of genital sensations (Van Anders, et al., 2009). However, cortisol may play a role in other emotional responses to sexual stimuli. Increased cortisol during sexual stimuli is associated with sexual anxiety (Heiman et al., 1991) and lower sexual satisfaction (Hamilton et al., 2008). In sum, for NSA women, cortisol release in response to sexual stimuli appears to interfere with physical sexual response and, to a lesser extent, emotional response.

Only one study has examined the relation between cortisol and sexual response in women with a history of CSA. Rellini, Hamilton, Delville and Meston (2009) compared changes in genital sexual arousal and cortisol during a sexual video in women with and without a history of CSA. The authors found that women with a history of CSA had smaller decreases, and in some cases increases, in cortisol to sexual stimuli relative to the NSA group. In the CSA group only, cortisol increase during sexual stimuli was associated with higher perceived sexual arousal, suggesting overlap between of the sensation of sexual arousal and stress in this population. However, genital arousal was recorded from a device inserted in the vagina, which may have selected out participants with strong sexual aversion. Women with CSA histories may be less likely to volunteer for studies involving vaginal photoplethysmography than other assessments (Wolchik, Spencer, & Lisi, 1983). Nevertheless, based on this study and the literature demonstrating cortisol abnormalities, we expected that cortisol release would not predict sexual functioning and satisfaction in women with CSA histories.

### Hypotheses

We hypothesized that physiological responses to sexual stimuli would differentially predict sexual function and satisfaction in women with and without CSA histories. Specifically, we predicted that decreases in SNS activation to sexual stimuli would predict higher sexual functioning and satisfaction in women with CSA histories, particularly those with low lifetime exposure to traumatic events. We predicted that for women without CSA histories, increases in SNS activation would predict better sexual functioning and satisfaction. We also hypothesized that decreases in cortisol would predict sexual functioning and satisfaction in NSA women but not in women with CSA histories. As age has been associated with sexual functioning (Avis et al., 2009; Laumann, Paik, & Rosen, 1999), we were interested in the predicted effects above and beyond age effects.

### Method

#### Participants

Participants were recruited via local newspapers and websites, and screened over the phone. All participants were required to be

over 18 years of age and sexually active. Women were excluded if they had experienced a traumatic event in the previous three months, been a victim of sexual abuse in the past two years, or had been diagnosed with a psychotic disorder in the previous six months. Participants with other mental health diagnoses were included if they reported no significant suicidality. Women taking corticosteroids, medications known to affect cardiovascular and/or cortisol function (e.g., beta blockers), or illicit drugs were also excluded. Participants taking other medications were included if they had been stabilized on that dose for at least three months.

**CSA group.** CSA women experienced at least one involuntary sexual experience prior to age 16 (and no fewer than 2 years prior) that included one or more of the following acts: Oral, anal, or vaginal intercourse, penetration of the vagina or anus using objects or digits, and genital touching or fondling. The CSA group ( $N = 136$ ) was of an average age of 34.2 ( $SD = 10.4$ ), predominantly Caucasian (66%), with some Latina (19%) and African American (10%) participants. The majority had at least some college education (72%) and were in a committed relationship or married (70%; see Table 1). Most of the women in the CSA group reported at least one incident of forced oral, anal, or vaginal intercourse (84%); a minority reported only sexual touching (13%); and the majority reported multiple incidents of unwanted sexual contact (93%).

**NSA group.** Women in the NSA group reported no incidents of sexual or physical abuse in childhood and no unwanted sexual experiences in adulthood. Comparable to the CSA group, the NSA

group ( $N = 102$ ) was of an average age of 32.7 ( $SD = 11.5$ ), mostly Caucasian (69%), with some Latina (11%) and African American (11%) participants, and a majority had at least some college education (71%). Forty-seven percent of women in the NSA group were in a committed relationship or married (see Table 1).

There were no statistically significant differences between groups in age, ethnicity or level of education. Although women in the CSA group were significantly more likely to be married than women in the NSA group, analyses did not change when controlling for type of relationship and thus results are reported without controlling for relationship type.

## Materials: Physiological Assessments

**Heart-rate variability (HRV).** Before stimulus presentation, we attached electrodes to participants using a 3-limb lead site paradigm based on Einthoven's triangle. The EKG signal was recorded using AcqKnowledge (Biopac Systems, Inc, Goleta, CA). After the experimental session, the EKG signal across neutral and sexual stimuli was cleaned for artifacts (e.g., electrodes becoming loose during the session). We collected RR intervals with a peak finder function, and calculated RRSD from these data with Kubios HRV Analysis Software (Biosignal Analysis and Medical Imagine Group, University of Kuopio, Kuopio, Finland).

**Salivary assays for cortisol.** We collected two saliva samples per participant, representing cortisol reactivity to the neutral

Table 1  
*Demographics and Participant Characteristics for CSA and NSA Samples*

	CSA ( $N = 136$ )		NSA ( $N = 102$ )		$\chi^2$
	n	%	n	%	
Education					
Less than high school/GED	3	2	2	2	2
Completed high school/GED	18	13	11	11	
Some college	98	70	72	69	
College degree or greater	17	12	17	16	
Data missing	4	3	2	2	
Relationship status					
Single, not dating	23	17	24	23	42.4*
Single, dating	14	10	28	27	
In a committed relationship	55	40	36	35	
Married	42	30	12	12	
Data missing	6	3	4	3	
Ethnicity					
Caucasian	90	66	70	68	9.8
Hispanic/Latina	26	19	11	11	
African-American or Black	14	10	11	11	
Asian-American	5	4	7	7	
Other	1	1	3	3	
Sexual orientation					
Heterosexual	50	35	50	48	10.25
Primarily heterosexual	63	46	44	42	
Bisexual	10	7	1	1	
Primarily homosexual	7	5	4	4	
Homosexual	2	1	2	2	
Data missing	8	5	3	3	

*Note.* The table includes raw values of physiological variables, not marginal means; tests for differences between groups reported in text controlled for age, depression, and lifetime history of trauma exposure.

\*  $p < .05$ .

stimulus and the sexual stimulus. We used Starstedt salivettes with untreated cotton swabs. Samples were frozen until processed, centrifuged at 3,500 rpm for a 10-min period, and analyzed with kits from Salimetrics (State College, PA). This kit restricts cross-reactivities for noncortisol-endogenous-steroid hormones to 1% or less. Interassay variability was 8.7% at 1.048  $\mu\text{g}/\text{dL}$  and 12.3% at .104  $\mu\text{g}/\text{dL}$  and the intraassay variability was 2.87%.

## Psychological Assessments

**Sexual functioning.** We measured sexual functioning using a modified version of the Female Sexual Function Index (FSFI; Rosen et al., 2000), a validated 19-item questionnaire with six domains supported by factor analysis: desire, arousal, lubrication, orgasm, satisfaction, and pain. We removed exclusive references to penile-vaginal penetration to capture nonheterosexual relationships. The FSFI reliably discriminates between women with and without sexual dysfunction (Meston, 2003; Rosen et al., 2000). The FSFI had excellent reliability (Cronbach's  $\alpha = .95$ ) in this sample.

**Sexual satisfaction.** We measured sexual satisfaction with the Sexual Satisfaction Scale for Women (SSS-W; Meston & Trapnell, 2005). The SSS-W is a validated 30-item questionnaire with five domains supported by factor analyses: comfort discussing sexual issues (communication), compatibility between partners (compatibility), sexual contentment (contentment), personal distress concerning sexual problems (intrapersonal distress), and distress about the impact of sexual problems on one's partner or relationship (interpersonal distress). The SSS-W reliably differentiates between women with and without sexual dysfunction (Meston & Trapnell, 2005). The SSS-W also had excellent reliability in this sample (Cronbach's  $\alpha = .93$ ).

**Lifetime exposure to trauma.** We measured lifetime exposure to traumatic events with the Trauma History Questionnaire (THQ; Green, 1996), a 24-item questionnaire that assesses exposure to crime, general trauma, natural disasters, and physical or sexual assault. Each item is assessed dichotomously (experienced/not experienced) as well as by frequency (how often it occurred). We calculated the sum of all traumatic events reported, which is the most commonly used metric of total trauma exposure (Green, Krupnick, Rowland, Epstein, & Stockton, 2005).

**Depression.** Depression is common in CSA survivors (Putnam, 2003). As depression is linked to sexual dysfunction in both women with CSA histories (Beitchman et al., 1992) and without (Kennedy, Dickens, Eisfeld, & Bagby, 1999), we measured current depression symptoms with the Beck Depression Inventory II (BDI-II; Beck, Steer, & Brown, 1996). This 21-item depression questionnaire has been extensively validated (Beck, Steer, & Carbin, 1988), and is considered the gold standard self-report measure of depression. Symptoms of depression were significantly higher in women with CSA histories than those without,  $F(1, 186) = 30.22$ ,  $p < .001$ , and thus we controlled for depression in all analyses that compared these two groups.

## Procedure

To control for daily fluctuations in cortisol and monthly fluctuations of other hormones, participants completed experimental sessions between 2:00 and 6:00 p.m., between the 5th and 10th

days of their menstrual cycles. To reduce artifacts in the salivary assays, participants were instructed to avoid foods and activities known to affect cortisol (e.g., caffeine, exercise) for at least 12 hours prior to their visit, and chewing gum, food, or drink for two hours prior to their visit. Compliance with these procedures was assessed by the experimenter immediately prior to the experimental session; only data from compliant participants were included in further analyses involving physiological data. Thirty-four participants (10 NSA, 24 CSA) were found noncompliant. Excluded participants did not differ significantly from those included in CSA history, demographics, sexual functioning, or satisfaction.

Participants were instructed to write continuously on each of two topics for 20 minutes: First an objective account of what they did in the previous day (neutral stimuli), and then about sex and their own sexuality (sexual stimuli; see <http://bit.ly/wKzXT8> for prompt scripts). The neutral essay has been used as a control condition in several writing studies and has no known effects on psychological function (Pennebaker, 1989). The sexual essay was selected to create a personal connection to the sexual stimuli. Rather than attempting to elicit only sexual arousal, we aimed to trigger women's general response to sexually relevant information, including but not limited to their sexual arousal. We anticipated that for the NSA women, this essay would elicit sexual response associated with positive affect, while for women with CSA histories, it would elicit a sexual response associated with negative affect. In other words, we expected the stimuli would reflect true population differences in responses to sexual information.

As per the guidelines provided by Pennebaker (1997), participants were encouraged to write continuously and to ignore spelling, grammar, and punctuation. A language-use analysis indicated that participants used significantly more sexuality-related words in the sexual essays than neutral essays, confirming the validity of our experimental manipulation (Lorenz & Meston, in press).

After completing the neutral essay, participants filled out non-stressful questionnaires (e.g., demographics) for 20 minutes. Because there is an approximate 20-minute delay between peak cortisol release from the adrenal glands and peak salivary cortisol levels (Elzinga et al., 2004), the neutral sample was collected 20 minutes after the neutral stimuli presentation, while the sexual response sample was collected 20 minutes after the sexual stimuli presentation. Electrocardiograph information was collected throughout the experimental session. To ensure confidentiality, participants typed both essays alone in a private room, and were instructed to close the file before the experimenter returned. Participants were debriefed at the end of the experimental session and compensated. The University of Texas at Austin Institutional Review Board approved the study from 2004–2011).

## Results

### Group Differences in Sexual Function and Satisfaction

To test for differences between women with and without CSA histories in sexual functioning and satisfaction, we conducted two multiple analyses of variance (MANOVA) for each full scale (FSFI, SSS-W) and component subscales, with group (CSA, NSA) as the independent variable, controlling for age, depression, and lifetime history of trauma exposure. There were several significant

differences (see Figure 1). The CSA group reported significantly lower sexual desire,  $F(1, 131) = 4.82, p < .05$ , arousal,  $F(1, 131) = 10.67, p < .001$ , lubrication,  $F(1, 131) = 17.26, p < .001$ , and orgasm functioning,  $F(1, 131) = 7.08, p < .001$ , as well as sexual functioning in general,  $F(1, 131) = 14.49, p < .001$ . The CSA group also reported lower sexual satisfaction,  $F(1, 141) = 9.24, p < .01$ . Specifically, the CSA group reported significantly lower contentment,  $F(1, 141) = 9.60, p < .01$  greater interpersonal sexual  $F(1, 141) = 20.18, p < .001$  and greater personal sexual distress,  $F(1, 141) = 9.32, p < .01$ .

### Group Differences in Physiological Variables

We assessed differences between women with and without CSA histories in the physiological variables (cortisol and HRV) with separate repeated-measures ANOVAs, with stimuli (neutral, sexual) as the repeated measures variable, group (CSA, NSA) as the between-subjects variable, and the interaction between group and time. There was a significant effect of stimuli (neutral vs. sexual) on our marker of HRV, the RRSd,  $F(1, 200) = 37.10, p < .001$ . The effect of group on HRV was not significant,  $F(1, 200) = .10, p = .75$ , indicating that the groups were similar in levels of HRV. There was also a significant decrease in cortisol between neutral and sexual stimuli,  $F(1, 213) = 10.42, p < .001$ ; that is, cortisol secretion decreased between the neutral and sexual stimuli. The effect of group on cortisol levels was not significant,  $F(1, 213) = .38, p = .54$ , indicating that the CSA and NSA samples did not differ in cortisol levels.

### Effects of Changes in Physiological Variables on Sexual Functioning and Satisfaction

For the two physiological variables (cortisol and HRV), we completed a separate repeated-measures ANOVA, with stimuli as the repeated-measures variable (neutral, sexual), group (CSA, NSA) as the between-subjects variable, control variables (age, depression) as covariates, and independent variables (sexual functioning, sexual satisfaction) as covariates. We also constructed

interactions (and all necessary constituent terms) of group (CSA, NSA), symptoms of traumatic stress, and independent variables (sexual functioning, sexual satisfaction). We evaluated only the highest order significant interaction for each independent variable: In other words, if the interaction of Stimuli  $\times$  CSA  $\times$  Traumatic stress  $\times$  Sexual functioning (4-way interaction) was not significant, we would next consider the interaction of Stimuli  $\times$  CSA  $\times$  Sexual functioning (3-way interaction).

In the model predicting HRV, there was a significant interaction of stimuli, CSA status, trauma exposure, and sexual functioning,  $F(1, 97) = 7.03, p < .01$ . In NSA women with low lifetime exposure to trauma, higher increases in HRV (and thus decreases in SNS activity) were associated with higher sexual functioning. For NSA women with high lifetime exposure to trauma, however, higher increases in HRV were associated with lower sexual functioning. In fact, for NSA women with high trauma exposure, higher sexual functioning was associated with decreased HRV to sexual stimuli (see Figure 2). For women with a history of CSA, there was a main effect of lifetime exposure to trauma: Women with high lifetime trauma exposure had higher HRV overall, and a greater increase between neutral and sexual stimuli. For those women with CSA and low lifetime exposure to trauma, higher increases in HRV were associated with lower sexual functioning (see Figure 2).

Also in the model predicting HRV, there was a significant interaction of stimuli, CSA status, trauma exposure, and sexual satisfaction,  $F(1, 97) = 5.82, p < .05$ . Women without a history of CSA and low lifetime exposure to trauma had the highest HRV overall. Within the NSA group, there was an effect of trauma exposure, such that women with high lifetime trauma exposure had larger increases in HRV to sexual stimuli than did women with low trauma exposure. However, for the NSA group, differences in HRV response did not appear to differentiate between higher or lower sexual satisfaction (see Figure 3). For women with a history of CSA and low lifetime exposure to trauma, larger increases in HRV were associated with higher sexual satisfaction. For those CSA survivors with high lifetime trauma exposure, however,

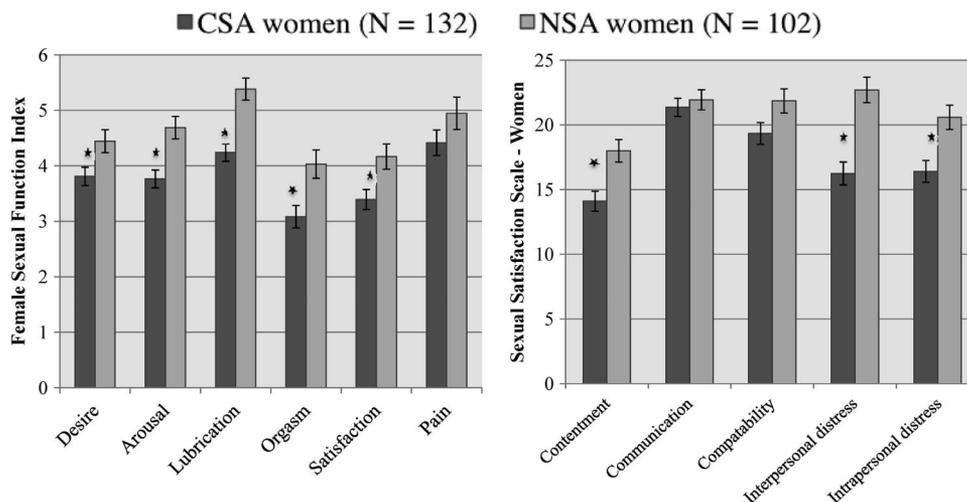


Figure 1. Sexual functioning and satisfaction in women with and without histories of CSA, controlling for age, depression, and history of trauma exposure.

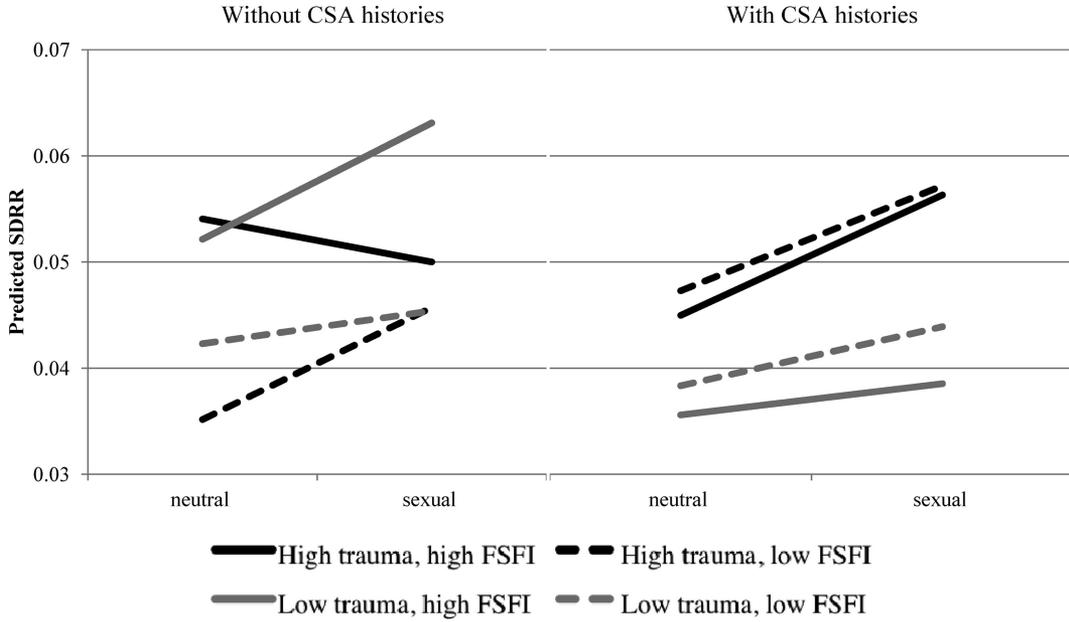


Figure 2. Changes in HRV predicted by lifetime exposure to trauma and sexual functioning, controlling for age and depression symptoms.

larger increases in HRV were associated with lower sexual satisfaction (see Figure 4).

In the model predicting change in cortisol, there was a significant interaction of stimuli, CSA status, and sexual functioning,  $F(1, 97) = 5.03, p < .05$ , such that women with a history of CSA had lower cortisol during both neutral and sexual stimuli than did women without CSA histories. For women without a history of CSA, an increase in cortisol between neutral and sexual stimuli was associated with lower sexual functioning, but a decrease in

cortisol was associated with higher sexual functioning. For women with a history of CSA, however, both high and low sexual functioning were associated with a decrease in cortisol. In other words, cortisol response to sexual stimuli did not differentially predict sexual functioning in women with CSA histories (see Figure 4). There was also a significant interaction between stimuli and sexual satisfaction,  $F(1, 97) = 7.87, p < .001$ , such that across groups, decreases in cortisol to sexual stimuli were associated with higher sexual satisfaction.

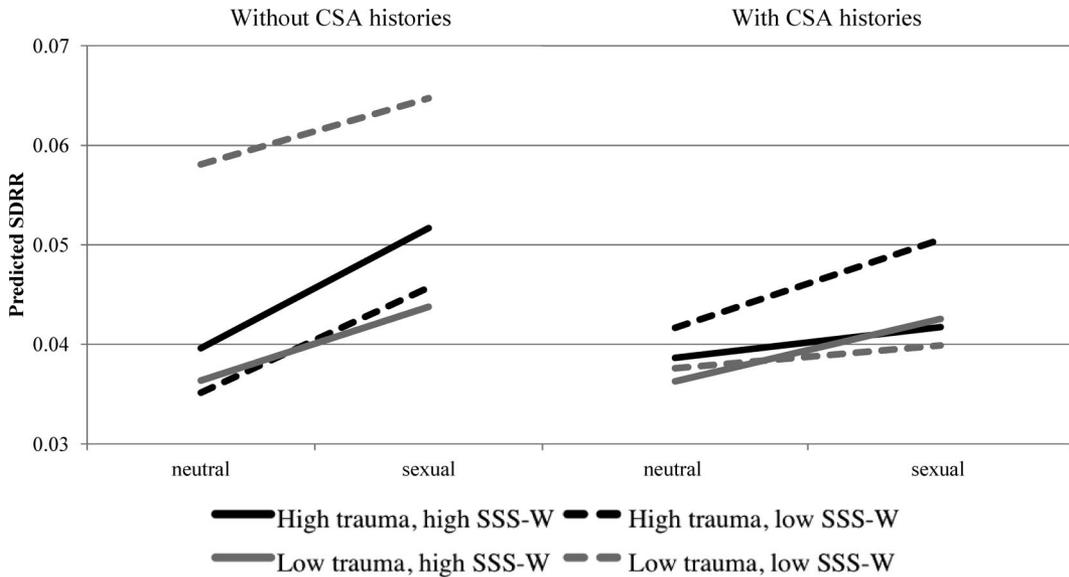


Figure 3. Changes in HRV predicted by lifetime exposure to trauma and sexual satisfaction, controlling for age and depression symptoms.

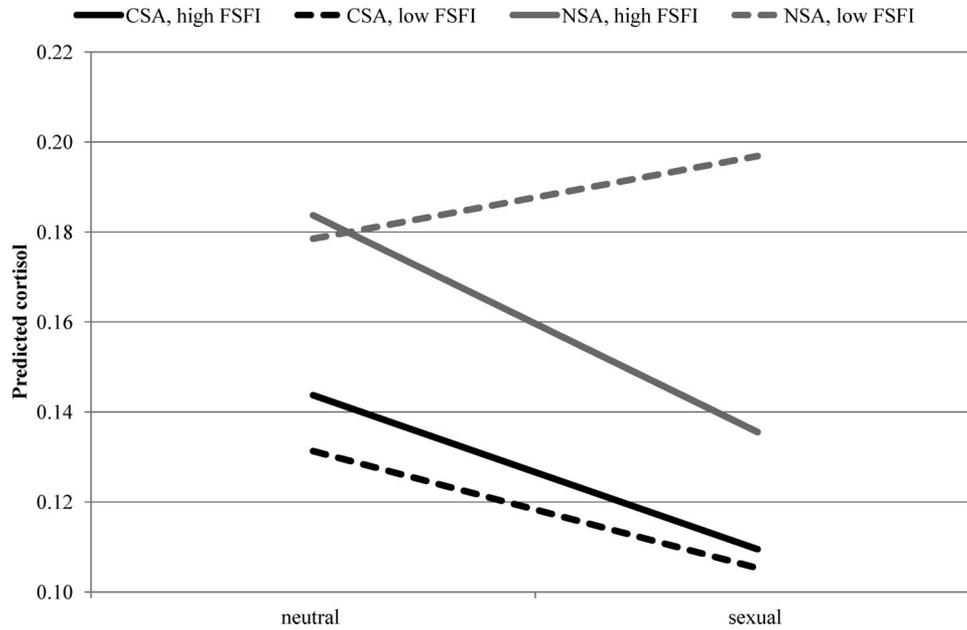


Figure 4. Changes in cortisol predicted by CSA status and sexual functioning, controlling for age, depression, and history of trauma exposure.

## Discussion

As predicted, cortisol and HRV differentially predicted sexual functioning and satisfaction in women with and without CSA histories. For NSA women with low lifetime trauma exposure, larger decreases in SNS activity from neutral to sexual stimuli were associated with better sexual functioning. In contrast, for NSA women with high lifetime trauma exposure, decreases in SNS activity to a sexual stimulus were associated with poorer sexual functioning, though increased SNS activity was associated with higher sexual functioning. For women who reported a history of CSA and low lifetime trauma, larger decreases in SNS activity to a sexual stimulus were associated with better sexual functioning and satisfaction. Cortisol response to sexual stimuli predicted sexual functioning in NSA women, such that increases were associated with lower sexual functioning and decreases with higher functioning. However, as hypothesized, cortisol responses in women with CSA histories did not predict differences in sexual functioning.

The relationship between SNS activation and sexual functioning and satisfaction was moderated by participants' CSA and trauma histories. In women with CSA histories, higher lifetime trauma exposure was associated with less SNS activity overall than was low lifetime exposure to trauma. Across women with CSA histories, greater decreases in SNS activity to sexual stimuli were associated with better sexual functioning; however this effect was exaggerated in those women with low lifetime exposure to trauma. Taken together, these findings suggest that for CSA survivors, singular sexual trauma may increase SNS stress responses to sexual stimuli, but that exposure to additional trauma may suppress these responses. There is evidence that for women whose SNS is chronically elevated, such as those CSA survivors with low trauma exposure (Lemieux & Coe, 1995), further elevation—including

that which is expected during sexually exciting stimuli—may actually inhibit sexual responding and in this way, sexual functioning and satisfaction (Rellini & Meston, 2006). In contrast, a decrease in SNS activity may facilitate physiological sexual response by bringing it to a moderate level. Our findings suggest that the curvilinear relationship between SNS activity and women's genital arousal (Lorenz et al., 2012) may extend to sexual functioning as well.

Similar to the findings on sexual functioning, for CSA survivors with low trauma exposure, larger decreases in SNS activity to a sexual stimulus were associated with higher sexual satisfaction. Unexpectedly, for CSA survivors with a history of multiple traumatic events, this pattern was reversed, such that larger decreases in SNS activity to a sexual stimulus were associated with lower sexual satisfaction. It is possible that for CSA survivors with high trauma exposure, decreases in SNS activity may elicit the moderate SNS levels conducive to sexual arousal, which in turn may cause distress by bringing up problematic memories or emotions. At least one study has found that increases in sexual function in women with CSA histories result in lower, not higher, sexual satisfaction (Stephenson, Pallatto, & Meston, in press).

Our cortisol findings in NSA women replicated those of Hamilton, Rellini and Meston (2008): Women increased in cortisol in response to sexual stimuli had lower sexual functioning, and those with decreased cortisol had higher sexual functioning. That cortisol did not differentiate high or low sexual functioning for women with CSA histories is consistent with the findings of Rellini et al. (2009), who found no significant relationship between cortisol and laboratory measures of sexual arousal in CSA survivors. This suggests that the hormonal disruptions due to CSA disconnect the body's long-term stress response from sexual response. That, in turn, suggests that reducing cortisol responses would have at best

an indirect impact on improving sexual dysfunction in this population.

Given that HRV predicted sexual satisfaction and functioning in women with a history of CSA, incorporating HRV measures into clinical work with this population may be of benefit. It has been shown that heart-rate information used during biofeedback improves treatment of PTSD (Bradley, Greene, Russ, Dutra, & Westen, 2005). Our findings suggest that for women with CSA histories, particularly those with low lifetime trauma, biofeedback targeting changes in HRV may lead to better sexual functioning and satisfaction. Although such therapies would also help CSA survivors with high lifetime exposure to trauma, we would expect less dramatic results. Some limitations should be considered in interpreting our findings. As with all laboratory studies, it is not known if these results would extend to sexual activity with a partner. Second, it is impossible to know if cortisol or HRV changed for reasons other than the sexual stimuli; however, it is unlikely that these factors would differ significantly between the groups. The present study was cross-sectional, thus we cannot infer causality in any reported relationship. Finally, it is possible that women's cortisol response to the sexual stimuli may have continued to decrease after the stimulus presentation ended. If so we would expect our findings to be exaggerated.

Our findings suggest that physiological effects of CSA play out in adult sexual functioning and satisfaction. Specifically, we found that SNS activity differentially predicted sexual functioning and satisfaction in women with and without CSA histories, and that decreases in cortisol predicted sexual functioning for NSA women but not CSA survivors. These findings add to the paucity of literature on physiological factors that play a role in the disruption of the sexual lives of CSA survivors.

## References

- Avis, N. E., Brockwell, S., Randolph, J. F., Shen, S., Cain, V. S., Ory, M., & Greendale, G. A. (2009). Longitudinal changes in sexual functioning as women transition through menopause: Results from the Study of Women's Health Across the Nation (SWAN). *Menopause, 16*, 442–452. doi:10.1097/gme.0b013e3181948dd0
- Bartoi, A. G., & Kinder, B. N. (1998). Effects of child and adult sexual abuse on adult sexuality. *Journal of Sex & Marital Therapy, 24*, 75–90. doi:10.1080/00926239808404921
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the BDI-II*. San Antonio, TX: Psychological Corporation.
- Beck, A. T., Steer, R. A., & Carbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*, 77–100. doi:10.1016/0272-7358(88)90050-5
- Beitchman, J. H., Zucker, K. J., Hood, J. E., DaCosta, G. A., Akman, K., & Cassavia, E. (1992). A review of the long-term effects of child sexual abuse. *Child Abuse & Neglect, 16*, 101–118. doi:10.1016/0145-2134(92)90011-F
- Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *The American Journal of Psychiatry, 162*, 214–227. doi:10.1176/appi.ajp.162.2.214
- Bremner, D., Vermetten, E., & Kelley, M. E. (2007). Cortisol, dehydroepiandrosterone, and estradiol measured over 24 hours in women with childhood sexual abuse-related posttraumatic stress disorder. *Journal of Nervous and Mental Disease, 195*, 919–927. doi:10.1097/NMD.0b013e3181594ca0
- Bremner, J. D., Krystal, J. H., Southwick, S. M., & Charney, D. S. (1996). Noradrenergic mechanisms in stress and anxiety: II. Clinical studies. *Synapse, 23*, 39–51. doi:10.1002/(SICI)1098-2396(199605)23:1<39::AID-SYN5>3.0.CO;2-I
- Carney, R. M., Blumenthal, J. A., Stein, P. K., Watkins, L., Catellier, D., Berkman, L. F., . . . Freedland, K. (2001). Depression, heart rate variability, and acute myocardial infarction. *Circulation, 104*, 2024–2028. doi:10.1161/hc4201.097834
- DiLillo, D. (2001). Interpersonal functioning among women reporting a history of childhood sexual abuse: Empirical findings and methodological issues. *Clinical Psychology Review, 21*, 553–576. doi:10.1016/S0272-7358(99)00072-0
- Elzinga, B. M., Schmahl, C. G., Vermetten, E., van Dyck, R., & Bremner, J. D. (2003). Higher cortisol levels following exposure to traumatic reminders in abuse-related PTSD. *Neuropsychopharmacology, 28*, 1656–1665. doi:10.1038/sj.npp.1300226
- Exton, N. G., Chau Truong, T., Exton, M., Wingenfeld, S., Leygraf, N., Saller, B., . . . Schedlowski, M. (2000). Neuroendocrine response to film-induced sexual arousal in men and women. *Psychoneuroendocrinology, 25*, 187–199. doi:10.1016/S0306-4530(99)00049-9
- Goldey, K. L., & van Anders, S. M. (2011). Sexy thoughts: Effects of sexual cognitions on testosterone, cortisol, and arousal in women. *Hormones and Behavior, 59*, 754–764. doi:10.1016/j.yhbeh.2010.12.005
- Gorcey, M., Santiago, J. M., & McCall-Perez, F. (1986). Psychological consequences for women sexually abused in childhood. *Social Psychiatry and Psychiatric Epidemiology, 21*, 129–133. doi:10.1007/BF00582682
- Green, B. L. (1996). Trauma History Questionnaire. In B. H. Stamm & E. M. Varra (Eds.), *Measurement of stress, trauma and adaptation* (pp. 366–368). Lutherville, MD: Sidran.
- Green, B. L., Krupnick, J. L., Rowland, J. L., Epstein, S., & Stockton, P. (2005). *Trauma History Questionnaire* [Electronic pamphlet]. Retrieved from <http://bit.ly/tre64A>
- Hamilton, L. D., & Meston, C. M. (2011). The role of salivary cortisol and DHEA-S in response to sexual, humorous, and anxiety-inducing stimuli. *Hormones and Behavior, 59*, 765–771. doi:10.1016/j.yhbeh.2010.12.011
- Hamilton, L. D., Rellini, A. H., & Meston, C. M. (2008). Cortisol, sexual arousal, and affect in response to sexual stimuli. *Journal of Sexual Medicine, 5*, 2111–2118.
- Heiman, J. R., Rowland, D. L., Hatch, J. P., & Gladue, B. A. (1991). Psychophysiological and endocrine responses to sexual arousal in women. *Archives of Sexual Behavior, 20*, 171–186. doi:10.1007/BF01541942
- Jehu, D. (1988). *Beyond sexual abuse: Therapy with women who were childhood victims*. New York: John Wiley and Sons.
- Keary, T. A., Hughes, J. W., & Palmieri, P. A. (2009). Women with posttraumatic stress disorder have larger decreases in heart rate variability during stress tasks. *International Journal of Psychophysiology, 73*, 257–264. doi:10.1016/j.ijpsycho.2009.04.003
- Kennedy, S. H., Dickens, S. E., Eisfeld, B. E., & Bagby, R. M. (1999). Sexual dysfunction before antidepressant therapy in major depression. *Journal of Affective Disorders, 56*, 201–208. doi:10.1016/S0165-0327(99)00050-6
- King, J. A., Mandansky, D., King, S., Fletcher, K. E., & Brewer, J. (2001). Early sexual abuse and low cortisol. *Psychiatry and Clinical Neurosciences, 55*, 71–74. doi:10.1046/j.1440-1819.2001.00787.x
- Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: Recent developments and applications. *Psychoneuroendocrinology, 19*, 313–333. doi:10.1016/0306-4530(94)90013-2
- Kobayashi, I. (2010). *Ambulatory sleep and heart rate variability monitoring in trauma-exposed college students* (Doctoral dissertation). Kent State University, Kent, OH. Retrieved from <http://bit.ly/s8pMij>

- Laumann, E. O., Paik, A., & Rosen, R. C. (1999). Sexual dysfunction in the United States: Prevalence and predictors. *JAMA: The Journal of the American Medical Association*, *281*, 537–544. doi:10.1001/jama.281.6.537
- Lemieux, A., & Coe, C. (1995). Abuse-related post-traumatic stress disorder: Evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine*, *57*, 105–115.
- Lorenz, T., Harte, C. B., Hamilton, L. D., & Meston, C. M. (2012). Evidence for a curvilinear relationship between sympathetic nervous system activation and women's physiological sexual arousal. *Psychophysiology*, *49*, 111–117. doi:10.1111/j.1469-8986.2011.01285.x
- Lorenz, T. A., & Meston, C. M. (in press). Associations among childhood sexual abuse, language use and adult sexual functioning and satisfaction. *Child Abuse & Neglect*. doi:10.1016/j.chiabu.2011.09.014
- Meston, C. M. (2003). Validation of the Female Sexual Function Index (FSFI) in women with female orgasmic disorder and in women with hypoactive sexual desire disorder. *Journal of Sex & Marital Therapy*, *29*, 39–46. doi:10.1080/713847100
- Meston, C. M., & Gorzalka, B. B. (1996). The effects of immediate, delayed, and residual sympathetic activation on sexual arousal in women. *Behaviour Research and Therapy*, *34*, 143–148. doi:10.1016/0005-7967(95)00050-X
- Meston, C. M., & Trapnell, P. (2005). Development and validation of a five-factor sexual satisfaction and distress scale for women: The Sexual Satisfaction Scale for Women (SSS-W). *Journal of Sexual Medicine*, *2*, 66–81. doi:10.1111/j.1743-6109.2005.20107.x
- Pennebaker, J. W. (1989). Confession, inhibition, and disease. *Advances in Experimental Social Psychology*, *22*, 211–244. doi.org/10.1016/S0065-2601(08)60309-3
- Pennebaker, J. W. (1997). Writing about emotional experiences as a therapeutic process. *Psychological Science*, *8*, 162–166. doi:10.1111/j.1467-9280.1997.tb00403.x
- Pierrehumbert, B., Torrisi, R., Glatz, N., Dimitrova, N., Heinrichs, M., & Halfon, O. (2009). The influence of attachment on perceived stress and cortisol response to acute stress in women sexually abused in childhood or adolescence. *Psychoneuroendocrinology*, *34*, 924–938. doi:10.1016/j.psyneuen.2009.01.006
- Polusny, M., & Follette, V. (1995). Long-term correlates of child sexual abuse: Theory and review of the empirical literature. *Applied & Preventive Psychology*, *4*, 143–166. doi:10.1016/S0962-1849(05)80055-1
- Putnam, F. W. (2003). Ten year research update review: Childhood sexual abuse. *Journal of the American Academy of Child & Adolescent Psychiatry*, *42*, 269–278. doi:10.1097/00004583-200303000-00006
- Rellini, A. H., Hamilton, L. D., Delville, Y., & Meston, C. M. (2009). The cortisol response during physiological sexual arousal in adult women with a history of childhood sexual abuse. *Journal of Traumatic Stress*, *22*, 557–565.
- Rellini, A. H., & Meston, C. M. (2006). Psychophysiological sexual arousal in women with a history of child sexual abuse. *Journal of Sex & Marital Therapy*, *32*, 5–22. doi:10.1080/00926230500229145
- Resnick, H. S., Yehuda, R., Pitman, R. K., & Foy, D. W. (1995). Effect of previous trauma on acute plasma cortisol level following rape. *The American Journal of Psychiatry*, *152*, 1675–1677.
- Rosen, R., Brown, C., Heiman, J. R., Leiblum, S., Meston, C. M., Shabsigh, R., . . . D'Agostino, R. (2000). The Female Sexual Function Index (FSFI): A multidimensional self-report instrument for the assessment of female sexual function. *Journal of Sex & Marital Therapy*, *26*, 191–208. doi:10.1080/009262300278597
- Scheeringa, M. S., Zeanah, C. H., Myers, L., & Putnam, F. (2004). Heart period and variability findings in preschool children with posttraumatic stress symptoms. *Biological Psychiatry*, *55*, 685–691. doi:10.1016/j.biopsych.2004.01.006
- Shalev, A. Y., & Rogel-Fuchs, Y. (1993). Psychophysiology of the post-traumatic stress disorder: From sulfur fumes to behavioral genetics. *Psychosomatic Medicine*, *55*, 412–423.
- Southwick, S. M., Yehuda, R., & Morgan, C. A. (1995). Clinical studies of neurotransmitter alterations in post-traumatic stress disorder. In M. J. Friedman & D. S. Charney (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to post-traumatic stress disorder* (pp. 335–350). Philadelphia, PA: Lippincott Williams & Wilkins.
- Stephenson, K. R., Pallatto, C. A., & Meston, C. M. (in press). Childhood sexual abuse moderates the association between sexual functioning and sexual distress in women. *Child Abuse and Neglect*.
- Tan, G., Dao, T. K., Farmer, L., Sutherland, R. J., & Gevirtz, R. (2011). Heart rate variability (HRV) and post-traumatic stress disorder (PTSD): A pilot study. *Applied Psychophysiology and Biofeedback*, *36*, 27–35. doi:10.1007/s10484-010-9141-y
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of measurement, physiological interpretation and clinical use. *Circulation*, *93*, 1043–1065. doi:10.1161/01.CIR.93.5.1043
- Terathongkum, S., & Pickler, R. H. (2004). Relationships among heart rate variability, hypertension, and relaxation techniques. *Journal of Vascular Nursing*, *22*, 78–82. doi:10.1016/j.jvn.2004.06.003
- Tsuji, H., Larson, M. G., Venditti, F. J., Manders, E. S., Evans, J. C., Feldman, C. L., & Levy, D. (1996). Impact of reduced heart rate variability on risk for cardiac events: The Framingham Heart Study. *Circulation*, *94*, 2850–2855.
- van Anders, S. M., Brotto, L., Farrell, J., & Yule, M. (2009). Associations among physiological and subjective sexual response, sexual desire, and salivary steroid hormones in healthy premenopausal women. *Journal of Sexual Medicine*, *6*, 739–751. doi:10.1111/j.1743-6109.2008.01123.x
- van Berlo, W., & Ensinnck, B. (2000). Problems with sexuality after sexual assault. *Annual Review of Sex Research*, *11*, 235–258.
- Wolchik, S. A., Spencer, S. L., & Lisi, I. S. (1983). Volunteer bias in employing vaginal measures of sexual arousal. *Archives of Sexual Behavior*, *12*, 399–408. doi:10.1007/BF01542883
- Yehuda, R. (2003). Adult neuroendocrine aspects of PTSD. *Psychiatric Annals*, *33*, 30–36.
- Yehuda, R., Halligan, S. L., Golier, J. A., Grossman, R., & Bierer, L. M. (2004). Effects of trauma exposure on the cortisol response to dexamethasone administration in PTSD and major depressive disorder. *Psychoneuroendocrinology*, *29*, 389–404. doi:10.1016/S0306-4530(03)00052-0
- Yehuda, R., & LeDoux, J. (2007). Response variation following trauma: A translational neuroscience approach to understanding PTSD. *Neuron*, *56*, 19–32. doi:10.1016/j.neuron.2007.09.006

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