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# Association of DHEA, DHEAS, and cortisol with childhood trauma exposure and posttraumatic stress disorder

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## Abstract

There has been a great deal of interest in the role of the neuroendocrine hormones of the hypothalamic-pituitary-adrenal (HPA) axis on the expression of stress-related psychopathology such as posttraumatic stress disorder (PTSD). This investigation examined the association of PTSD and childhood maltreatment with three key HPA axis hormones: cortisol, dehydroepiandrosterone (DHEA), and dehydroepiandrosterone sulfate (DHEAS). Regression analyses were undertaken on a sample of 43 participants with and 57 participants without PTSD. Results demonstrated that after controlling for age, gender, and PTSD status, exposure to childhood maltreatment was significantly associated with cortisol secretion ($F$[4,95]=11.68, $\Delta R^2$=0.11, $p$=.0009) and cortisol/DHEA ratio ($F$[4,95]=6.20, $\Delta R^2$=.05, $p$=.01). PTSD status was not associated with any of these neuroendocrine variables. Findings are discussed in the context of the complexity of the relationship of these neuroendocrine variables with trauma exposure and trauma-related psychopathology. It is suggested that DHEA(S) or cortisol/DHEA(S) ratios may not be biomarkers of specific forms of psychopathology per se, but that instead, the severity and developmental timing of trauma may set the HPA axis in ways that are reflected in interactions among these neuroendocrine hormones. In adulthood, these HPA axis hormones may continue to be dynamically affected by personal and environmental resources.

### Keywords

posttraumatic stress disorder; DHEA; cortisol; child abuse

It has long been understood that chronic or extreme stress contributes to the expression of a range of psychopathology, particularly posttraumatic stress disorder (PTSD) and major depressive disorder (MDD) (Brady et al., 2000, McEwen, 2004, Southwick et al., 2005, Yehuda et al., 2006b). There has been a growing interest in how neuroendocrine hormones may serve as a mechanism in the association of severe stress with PTSD and MDD (Butterfield et al., 2005, Charney, 2004, Eser et al., 2006, Haglund et al., 2007, Maninger et al., 2009, Meewisse et al., 2007, Southwick et al., 2005, Strous et al., 2006). The most

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extensively studied neurosteroid in this regard has been the glucocorticoid cortisol, the final product of the neuroendocrine hypothalamic-pituitary-adrenal (HPA) axis. Cortisol plays a critical role in the mammalian stress response by facilitating the release of glucose for energy and by diverting activity away from non-survival related processes such as immune function. Excessive exposure to cortisol has been found to have neurotoxic effects, however, and disruptions to HPA functioning with chronic activation have been demonstrated in both MDD and PTSD (Meewisse et al., 2007, Yehuda, 2006, Van Voorhees and Scarpa, 2004).

Two other key HPA axis hormones, dehydroepiandrosterone (DHEA) and its sulfated metabolite, DHEAS (often referred to together as DHEA[S]) have received less attention in this area of research (Kroboth et al., 1999). Yet there is evidence that these neurosteroids (Balieu et al., 1965, Traish et al., 2011) also play important roles in that both may have protective effects on brain functioning, including anti-glucocorticoid properties that mitigate the effects of cortisol on stress-induced neural degeneration and death. Because of the important interaction of cortisol and DHEA(S) in neuromodulation, researchers have suggested that examining cortisol/DHEA(S) ratios may be particularly relevant to understanding these hormones' overall effects on stress reactivity (Kroboth et al., 1999, Maninger et al., 2009).

Given that PTSD and MDD are both associated with exposure to high levels of stress, it is not surprising that comorbidity between these disorders is high: data from the National Comorbidity Study indicate that 48% of men and 49% of women with current PTSD had lifetime MDD, making MDD the most highly comordid psychiatric disorder with PTSD (Brady et al., 2000). However, neuroendocrine variables are commonly studied separately in these two disorders, and more is known about the role of these neurosteroids in MDD than in PTSD.

Reviews of the evidence examining the association between neuroendocrine variables and MDD, suggest that depression is associated with increased blood and saliva levels of cortisol, and escape from cortisol suppression in response to the dexamethasone suppression test (DST) (Parker et al., 2003, Pariante and Lightman, 2008). DHEA(S) levels have been found to be decreased, and the ratio of cortisol to DHEA has been found to be increased (Kroboth et al., 1999, Maninger et al., 2009). Finally, DHEA supplementation has been associated with improvements in MDD symptoms (Schmidt et al., 2005, Wolkowitz et al., 1999).

Findings have been less clear with respect to the relationship between PTSD and cortisol, DHEA, and DHEAS. As opposed to hypo-suppression of cortisol to the DST as found in MDD, increased suppression has been more commonly observed in PTSD (de Kloet et al., 2006). While some studies have found low levels of cortisol in individuals with PTSD (Bicanic et al., 2012), overall data have been inconsistent (Meewisse et al., 2007). In males undergoing military survival training school, increases in DHEAS and DHEAS/cortisol ratio predicted fewer symptoms of dissociation and better performance during and after stress (Morgan et al., 2004). Similarly, DHEA levels have been observed to increase in individuals with PTSD who responded successfully to psychotherapy (Olff et al., 2007). Yet compared to healthy controls, increased levels of DHEA(S) have been found in some (Sondergaard et al., 2002, Spivak et al., 2000, Yehuda et al., 2006a) but not all (Kanter et al., 2001, Bicanic et al., 2012), samples of individuals with PTSD. Though difficult to interpret, this pattern of findings has led some researchers to speculate that among those coping with psychological trauma exposure, increased levels of DHEA and DHEAS may be associated with protective effects. This hypothesis is supported by findings that *within* samples of individuals with PTSD, DHEA and DHEAS levels have been found to correlate negatively with symptom

severity (Charney, 2004, Haglund et al., 2007, Rasmusson et al., 2004, Maninger et al., 2009).

In addition to the complexity introduced by the high level of comorbidity between PTSD and MDD, some of the inconsistent findings in the relationship among cortisol, DHEA(S), and PTSD may be due to the lack of a developmental perspective in considering the association between neuroendocrine functioning and adult stress-related psychopathology. This oversight is surprising, considering the wealth of human data that demonstrates the association between childhood stress and adult PTSD and MDD, as well as the decades of animal research suggesting that early stress "sets" the HPA axis in ways that profoundly influence neuroendocrine responses to stress in maturity (see Van Voorhees and Scarpa, 2004, and McCrory et al., 2010, for reviews). One recent study by Kellner and colleagues (2010), however, did take a developmental perspective by examining DHEA and DHEAS in 33 patients with PTSD, 15 of whom had experienced severe childhood sexual or physical abuse, and 18 of whom had not. Results pointed to the importance of considering developmental variables in adult trauma-related psychopathology. Specifically, in response to the DST, increased plasma DHEA and DHEAS and decreased cortisol/DHEA(S) ratios were found in patients with childhood abuse compared to those without (Kellner et al., 2010). The authors concluded that adults with PTSD and childhood abuse may demonstrate altered response to the DST in the form of relatively weak suppression of DHEA(S), relatively strong suppression of cortisol, or both (Kellner et al., 2010).

Finally, cigarette smoking may also contribute to inconsistencies reported in this literature. Nicotine dependence is highly comorbid with PTSD. Depending upon the population sampled, between 45% and 60% of individuals with PTSD have been found to smoke (Lasser et al., 2000, Beckham, 1999, Beckham et al., 1997). Smoking effects cortisol, DHEA, and DHEAS levels (Marx et al., 2006, Rasmusson et al., 2006a, Rasmusson et al., 2006b, Van Voorhees et al., 2013), yet in several studies smoking has not been controlled (Bremner et al., 2007, Kellner et al., 2010, Spivak et al., 2000). Data for the analyses presented here is drawn from the baseline phase of a larger study investigating mechanisms of relapse in smokers with and without PTSD; as such, all of the participants were current cigarette smokers.

In this investigation we sought to replicate and extend previous findings on the relationship among PTSD and cortisol, DHEA, and DHEAS in a sample of 100 participants including both individuals with PTSD and controls without the disorder. To replicate earlier results, we developed Hypothesis I to predict that PTSD diagnosis would be associated with higher levels of DHEA(S) after controlling for age and gender (Sondergaard et al., 2002, Yehuda et al., 2006a). We also developed the following two hypotheses to extend previous work: Hypothesis II predicted that in the entire sample, childhood abuse would explain a significant amount of the variance in cortisol and DHEA(S) levels and in cortisol/DHEA(S) ratios even after the variance associated with PTSD was accounted for; Hypothesis III predicted that the relationship between childhood abuse and altered neuroendocrine functioning would persist even when both PTSD and MDD were accounted for in the model. In sum, we hypothesized that exposure to psychological trauma during childhood would account for variance in neuroendocrine functioning beyond that which was explained by adult diagnoses of PTSD and MDD.

## Materials and methods

### Participants and Procedures

Data presented here are baseline measures administered as part of a larger smoking cessation study investigating mechanisms of relapse in 55 smokers with PTSD and 68 smokers

without PTSD. Twenty three of these cases were eliminated from the current analyses because the blood draws for DHEA(S) and cortisol assays were not conducted between 10:00 am and 2:00pm. As such, complete data were available for 43 smokers with PTSD and 57 smokers without PTSD. Eligible participants were between 18 and 65 years of age, generally healthy, not currently seeking treatment for nicotine dependence, and currently smoking least 10 cigarettes/day. Participants were excluded for major unstable medical problems, using non-cigarette forms of nicotine, pregnancy, non-English speaking, current substance abuse/dependence, schizophrenia, current manic syndrome, lifetime but not current PTSD, and current bupropion or benzodiazepine use. This study was approved by the Duke University School of Medicine Institutional Review Board and the Durham VA IRB and Research and Development Committees.

## Trauma and Psychological Functioning Measures

**Traumatic Life Events Questionnaire (TLEQ)—**The Traumatic Life Events Questionnaire (TLEQ) (Kubany et al., 2000) is a 23-item self-report measure that systematically asks about exposure to a range of potentially traumatic events including natural disaster, motor vehicle accident, childhood physical and sexual abuse, exposure to family violence, adult physical and sexual assault, severe illness, and sudden and unexpected death of loved ones. The TLEQ has been found to have acceptable reliability, and it has been employed in studies across a number of participant populations (Clancy et al., 2006, Dedert et al., 2009, Kubany et al., 2000, Peirce et al., 2009).

Though the entire TLEQ was administered as part of the larger research protocol, we limited our analyses to the following five questions that specifically addressed abuse in childhood: “When growing up, were you physical punished in a way that resulted in bruises, burns, cuts, or broken bones?”; “When growing up, did you see or hear family violence (such as your father hitting your mother; or any family member beating up or inflicting bruises, burns, or cuts on another family member)?”; “Before your 13$^{th}$ birthday, did anyone who was at least 5 years older than you touch or fondle your body in a sexual way or make you touch or fondle their body in a sexual way?”; “Before your 13$^{th}$ birthday, did anyone close to your age touch sexual parts of your body or make you touch sexual parts of their body against your will or without your consent?”; and, “After your 13$^{th}$ birthday and before your 18$^{th}$ birthday, did anyone touch sexual parts of your body or make you touch sexual parts of their body against your will or without your consent?” Consistent with other studies using the TLEQ in veteran samples, we coded responses indicating ever having experienced one or more of these events as childhood abuse being “present”, and responses of never experienced any of these events as childhood abuse being “absent” (Clancy et al., 2006, Dedert et al., 2009).

**Clinician-Administered PTSD Scale (CAPS) and Structured Clinical Interview for DSM-IV Disorders (SCID)—**The CAPS (Blake et al., 1995) was used to determine PTSD diagnosis. This instrument is a clinical structured interview that is considered the “gold standard” for PTSD assessment. PTSD symptoms were considered present based on the CAPS frequency ≥ 1/intensity ≥ 2 rule (Blake et al., 1995, Weathers et al., 2001). The Structured Clinical Interview for *DSM-IV* (First et al., 2002) was used to determine MDD diagnosis. The CAPS and the SCID were administered by clinical raters trained using standardized SCID training. Inter-rater reliability for diagnoses based on videotapes of patient interviews was high (kappa = .96).

## Endocrine testing

Blood for serum analyses was drawn between 10:00 am and 2:00 pm on the day of the study. Samples were centrifuged at 3000 rpm for 15 minutes and frozen within 60 minutes

of venipuncture, and were stored in at −80 degree C until they were shipped on dry ice to the Clinical Assay Ligand Service Satellite (CLASS) Laboratory in the School of Public Health, Department of Epidemiology, at the University of Michigan for analysis.

Cortisol and DHEAS levels were measured using the ADVIA Centaur Cortisol assay and the ADVIA Centaur DHEAS assay, respectively. These assays are competitive immunoassays using direct chemiluminescent technology. CLASS lab cortisol and DHEAS analyses are automated and are therefore conducted in singleton. CLASS lab in-house intra- and inter-assay CV's for DHEAS are between 3.2% – 6.5% and 3.3% – 5.8%, respectively; in-house intra- and inter-assay CV's for cortisol are both between 3.5% – 4.7%. Plasma DHEA samples were analyzed in duplicate using the DRG DHEA ELISA Kit, a solid phase enzyme-linked immunosorbent assay (ELISA); intra-assay CV for DHEA was 4.5%.

### Statistical analysis

Log transformations were used to normalize the distributions for cortisol, DHEA, and DHEAS. Raw scores were used for cortisol/DHEA and cortisol/DHEAS ratios, as these variables more closely approximated a normal distribution. Multiple regression analyses were used to evaluate all hypotheses, and Bonferroni corrections were applied such that the significance level was set at $p < .05/4$, or $p < .01$. Both DHEA and DHEAS are age and gender dependent: secretion levels of both hormones decline from early adulthood through old age, and the ratio of DHEA to DHEAS is higher in women than in men (Kroboth et al., 1999). Therefore, age and gender were entered as covariates in all models.

To test Hypothesis I, two linear regression analyses were conducted using the entire sample ($n$=100). Age, gender, and PTSD status were the independent variables in both models. For the first model the outcome variable was DHEA level, and for the second the outcome variable was DHEAS level. To test Hypotheses II and III, we conducted linear regression analyses using the entire sample. For Hypothesis II, four models were tested using the independent variables age, gender, PTSD status, and childhood abuse, with each of the following outcome variables: DHEA level; DHEAS level; cortisol/DHEA ratio; and cortisol/DHEAS ratio. For Hypothesis III, we re-ran the same four models, adding MDD status to the independent variables.

## Results

### Clinical and demographic characteristics

Gender distribution, age, minority status, education level, employment status, and veteran status are presented in the Table. No significant differences were found between participants with and without PTSD on any of these variables with the exception of employment status: significantly more participants without PTSD were employed compared to those with PTSD (70% without PTSD vs. 30% with PTSD, $\chi^2$=6.9, $p$=.009). Compared to participants without PTSD, a significantly higher percentage of participants with PTSD met criteria for current MDD (16% without PTSD vs. 84% with PTSD, $\chi^2$=10.6, $p$=.0012). Most participants in the sample had experienced at least one significant trauma accompanied by fear, helplessness, and horror at some time in their lives; however, participants with PTSD had experienced significantly more such traumas compared to those without (9.5 [3.6] traumas in those with PTSD vs. 4.4 [3.9] traumas in those without PTSD; $t$=−6.75, $p$<.0001). Among those with PTSD 77% had experienced childhood maltreatment, whereas 21% of those without PTSD had experienced childhood maltreatment ($\chi^2$ =17.1, $p$<.0001).

**Hypothesis I: PTSD diagnosis will be associated with higher levels of DHEA(S)—**This hypothesis was not supported. Age was significantly associated with

DHEA level ($F$[3,96]=21.44, $\Delta R^2$=0.18, $p$<.0001), but gender ($F$[3,96]=0.28, $\Delta R^2$=.002, $p$=.60) and PTSD status ($F$[3,96]=0.83, $\Delta R^2$=.007, $p$=.36) were not. Similarly, age ($F$[3,96]=18.71, $\Delta R^2$=0.16, $p$<.0001) and gender ($F$[3,96]=14.51, $\Delta R^2$=0.11, $p$=.0002) were significantly associated with DHEAS level, but PTSD status was not ($F$[3,96]=0.99, $\Delta R^2$=.007, $p$=.32).

**Hypothesis II: After controlling for age, gender and PTSD status, childhood abuse will explain a significant amount of the variance in cortisol, DHEA, and DHEAS, cortisol/DHEA and cortisol/DHEAS ratios—**After accounting for age, gender, and PTSD status, childhood trauma was significantly inversely associated with cortisol secretion ($F$[4,95]=11.68, $\Delta R^2$=0.11, $p$=.0009) and cortisol/DHEA ratio ($F$[4,95]=6.20, $\Delta R^2$=.05, $p$=.01). Childhood trauma was not significantly associated with DHEA ($F$[4,95]=3.05, $\Delta R^2$=0.02, $p$=.08), DHEAS ($F$[4,95]=.40, $\Delta R^2$=.003, $p$=.53) or cortisol/DHEAS ratio ($F$[4,95]=1.18, $\Delta R^2$=.01, $p$=.28).

**Hypothesis III: The relationship among childhood abuse and altered neuroendocrine functioning will persist even when both PTSD and MDD are accounted for in the models—**After accounting for age, gender, PTSD status, and MDD status, childhood trauma was significantly inversely associated with cortisol secretion ($F$[5,94]=11.56, $\Delta R^2$=.11, $p$=.001) and dropped just below the Bonferroni-corrected level of significance in the inverse association with cortisol/DHEA ratio ($F$[5,94]=6.14, $\Delta R^2$=.05, $p$=.02). Childhood trauma was not significantly associated with DHEA ($F$[5,94]=.40, $\Delta R^2$=.003, $p$=.53), DHEAS ($F$[5,94]=3.08, $\Delta R^2$=.02, $p$=.08), or cortisol/DHEAS ratio ($F$[5,94]=1.17, $\Delta R^2$=.01, $p$=.28).

## Discussion

In this study, controlling for age, gender, and PTSD status, childhood trauma was significantly inversely associated with cortisol secretion and cortisol/DHEA ratio. These significant inverse associations remained significant for cortisol but fell to just below significance for cortisol/DHEA ratio when diagnosis of MDD was accounted for in the models. The effect sizes ($\Delta R^2$ values) fell between .05 and .11, which can be classified as small (.01-.08) to medium (.09-.24) (Cohen, 1988).

Contrary to our hypothesis, we detected no association of PTSD with either DHEA or DHEAS levels in our sample. When considered with the positive findings, this failure to replicate some (but not all, i.e. Bicanic et al., 2012, Kellner et al., 2010) previous research underscores the complexity of the relationship of these neuroendocrine variables with trauma exposure and trauma-related psychopathology. One way to interpret these data is to suggest that it may not be the presence of PTSD that drives elevations in DHEA(S) levels, but rather that DHEA(S) may be associated with efforts to cope in the context of severe and overwhelming stress (Olff et al., 2007, Rasmusson et al., 2004, Wilkins et al., 2005, Yehuda et al., 2006a). Exposure to trauma early in development may alter the HPA axis in ways that affect not only cortisol secretion (Van Voorhees and Scarpa, 2004) but also cortisol/DHEA ratios. That is, while early trauma exposure may alter patterns of cortisol secretion in potentially damaging ways, concomitant alterations to DHEA secretion in the context of ongoing stress and coping may serve as an adaptive neurobiological effort to mitigate some of the long-term negative effects of chronic stress exposure.

Several studies support the interpretation that DHEA(S) levels may be affected by early developmental trauma, and that increases in DHEA(S) levels may be associated with positive coping to ongoing stress or trauma among those with a significant history of trauma exposure. For example, Yehuda et al (2006) found that lower cortisol/DHEA ratio was

associated with greater childhood trauma in combat-exposed veterans with and without PTSD. Among adults with PTSD, Kellner et al (2010) found increases in DHEA and DHEAS, and decreases in cortisol/DHEA and cortisol/DHEAS ratios, in response to dexamethasone administration in participants with (vs. without) childhood abuse. DHEA levels were found to increase after psychotherapy for PTSD in those who responded the treatment (Olff et al, 2007), and among recently resettled refugees, progress toward family reunion was associated with increased levels of DHEAS, whereas decreased levels of DHEAS were associated with "conflict at school" and "feeling misunderstood by an important person" (Sondergaard & Theorell, 2003), p. 55).

In conclusion, findings may suggest that DHEA(S) or cortisol/DHEA(S) ratios are not biomarkers of specific forms psychopathology per se. Instead, the severity and developmental timing of trauma "sets" the HPA axis in ways that are reflected in the levels and interaction of these neuroendocrine hormones (Heim and Nemeroff, 2001, McCrory et al., 2010, Van Voorhees and Scarpa, 2004, van Zuiden et al., 2012, Haglund et al., 2007, Charney, 2004, Rasmusson et al., 2006b). In adulthood, stress reactivity and the HPA axis continue to be dynamically affected by personal and environmental resources (Hobfoll, 1989) including coping skills (Yehuda et al., 2006a), availability of social support (Sondergaard and Theorell, 2003), psychotherapy (Olff et al., 2007), and possibly even direct supplementation with DHEA (Kroboth et al., 1999, Maninger et al., 2009, Sageman and Brown, 2006). These hypotheses, however, would need to be assessed in a longitudinal study to ascertain the timing, strength and direction of the associations between trauma exposure, coping and neurosteroids.

The current study has a number of limitations. First, the assessment of hormone variables was undertaken at a single point in time (i.e., cross-sectional), and the blood samples for serum analyses were collected between 10:00 am and 2:00 pm. While there is significant variation in the literature in the timing of blood draws for cortisol, DHEA, and DHEAS assays, collection at multiple time points is ideal for establishing reliability of hormone levels, and early morning collection is preferred because this is the time when hormone levels are at their highest point in the diurnal cycle (Hucklebridge et al., 2005). However, the data from this investigation are drawn from a larger study of mechanisms of relapse in individuals with PTSD, and we were constrained by practical considerations regarding when participants were able to attend study visits. Following the approach of previous researchers in this area (Marx et al., 2006), we attempted to minimize the effects of these constraints by limiting analyses to data collected within this four hour window; however, future research should attempt to collect samples upon awakening over multiple days.

Second, despite the fact that our sample is relatively large compared to most studies in the area (Bremner et al., 2007, Kellner et al., 2010, Marx et al., 2006, Olff et al., 2007, Spivak et al., 2000, Yehuda et al., 2006a), the study was nonetheless underpowered to detect even a small effect size (i.e. 1- $\beta$=.12 for the regression analyses if $\alpha$ is set to .01, 1- $\beta$=.29 if $\alpha$ is set to .05). While this may increase the confidence in the veracity of the detected effects, it also suggests that extreme caution should be taken in interpreting the null results reported here. The limited power also influenced the number of variables we could include in the regression models. Future research should include other potentially relevant covariates such as number of cigarettes smoked per day; substance abuse history; and number of traumatic experiences. Finally, the sample consisted exclusively of smokers, which inherently controlled for the possible effects of smoking on the neuroendocrine measures but limits generalizability beyond smokers.

Despite the limitations, the results generated in this study add to the growing evidence that neurosteroids may play an important role in the development of responses to trauma. Even

when psychobiological effects range from small to medium, examination of neurosteroids may contribute to the overall understanding of trauma exposure outcomes. The sample was carefully characterized in terms of trauma exposure and psychiatric diagnostic status. Age and gender associations with neuroendocrine measures were significant and in the expected direction, and these effects were controlled in all of the models. The finding that the ratio of cortisol/DHEA was higher among those with childhood trauma exposure but was unrelated to PTSD or MDD suggests that DHEA could be investigated as a potential biomarker for resilience; studies indicating that DHEA supplementation improves depressive and PTSD symptoms (Kroboth et al., 1999, Maninger et al., 2009, Sageman and Brown, 2006) are not inconsistent with this hypothesis. Furthermore, given that the HPA axis plays a pivotal role in the occurrence of PTSD and depression following trauma (de Kloet et al., 2006, Ehlert et al., 2001, Eser et al., 2006, Haglund et al., 2007, Heim and Nemeroff, 2001, Kanter et al., 2001, Klaassens et al., 2011, Lovallo et al., 2012, Meewisse et al., 2007, van Zuiden et al., 2012, Yehuda, 2006, Yehuda et al., 1996), continued study of neuroendocrine effects among individuals who are traumatized is warranted.

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## Table

Demographics and Clinical Characteristics

| | PTSD (n=43) | Without PTSD (n=57) | Test Statistic |
|---|---|---|---|
| Gender (female/male) | 23/20 | 24/33 | $\chi^2$=1.27, ns |
| Age (years) | 42.2 (9.9) | 41.5 (10.2) | $t$=.36, ns |
| Minority (yes/no) | 30/13 | 39/18 | $\chi^2$=.02, ns |
| Education | 12.6 (2.9) | 12.1 (1.9) | $t$=.89, ns |
| Employed (yes/no) | 15/28 | 35/22 | $\chi^2$=6.9, $p$=.009 |
| Veteran (yes/no) | 11/32 | 12/45 | $\chi^2$=.28, ns |
| Depression (yes/no) | 11/32 | 2/55 | $\chi^2$=10.6, $p$=.0012 |
| # Trauma types with fear, helplessness, or horror | 9.5 (3.6) | 4.4 (3.9) | $t$=−6.75, $p$<.0001 |
| Childhood abuse (yes/no) | 33/10 | 10/37 | $\chi^2$=17.1, $p$<.0001 |