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This research has been conducted in compliance with all applicable federal regulations governing the protection of human subjects in research.

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Sex differences in cardiovascular and subjective stress reactions: prospective evidence in a realistic military setting

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Abstract

Evidence points to heightened physiological arousal in response to acute stress exposure as both a prospective indicator and a core characteristic of posttraumatic stress disorder (PTSD). Because females may be at higher risk for PTSD development, it is important to evaluate sex differences in acute stress reactions. This study characterized sex differences in cardiovascular and subjective stress reactions among military survival trainees. One hundred and eighty-five military members (78% males) were studied before, during, and 24 h after stressful mock captivity. Cardiovascular (heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP]) and dissociative states were measured at all three time points. Psychological impact of mock captivity was assessed during recovery. General linear modeling with repeated measures evaluated sex differences for each cardiovascular endpoint, and causal steps modeling was used to explore interrelationships among sex, cardiovascular reactions and psychological impact of mock captivity. Although females had lower SBP than males at all three time points, the difference was most pronounced at baseline and during stress. Accordingly, females showed greater residual elevation in SBP during recovery. Females had lower DBP at all three time points. In addition, females reported greater psychological impact of mock captivity than males. Exploratory causal steps modeling suggested that stress-induced HR may partially mediate the effect of sex on psychological impact of mock captivity. In conclusion, this study demonstrated sex-specific cardiovascular stress reactions in military personnel, along with greater psychological impact of stress exposure in females. This research may elucidate sex differences in PTSD development.

Keywords

Cardiovascular, dissociation, military, sex differences, stress, survival

History

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Introduction

Current evidence suggests that heightened physiological arousal in response to acute stress exposure is a prospective marker as well as a core characteristic of posttraumatic stress disorder (PTSD). For example, numerous studies show that elevated heart rate (HR) in trauma victims is a longitudinal predictor of PTSD development (Bryant et al., 2004; Kassam-Adams et al., 2005; Kuhn et al., 2006; Shalev & Freedman, 2005). This trend persists across varied populations including motor-vehicle-accident victims (Kuhn et al., 2006), brain-injured victims (Bryant et al., 2004), injured children (Kassam-Adams et al., 2005) and victims of terrorist attacks (Shalev & Freedman, 2005). This line of research is complemented by other work suggesting that beta-adrenergic antagonists, such as propranolol (which attenuates HR, blood pressure [BP] and myocardial contractility), may decrease the risk of PTSD development proximal to trauma exposure (Searcy et al., 2012). Another important area of work identifies heightened physiological arousal as a core characteristic of PTSD patients (Pole, 2007), particularly with respect to skin conductance, facial electromyography and HR.

While substantial evidence highlights the importance of acute stress reactions in relation to PTSD development, several gaps in the literature remain. First, it has been noted that a disproportionate focus of this research has been placed on males (Pole, 2007). Considering females may be at higher risk of PTSD development than males (Mota et al., 2012), it is important to evaluate sex differences in acute stress reactions. There is a modest but productive literature evaluating sex differences in cardiovascular responses to laboratory stress in both healthy (Allen et al., 1993) and traumatized individuals (Inslicht et al., 2013). In healthy participants, studies have shown that men respond to selected laboratory stressors with

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greater systolic (Allen et al., 1993; Steptoe et al., 1996) and diastolic BP increases, as well as greater increases in total peripheral resistance than females. Conversely, females tend to respond with greater increases in HR (Allen et al., 1993). Consolidating this evidence, males have been characterized as ''vascular reactors,'' while females have been characterized as ''myocardial reactors.'' In terms of subjective distress, there is evidence that females are more affectively responsive to fear-inducing and stressful experiences than males (Kelly et al., 2006, 2008), and more recent work demonstrates greater fear conditioning in female versus male participants with PTSD (Inslicht et al., 2013).

Despite numerous strengths of the aforementioned acute trauma studies, some limitations reduce their generalizability. For example, although previous studies have assessed real-world trauma (such as motor-vehicle accidents), these studies lack the ability to assess pretrauma characteristics (e.g. baseline physiological values under rested conditions). Conversely, the imposed stressors used in laboratory studies are relatively mild, and the context in which they are delivered may not generalize to real-world events. A program of research examining human responses to intense military stress has the potential to reconcile these limitations. Morgan and colleagues pioneered a scientific paradigm evaluating stress reactivity among trainees in a standardized yet highly realistic survival training involving mock captivity (Dimoulas et al., 2007; Morgan et al., 2000, 2004). One of these studies provided an initial exploration of sex differences in acute military stress reactions. Dimoulas et al. (2007) studied female survival trainees and found that their dissociative states at baseline and in response to intense stress exposure were similar (in both magnitude and characteristics) to those found in previously studied samples of male trainees (Morgan et al., 2001, 2004). There is a need to further characterize sex differences in acute stress reactions in this context. The purpose of this study was to evaluate sex differences in cardiovascular and subjective stress reactions in military survival trainees. In light of previous research in laboratory settings, we anticipated greater BP responses in males and greater HR responses in females. We also expected greater psychological impact of mock captivity in females during recovery. To be clear, this is not a study of PTSD per se, but rather an investigation of stress reactions in healthy individuals exposed to intense stress.

Methods

Military survival training

Survival, evasion, resistance and escape (SERE) training is described in other reports (Dimoulas et al., 2007; Morgan et al., 2000, 2001, 2004; Taylor et al., 2012), and some portions of the curriculum are classified. U.S. military members who are deemed ''high risk of capture'' are required to attend this course, which includes a period of mock captivity. After an initial phase of classroom-based didactic training (5 days), students are taken to a field site where they are trained in SERE techniques (7 days). Training tasks include evasion from a simulated enemy, and, upon eventual ''capture,'' students must practice resistance to various forms of simulated exploitation in stressful mock-captivity training challenges. The entire course lasts 12 days, including 1 debrief day after the conclusion of mock captivity. Because SERE training is designed in part to simulate a captivity experience, it offers a unique medium to prospectively evaluate the effects of highly realistic stress on human functioning. Recent studies confirm its validity as a sustained stressor, as evidenced by substantial alterations in both physiological and self-report indices (Morgan et al., 2004; Taylor et al., 2012).

Inclusion, exclusion and compliance criteria

Subjects met inclusion criteria if they were active-duty military members enrolled in SERE training at the Center for Security Forces, SERE Learning Site West (San Diego, CA) as part of their military duties, and were deemed healthy as indicated by a medical records review conducted by the SERE medical officer. Per the record review, examples of criteria for exclusion from SERE training include disorders of endocrine, nervous/psychological and neuromuscular function, as well as history of clinically significant electrocardiographic abnormality. Additional exclusion criteria imposed for this study included smoking; caffeine dependence; any use of anabolic (e.g. DHEA, growth hormone) or ergogenic (e.g. creatine monohydrate) substance, drug, or supplement within the past 3 months; antihypertensive medication use (e.g. beta-blockers); and current diagnosis of type 1 or type 2 diabetes with prescribed medication.

Compliance requirements were imposed during baseline and recovery assessments. Specifically, subjects were asked to refrain from alcohol ingestion within 12 h of assessments, major meals within 1 h of assessments, and caffeine ingestion within 30 min of assessments. Compliance during mock captivity was implicitly controlled by the training context.

Participants

Two hundred military members were originally enrolled in a larger study evaluating stress and health in survival trainees. Those who expressed an interest in participating attended an in-person meeting on the first day of the academic phase of survival school to review the details of the study and provide written informed consent. This protocol was approved by the Naval Health Research Center Institutional Review Board. Fifteen participants were excluded from the current study because they were delayed at a collection point directly after the mock-captivity event by >3 min (autonomically induced cardiovascular stress responses are known to dissipate substantially within 2–3 min of acute stress exposure; Goswami et al., 2010). This yielded a sample size of 185 (mean [standard deviation (SD)] age, 25.2 [4.4] years; 77.8% males).

Protocol

Participants completed baseline physiological assessments (HR, systolic blood pressure [SBP], diastolic blood pressure [DBP], tympanic temperature and arterial peripheral oxygen saturation [SpO_2]) and self-report measures on the first day of the academic phase of SERE training (Time 1 [T1]; prestress). Subsequently, all subjects experienced a rigorous evasion exercise, after which they participated in a highly

realistic mock-captivity scenario. Physiological measures and dissociative states were assessed again directly after a stressful mock-captivity event (Time 2 [T2]; mock-captivity stress). Finally, approximately 24 h after the release from mock captivity (marking the completion of training), all the subjects completed the physiological assessment and measure of dissociative states a third time, as well as a measure of psychological impact of mock captivity (Time 3 [T3]; recovery).

Measures

Physiological measures

For T1 and T3 assessments, participants were seated in a private, sound-minimized room for 5 min in a chair with back supported, arms bared and supported at heart level, and feet uncrossed and flat on the floor (Pickering et al., 2005; Shapiro et al., 1996). To characterize peak responses to the mock-captivity stressor at T2, an identical assessment occurred within 3 min after the stressful event, inclusive of approximately 2 min of quiet rest in a seated position (all other procedures were held constant). After the period of quiet rest, the finger pulse oximeter (MedSource International, Mound, MN) was applied to the left index finger, and a stable HR value (defined as a continuous, credible reading ± 2 beats per minute [bpm] for 10 s) was recorded. SpO_2 and tympanic temperature were then recorded with pulse oximeter (Medline Industries, Inc., Mundelein, IL). Following standard guidelines (Pickering et al., 2005; Shapiro et al., 1996), BP was then assessed via acoustic sphygmomanometer (Welch Allyn, Inc., Skaneateles Falls, NY). All recordings were estimated to the nearest 2 mmHg. Two members of the research team (MKT and GAP) performed the BP measurements. Inter-rater reliability established in a convenience sample of 10 subjects (70% male) was very high (SBP, $r = 0.98$; DBP, $r = 0.96$).

Self-report measures

Background questionnaire. This questionnaire assesses basic background and demographic information.

Perceived stress scale-10. This is a 10-item questionnaire (Cohen & Williamson, 1988) examining the role of nonspecific appraised stress that people have experienced during the last month, with a possible total score of 40. The mean perceived stress score was 21.9 (SD = 5.9), and Cronbach's alpha reliability in the current sample was 0.80.

Internality, powerful others and chance scales. Locus of control (i.e. the extent to which individuals believe that they can control events that affect them) was assessed using a 24-item measure consisting of three 8-item subscales, one assessing internal locus of control and two assessing specific dimensions of external locus of control (powerful others and chance) (Levenson, 1981). Cronbach's alpha reliabilities in this sample were 0.55, 0.77 and 0.70 for internality, powerful others and chance, respectively.

Brief trauma questionnaire (BTQ). The BTQ (Schnurr et al., 2009) assesses the history of exposure to potentially traumatic events. This questionnaire consists of 10 questions regarding exposure to different types of trauma (e.g. natural disasters, child physical abuse, sexual abuse, muggings, assaults), yielding a total number of traumas experienced.

Dispositional resilience scale-15 (DRS-15). This 15-item scale (Bartone, 1999) measures psychological hardiness. Cronbach's alpha coefficient in the present sample was 0.70.

Clinician-administered dissociative states scale (CADSS). The 19 self-report items from the CADSS (Bremner et al., 1998) reliably and validly assess the frequency and intensity of dissociative states (Morgan et al., 2004; Taylor et al., 2012). This scale is designed to assess how perceptually connected or disconnected an individual is relative to his or her environment, with a maximum possible score of 76. The mean CADSS scores are listed in Table 3. Cronbach's alpha reliability scores for the CADSS were 0.83 at T1, 0.90 at T2 and 0.93 at T3.

Impact of event scale – revised (IES-R). The IES-R is a valid and reliable self-report measure designed to assess current subjective distress for any specific life event (Weiss & Marmar, 1997). It has three subscales comprising 22 items: avoidance (IES-R-Avoid; mean of eight items measuring the extent to which the respondent avoids situations that remind him or her of the stressful or traumatic event); intrusion (IES-R-Intrusion; mean of eight items assessing the extent to which one experiences intrusive thoughts) and hyperarousal (IES-R-Arousal; mean of six items measuring anger, irritability, heightened startle response and hyperarousal). The mean score (IES-R-Mean) is the mean of all 22 items. We have also provided the sum of all items, which is another common data reporting method. In the current study, respondents completed the IES-R at T1 and T3. At T1, the directions were modified to ask the participant to indicate how distressing each difficulty has been relative to the most traumatic event endorsed on the BTQ (described earlier). At T3 (24 h after the conclusion of survival training), the directions were modified to ask the participant to indicate how distressing each difficulty has been relative to the stressful mock-captivity event. At T1, Cronbach's alpha reliabilities in the present sample were 0.61, 0.84, 0.80 and 0.89 for IES-R-Arousal, IES-R-Avoid, IES-R-Intrusion and IES-R-Total, respectively. At T3, they were 0.85, 0.86, 0.79 and 0.93, respectively. Mean IES-R scores are provided in Table 1.

Statistical analysis

Data were analyzed using SPSS software version 19 (SPSS, Inc., Chicago, IL). Distribution characteristics for all continuous variables were examined to determine if assumptions of normality were met, following conservative predefined limits (e.g. skewness between -1 and 1 [Leech et al., 2005], kurtosis between -3 and 3). Variables exceeding any of these limits were either transformed prior to performing the relevant statistical test or, in the presence of a large number of zero values (>30%), dichotomized prior to conducting nonparametric tests. All data transformations reduced skewness and kurtosis to acceptable levels.

Table 1. Participant characteristics.

	Total sample		Males		Females	
Characteristic	*N*	*M* (SD) or %	*N*	*M* (SD) or %	*N*	*M* (SD) or %
Age	184	25.2 (4.5)	143	25.2 (4.8)	41	25.3 (3.2)
BMI**	172	24.6 (2.7)	133	24.9 (2.7)	39	23.5 (2.1)
Military service, years	184	4.6 (3.7)	143	4.6 (3.9)	41	4.6 (3.3)
Education***						
High school	91	49.2%	80	55.6%	11	26.8%
Four-year college	94	50.8%	64	44.4%	30	73.2%
Paygrade**						
Officer	81	44.8%	55	39.3%	26	63.4%
Enlisted	100	55.2%	85	60.7%	15	36.6%
Ethnicity						
Caucasian	124	67.8%	95	66.9%	29	70.7%
Hispanic	21	11.5%	18	12.7%	3	7.3%
Asian	10	5.5%	7	4.9%	3	7.3%
Native American	1	0.5%	1	0.7%	0	0.0%
African American	9	4.9%	7	4.9%	2	4.9%
Mixed ethnicity	15	8.2%	13	9.2%	2	4.9%
Other	3	1.6%	1	0.7%	2	4.9%
Prior head injury						
Yes	37	20.1	33	23.1%	4	9.8%
No	147	79.9	110	76.9%	37	90.2%
Combat experience[a]						
Yes	29	15.8	26	18.2%	3	7.5%
No	154	84.2	117	81.8%	37	92.5%
Prior trauma exposure*	183	2.0 (1.8)	143	2.2 (1.8)	40	1.6 (1.6)
Psychological impact of prior trauma						
Arousal	142	0.2 (0.3)	114	0.1 (0.3)	28	0.3 (0.4)
Avoidance	142	0.4 (0.6)	114	0.4 (0.5)	28	0.6 (0.7)
Intrusion	142	0.3 (0.5)	114	0.3 (0.4)	28	0.5 (0.6)
Total (mean)	142	0.3 (0.4)	114	0.3 (0.4)	28	0.5 (0.5)
Total (sum)	142	7.1 (9.1)	114	6.2 (8.1)	28	10.9 (11.6)
Hardiness	182	31.7 (4.9)	142	31.5 (5.0)	40	32.3 (4.6)
Perceived stress	181	11.7 (5.0)	141	11.4 (4.9)	40	12.7 (5.2)
Locus of control						
Internality	183	36.3 (5.1)	143	36.4 (5.0)	40	35.9 (5.6)
Powerful others	183	16.7 (7.8)	143	17.3 (7.9)	40	15.3 (7.7)
Chance	183	16.4 (6.7)	143	16.1 (7.2)	40	15.1 (5.5)

BMI, body mass index; *M*, mean; SD, standard deviation.
[a]Did not meet assumption for nonparametric analysis (less than five cases in one cell). No formal statistical comparison was performed.
*Difference between males and females $p < 0.05$.
**Difference between males and females $p < 0.01$.
***Difference between males and females $p < 0.001$.

Untransformed means are reported for ease of interpretation. Descriptive analyses were conducted to summarize subject characteristics, and independent *t*-tests or χ^2-tests compared males and females on demographic and background characteristics. For each hypothesis test, a theoretically relevant variable was selected as a covariate if it differed between males and females and associated with the endpoint of interest, thus qualifying as a potential confounder (MacKinnon et al., 2000). Separate 2 (sex) × 3 (time) analysis of variance or analysis of covariance (ANCOVA) with repeated measures evaluated sex differences across time for each endpoint. Greenhouse–Geisser corrections were implemented when sphericity assumptions were not met. Post hoc independent *t*-tests or univariate ANCOVA decomposed the observed group effects at each time point, while post hoc paired *t*-tests decomposed the overall time effects. Absolute (value 2 − value 1) and relative Δ scores ([value 2 − value 1/value 1] × 100%) were also computed and then compared across groups via independent *t*-test or univariate ANCOVA. These calculations were used to operationally define ''reactivity'' (i.e. initial response from baseline to mock-captivity stress), ''recovery'' (i.e. change from mock-captivity stress to 24-h recovery) and ''residual elevation'' (i.e. sustained disruption from baseline to 24-h recovery). Post hoc causal steps modeling (Baron & Kenny, 1986) was used to explore mediated effects. All formal hypothesis tests were two-sided, and the probability of committing a type I error was set at 0.05, though it was acknowledged when more stringent conventional criteria were met (0.01 or 0.001). Bonferroni corrections were implemented for each family of group, time series and delta comparisons (absolute and relative) at 0.05/3 = 0.017. Effect sizes were estimated via partial eta-squared (η_p^2; Ferguson, 2009; Richardson, 2011) (0.04 = minimal effect of ''practical significance,'' 0.25 = moderate effect, 0.64 = strong effect [Ferguson, 2009]).

Results

Participant characteristics

Participant characteristics are shown in Table 1. Demographically, this sample is comparable to that of our previously published studies of Navy survival trainees (Taylor, 2013; Taylor et al., 2012), except that females comprised nearly one quarter of the present sample.

Selection of covariates

As shown in Table 1, males and females did not differ with respect to age, years of military service, ethnicity, prior head injury, dispositional resilience (hardiness), baseline perceived stress or locus of control (all $p > 0.05$). Males had a higher body max index (BMI) ($t(170) = 3.1$, $p < 0.01$) and reported more prior trauma exposure ($t(181) = 2.1$, $p < 0.05$) than females. Of those endorsing any prior trauma ($n = 142$, 80.3% male), no sex differences prevailed with respect to psychological impact or any subdimensions (arousal, intrusion, or avoidance; all $p > 0.05$). A greater proportion of females were college educated ($\chi^2(1, N = 185) = 10.5$, $p < 0.001$). Because military occupational specialty ($\varphi = 0.73$, $p < 0.001$) and paygrade ($\varphi = 0.83$, $p < 0.001$) each covaried substantially with education, neither was evaluated as a possible covariate in any of the models.

Of the above-mentioned characteristics differentiating males and females, BMI (main effect $F(1, 160) = 16.3$, $p < 0.001$, $\eta_p^2 = 0.09$) and education (interaction effect $F(1.66, 295.15) = 4.4$, $p < 0.05$, $\eta_p^2 = 0.02$) related to the SBP stress trajectory and were thus selected as covariates in the hypothesis test concerning sex differences in SBP reactions. Similarly, BMI associated with the DBP stress trajectory (interaction effect $F(1.82, 291.48) = 3.5$, $p < 0.05$, $\eta_p^2 = 0.02$) and was therefore covaried in the corresponding hypothesis test concerning sex differences in DBP reactions.

Overall effects of intense stress exposure

Exposure to mock captivity substantially disrupted all three primary cardiovascular endpoints. Overall time effects were observed for HR ($F(1.52, 270.64) = 607.7$, $p < 0.001$, $\eta_p^2 = 0.77$), SBP ($F(1.65, 260.36) = 7.3$, $p < 0.010$, $\eta_p^2 = 0.04$) and DBP ($F(1.81, 287.98) = 56.0$, $p < 0.001$, $\eta_p^2 = 0.26$). As shown in Table 2, HR, SBP and DBP increased an average of 66.9% (41 bpm), 22.6% (25.1 mmHg) and 14.0% (9.7 mmHg), respectively, from T1 to T2, each followed by incomplete recovery at T3. Mean residual elevation (T1–T3Δ) for HR, SBP and DBP were 2.0 bpm (4.3%), 2.1 mmHg (2.3%) and 3.8 mmHg (6.0%), respectively. Overall exposure effects were also evident for SpO_2 ($F(2, 356) = 26.7$, $p < 0.001$, $\eta_p^2 = 0.13$), characterized by a decrease from T1 to T2 ($t(183) = -8.5$, $p < 0.017$; mean T1–T2Δ = −0.8 [−0.8%]) followed by complete recovery at T3. As shown in Table 3, dissociative states were clearly elevated in response to mock-captivity stress (T2), returning toward baseline at 24-h recovery (T3). Formal time-series comparisons were not performed on this endpoint due to non-normality and >30% zero values at T1 and T3. This is an expected finding under nonstressful conditions, which is consistent with prior research (Morgan et al., 2004; Taylor et al., 2012).

Sex differences in acute stress trajectories

Sex differences in cardiovascular and subjective endpoints are detailed in Tables 2 and 3, respectively. Overall group effects emerged for HR ($F(1,178) = 5.6$, $p < 0.05$, $\eta_p^2 = 0.03$), with post hoc paired *t*-tests indicating that females had higher HR at T1 ($t(183) = -2.5$, $p < 0.017$) and noticeably higher mean values at T2 (110.3 vs. 102.6 bpm), which was rendered nonsignificant with Bonferroni correction ($p > 0.017$). No group differences prevailed in terms of absolute or relative HR reactivity, recovery or residual elevation (all $p > 0.017$). Controlling for BMI and education, a group × time interaction was demonstrated for SBP ($F(1.65, 260.36) = 4.2$, $p < 0.05$, $\eta_p^2 = 0.03$). Specifically, although females demonstrated lower SBP values than males at all three time points, the difference was more pronounced at T1 ($F(1, 163) = 41.3$, $p < 0.017$, $\eta_p^2 = 0.20$) and T2 ($F(1, 161) = 16.1$, $p < 0.017$, $\eta_p^2 = 0.09$) than T3 ($F(1, 159) = 7.8$, $p < 0.017$, $\eta_p^2 = 0.05$). Accordingly, females showed greater residual elevation in SBP, quantified by larger absolute (+7.2 mmHg vs. +1.0 mmHg; $F(1, 159) = 10.4$, $p < 0.017$, $\eta_p^2 = 0.06$) and relative T1–T3Δ (+7.0% vs. +1.2%; $F(1, 159) = 12.1$, $p < 0.017$, $\eta_p^2 = 0.07$). No group differences emerged in terms of absolute or relative SBP reactivity or recovery (all $p > 0.017$). Controlling for BMI, overall group effects appeared for DBP ($F(1,159) = 22.2$, $p < 0.001$, $\eta_p^2 = 0.12$), with post hoc comparisons indicating lower values for females at T1 ($F(1, 164) = 28.4$, $p < 0.017$, $\eta_p^2 = 0.15$), T2 ($F(1, 162) = 8.2$, $p < 0.017$, $\eta_p^2 = 0.05$) and T3 ($F(1, 160) = 6.6$, $p < 0.017$, $\eta_p^2 = 0.04$). No group differences were observed for DBP reactivity, recovery or residual elevation (all $p > 0.017$). Overall group effects emerged for tympanic temperature ($F(1, 179) = 13.1$, $p < 0.001$, $\eta_p^2 = 0.07$), with post hoc paired *t*-tests showing that females registered slightly higher temperatures at T1 ($t(183) = -3.7$, $p < 0.017$) and T3 ($t(179) = -3.1$, $p < 0.017$). Although overall group differences were shown for SpO_2 ($F(1, 178) = 4.8$, $p < 0.05$, $\eta_p^2 = 0.03$), post hoc analyses revealed that this was primarily due to higher mean saturation values for males at baseline ($t(183) = 2.4$, $p < 0.017$).

Females did not differ from males in dissociative states at any time point (T1, $\chi^2(1, N = 183) = 0.6$, $p > 0.017$; T2, $t(181) = -1.2$, $p > 0.017$; T3, $\chi^2(1, N = 183) = 2.8$, $p > 0.017$). However, pronounced sex differences were noted for total psychological impact of mock captivity ($F(1, 178) = 5.2$, $p < 0.05$, $\eta_p^2 = 0.03$), as well as for the subdimensions of arousal ($F(1, 178) = 4.9$, $p < 0.05$, $\eta_p^2 = 0.03$) and intrusion ($F(1, 178) = 6.7$, $p < 0.01$, $\eta_p^2 = 0.04$). Dissociative states at T2 predicted subsequent psychological impact of mock captivity ($r(178) = 0.56$, $p < 0.001$). Stratified by sex, this association was slightly higher for females ($r(37) = 0.63$, $p < 0.001$) than for males ($r(139) = 0.54$, $p < 0.001$).

Exploration of mediated effects

Considering the observed sex differences in cardiovascular stress profiles and psychological impact of mock captivity, we then explored whether any of the primary physiological variables mediated the influence of sex upon psychological impact using Baron and Kenny's causal steps approach

Table 2. Cardiovascular responses to acute stress, stratified by sex.

	Total sample		Males		Females	
Endpoint	*N*	*M* (SD) or %	*N*	*M* (SD) or %	*N*	*M* (SD) or %
HR						
T1	185	63.2 (10.1)	144	62.3 (10.0)	41	66.7 (9.7)*
T2	184	104.3 (19.1)**	144	102.6 (18.6)	40	110.3 (20.0)
T3	180	65.1 (10.8)******	140	64.7 (9.6)	40	66.6 (14.3)
Delta T1–T2	184	66.9% (30.0)	144	66.8% (31.0)	40	66.9% (26.5)
Delta T2–T3	180	−36.1% (13.2)	140	−35.3% (13.4)	40	−38.8% (12.1)
Delta T1–T3	180	4.3% (16.5)	140	5.5% (16.6)	40	0.2% (15.5)
SBP						
T1	184	113.4 (10.8)	143	116.1 (10.2)	41	104.1 (7.4)*
T2	183	138.4 (16.2)**	143	141.0 (16.1)	40	129.1 (12.7)*
T3	181	115.5 (9.6)******	141	116.7 (9.5)	40	111.1 (8.7)*
Delta T1–T2	182	22.6% (13.4)	142	22.1% (13.8)	40	24.3% (11.8)
Delta T2–T3	180	−15.7% (9.4)	140	−16.4% (9.6)	40	−13.4% (8.6)
Delta T1–T3	180	2.3% (8.6)	140	1.0% (8.6)	40	6.9% (6.7)*
DBP						
T1	184	73.9 (8.2)	143	75.7 (8.0)	41	67.8 (5.6)*
T2	183	83.6 (10.6)**	143	84.8 (10.4)	40	79.4 (10.5)*
T3	181	77.6 (7.5)******	141	78.5 (7.4)	40	74.5 (7.3)*
Delta T1–T2	182	14.0% (15.4)	142	13.0% (15.6)	40	17.3% (14.3)
Delta T2–T3	180	−6.0% (13.0)	140	−6.2% (13.6)	40	−5.2% (10.9)
Delta T1–T3	180	6.1% (13.2)	140	4.9 (13.6)	40	10.2% (10.4)
Tympanic temperature						
T1	185	97.4 (0.8)	144	97.3 (0.8)	41	97.7 (0.7)*
T2	184	97.9 (1.2)	144	97.8 (1.2)	40	98.2 (0.8)
T3	181	97.4 (0.8)***	141	97.3 (0.8)	40	97.7 (0.8)*
Delta T1–T2	184	0.6 (1.2)	144	0.6% (1.2)	40	0.5% (1.1)
Delta T2–T3	181	−0.6 (1.3)	141	−0.6% (1.3)	40	−0.5% (1.1)
Delta T1–T3	181	−0.01(0.8)	141	0.0% (0.8)	40	−0.1% (0.8)
SpO_2						
T1	185	98.2 (1.4)	144	98.3 (1.1)	41	97.8 (1.9)*
T2	184	97.5 (1.2)**	144	97.5 (1.2)	40	97.4 (1.1)
T3	180	98.3 (0.8)***	140	98.3 (0.8)	40	98.2 (0.9)
Delta T1–T2	184	−0.8 (1.8)	144	−0.9% (1.6)	40	−0.4% (2.3)
Delta T2–T3	180	0.9 (1.5)	140	0.9% (1.5)	40	0.8% (1.7)
Delta T1–T3	180	0.1 (1.7)	140	0.0% (1.5)	40	0.4% (2.2)

DBP, diastolic blood pressure; HR, heart rate; *M*, mean; SBP, systolic blood pressure; SD, standard deviation; SpO_2, peripheral oxygen saturation; T1, Time 1; T2, Time 2; T3, Time 3.
*Difference from males $p < 0.017$.
**Difference from T1 $p < 0.017$.
***Difference from T2 $p < 0.017$.

Table 3. Subjective responses to acute stress, stratified by sex.

Characteristic	Total sample		Males		Females	
	N	*M* (SD) or %	*N*	*M* (SD) or %	*N*	*M* (SD) or %
Dissociation						
T1	183	1.0 (2.6)	143	1.0 (2.6)	40	1.0 (2.7)
T2	183	13.4 (11.8)	144	12.9 (11.4)	39	15.4 (13.2)
T3	180	3.4 (7.5)	141	2.8 (4.1)	39	5.6 (10.5)
Psychological impact						
Arousal	180	0.8 (0.8)	141	0.7 (0.8)	39	1.0 (0.9)*
Intrusion	180	1.0 (0.8)	141	1.0 (0.8)	39	1.3 (0.8)**
Avoidance	180	0.8 (0.8)	141	0.8 (0.7)	39	0.9 (0.7)
Total (mean)	180	0.9 (0.7)	141	0.8 (0.7)	39	1.1 (0.7)*
Total (sum)	180	19.3 (15.7)	141	17.9 (15.7)	39	24.3 (14.7)*

M, mean; SD, standard deviation.
*Difference from males $p < 0.05$.
**Difference from males $p < 0.01$.

(Baron & Kenny, 1986). Of the notable group effects in Table 2, only HR during mock captivity predicted psychological impact ($r(178) = 0.15$, $p < 0.05$), thus justifying further exploratory testing. Following the hypothesis that HR mediates the influence of sex upon psychological impact, sex was identified as the independent variable, psychological impact as the dependent variable and HR as the candidate mediator. Sex predicted psychological impact in the

regression model (path c; $\beta = 0.17$, $p < 0.05$). When HR was added, the direct effect of sex on psychological impact (path c') was attenuated by >10% ($\beta = 0.15$, $p = 0.05$). HR marginally contributed to the mediation model (path b) ($\beta = 0.13$, $p = 0.10$). Adjusted R^2 values for the regression models testing paths a, b, c and c' were 0.02, 0.04, 0.02 and 0.03, respectively. The mediation model was then tested with an alternate statistical approach (Sobel test; Preacher & Hayes, 2004), evaluating the significance of the indirect effect of the mediator by testing the null hypothesis of no difference between the total effect (path c) and the direct effect (path c'). The Sobel test was marginally significant (test statistic = 1.68, $p = 0.09$).

Discussion

This study characterized sex differences in acute stress reactions among military survival trainees. Females had lower SBP and DBP throughout the trajectory, yet registered greater residual elevation in SBP. Pronounced sex differences were also observed with respect to psychological impact of mock captivity, with females scoring higher than males.

To our knowledge, this is the first report of cardiovascular responses to stressful survival training. Compared with baseline, HR was elevated following stress exposure an average of 41 bpm, SBP by 25 mmHg and DBP by 10 mmHg. All three of these endpoints remained elevated 24 h after the conclusion of mock captivity, suggesting sustained disruption. The mean HR at T2 exceeds that typically observed in the well-validated Trier Social Stress Test (Andrews et al., 2012; Kelly et al., 2008; Kirschbaum et al., 1993), approximates 57% of age-predicted maximum HR for the mean age of this cohort (24 years) (Tanaka et al., 2001) and corresponds to the metabolic demand of exercise at 35–45% of maximum aerobic capacity (Londeree et al., 1995). The BP reactions captured in this study were also comparable to those seen in healthy young subjects exposed to the Trier Social Stress Test (Andrews et al., 2012). Finally, the observed reduction in peripheral SpO_2 resonates with a small literature describing peripheral deoxygenation (Beder et al., 2008) along with increased cerebral oxygenation during acute stress (Paisansathan et al., 2007), perhaps suggesting shunted resources to preserve or enhance brain oxygenation. Synthesized, these observations are consistent with a substantial cardiovascular stress response (Sapolsky, 2004) and, consequently, imply a profound manipulation effect.

Females recorded higher HR than males at baseline, a notable but nonsignificant trend for higher HR during mock captivity, lower SBP and DBP at all time points and greater residual elevation in SBP during recovery. This pattern is partially consistent with a literature showing that men respond to selected laboratory stressors with greater SBP (Allen et al., 1993; Steptoe et al., 1996) and DBP increases (i.e. ''vascular reactors''), while females tend to respond with greater increases in HR (i.e. ''myocardial reactors'') (Allen et al., 1993). In the present study, however, group differences were not observed in absolute or relative *reactivity* or *recovery* for these endpoints, implying that sex differences during mock captivity, while sizeable, are largely a function of baseline differences (Reckelhoff, 2001). Regardless of these findings, the existing literature in ecologically valid settings suggests that absolute HR under stress is a consistent predictor of subsequent PTSD development (Bryant et al., 2004; Kassam-Adams et al., 2005; Kuhn et al., 2006; Shalev & Freedman, 2005). Also, there were notable sex differences in residual elevation of SBP. This is noteworthy in that individual differences in recovery at more extended periods after a stressor may have more important health implications than those observed shortly after the stressor (Goswami et al., 2010).

Pronounced sex differences were also observed in terms of psychological impact of mock captivity, with females scoring higher on total impact, arousal and intrusion. Although it is known that females are at higher risk of PTSD development than males (Mota et al., 2012), far less is known regarding sex differences in subjective distress in the aftermath of acute stress exposure. There is some supporting evidence that females report more subjective distress to fear-inducing and stressful experiences than males (Kelly et al., 2006, 2008).

Stress-induced dissociative states prospectively predicted psychological impact of mock captivity measured during recovery. This finding is consistent with our prior study of male survival trainees (Taylor et al., 2007), as well as a broader literature linking peritraumatic dissociative states to posttraumatic symptoms in both sexes (Werner & Griffin, 2012), across age groups (Sugar & Ford, 2012), and in both civilian (Bryant et al., 2011) and military populations (Agorastos et al., 2013). Although stratified analyses suggested a slightly higher strength of relationship in females, its clinical significance is not clear at this time. That said, this finding complements Dimoulas et al.'s (2007) work showing that dissociative states during mock captivity were highly predictive of subsequent *somatic* symptoms in a sample of female survival trainees. Taken together, it is possible that stress-induced dissociation is a more robust predictor of poststress health outcomes in females.

Exploratory causal steps modeling suggested that stress-induced HR may partially mediate sex differences in psychological impact of acute stress. This finding builds upon an elegant line of prospective research linking HR during emergency room visits to subsequent PTSD development (Bryant et al., 2004; Kassam-Adams et al., 2005; Kuhn et al., 2006; Shalev & Freedman, 2005), as well as related work showing greater physiological (primarily HR) responses to trauma reminders in patients with acute PTSD (Ehlers et al., 2010; Pole, 2007). The present findings may help to elucidate sex differences influencing these longitudinal relationships and may, as a result, improve our understanding of sex differences in PTSD. To be clear, there was only limited support for a mediating role of cardiovascular function, implying the distinct possibility of alternate explanatory mechanisms. Accordingly, our planned research is slated to explore the roles of hypothalamic–pituitary–adrenal and sex hormones.

Limitations of this study require comment. Importantly, we did not control for menstrual phase or oral contraceptive use. Reproductive hormones may be implicated in fear conditioning and extinction (Inslicht et al., 2013), as well as BP regulation at rest and during stress (Davis & Matthews, 1990). Also, this study did not account for ''anticipatory stress''

(Juster et al., 2012) during the baseline data collection period, which is a realistic possibility for survival trainees. Also, although HR, SBP and DBP are clinically relevant, they do not embody a comprehensive suite of cardiovascular metrics.

Conclusion

This study demonstrated sex-specific cardiovascular stress reactions in military personnel, along with greater psychological impact of stress exposure in females. This research may elucidate sex differences in PTSD development. Future studies of sex differences in cardiovascular stress may benefit from multidimensional assessment including heart rate variability, cardiac output, stroke volume, peripheral resistance and pre-ejection period.

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14. ABSTRACT

Objective: Evidence points to heightened physiological arousal in response to acute stress exposure as both a prospective indicator and core characteristic of posttraumatic stress disorder (PTSD). Since females are at higher risk of PTSD development, it is important to evaluate sex differences in acute stress reactions. This study characterized sex differences in cardiovascular and subjective stress reactions among military survival trainees.
Method: One hundred eighty-five military members (78% males) were studied before, during, and 24 hours after stressful mock captivity. Cardiovascular (heart rate [HR], systolic blood pressure [SBP], diastolic blood pressure [DBP]) and dissociative states were measured at all three time points. Psychological impact of mock captivity was assessed during recovery. General linear modeling with repeated measures evaluated sex differences for each cardiovascular endpoint, and causal steps modeling was used to explore interrelationships of sex, cardiovascular reactions, and psychological impact of mock captivity.
Results: Although females had lower SBP than males at all three time points, the difference was most pronounced at baseline and during stress. Accordingly, females showed greater residual elevation in SBP during recovery. Females had lower DBP at all three time points. Additionally, females reported greater psychological impact of mock captivity than males. Exploratory causal steps modeling suggested that stress-induced HR may partially mediate the effect of sex on psychological impact of mock captivity.
Conclusions: This study demonstrated sex-specific cardiovascular stress reactions in military personnel, along with greater psychological impact of stress exposure in females. This may elucidate sex differences in PTSD development.

15. SUBJECT TERMS
stress, sex differences, cardiovascular, dissociation, military, survival

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