

# The Influence of Externalizing Comorbidity on Psychophysiological Reactivity Among Veterans With Posttraumatic Stress Disorder

Kathryn L. Humphreys  
University of California, Los Angeles

Kristen M. Foley and Brian A. Feinstein  
VA Boston Healthcare System, Boston, Massachusetts

Brian P. Marx, Danny G. Kaloupek, and Terence M. Keane  
VA Boston Healthcare System, Boston, Massachusetts, and Boston University School of Medicine

Although most individuals with posttraumatic stress disorder (PTSD) demonstrate heightened physiological reactivity to trauma-related cues, many of these individuals do not. The presence of comorbid externalizing disorders is a potential explanation for this inconsistency. This study investigated the psychophysiological reactions to both standardized and idiographic trauma-related cues among male Vietnam Veterans with PTSD only, PTSD and a comorbid substance use disorder (PTSD-SUD), PTSD and comorbid antisocial personality disorder (PTSD-ASPD), PTSD and both comorbid ASPD and SUD (PTSD-ASPD/SUD), and healthy controls. Results showed that the heart rate reactivity of the PTSD-ASPD and PTSD-ASPD/SUD groups failed to exceed that of the No Disorder group during the imagery-based task, and the PTSD-ASPD/SUD group showed less skin conductance reactivity than the other three PTSD groups in response to the standardized trauma cues. These findings implicate ASPD comorbidity in reduced physiological reactivity to trauma reminders in some individuals with PTSD.

*Keywords:* posttraumatic stress disorder, antisocial personality disorder, substance use disorder, comorbidity, physiological reactivity, Veterans

Research has shown that posttraumatic stress disorder (PTSD) is associated with heightened physiological reactivity to both standardized and idiographic trauma-related stimuli (Orr, Metzger, Miller, & Kaloupek, 2004; Pole, 2007). Yet, up to 40% of those with PTSD do not demonstrate physiological reactivity to trauma-related cues when tested in the laboratory (e.g., Blanchard et al., 1996; Keane et al., 1998; Orr & Roth, 2000; Pitman et al., 1990; Prins, Kaloupek, & Keane, 1995). Although numerous competing hypotheses have been offered to explain this variability (see Orr et al., 2004), one possibility involves co-occurring psychiatric disorders. Of particular relevance are co-occurring antisocial personality disorder (ASPD) and substance use disorder (SUD). Both of these disorders are frequently comorbid with PTSD (e.g., Kessler et al., 1996; Kulka et al., 1990). Research also has demonstrated a developmental link between PTSD and antisocial tendencies, with antisocial behavior in childhood linked to increased odds for both exposure to a traumatic event and subsequent risk for the development of PTSD (Koenen, Moffitt, Poulton, Martin, & Caspi, 2007).

Previous factor analytic studies have demonstrated that ASPD and SUD load on a broad externalizing factor (Krueger, 1999) along with disinhibited personality traits (Krueger et al., 2002), and it has been suggested that there may be subtypes of PTSD, one of which is characterized by the presence of comorbid externalizing psychopathology (e.g., low constraint, antisocial behavior, substance use; Miller, Greif, & Smith, 2003; Miller, Kaloupek, Dillon, & Keane, 2004; Miller & Resick, 2007). The potential influence of comorbid ASPD and SUD on psychophysiological reactivity to trauma-related cues among individuals with PTSD is suggested by evidence that both disorders are associated with psychophysiological hyporeactivity. Antisocial traits have been linked with a pattern of reduced heart rate (HR) and skin conductance (SC) response (Ishikawa, Raine, Lencz, Bihle, & LaCasse, 2001; Sylvers, Brubaker, Alden, Brennan, & Lilienfeld, 2008). Similarly, alcohol and other substance use has been linked to reduced HR and SC response (Iacono, Carlson, & Malone, 2000; Taylor, Carlson, Iacono, Lykken, & McGue, 1999; Taylor, 2004). In addition, high impulsivity or disinhibition, a trait hypothesized to underlie both ASPD and SUD (Krueger et al., 2002), has been linked to electrodermal hyporeactivity (Fowles, 2000).

In this investigation, we examined whether the presence of comorbid ASPD and/or SUD along with PTSD is related to reduced physiological reactivity to trauma-related stimuli. We hypothesized that those with PTSD without ASPD or SUD comorbidity would be significantly more reactive to trauma-related stimuli than a reference group without any diagnosed psychiatric conditions. We also hypothesized that PTSD individuals with ASPD and/or SUD would not differ from the No Disorder reference group. Finally, we expected additive effects, such that indi-

---

Kathryn L. Humphreys, Department of Psychology, University of California, Los Angeles; Kristen M. Foley and Brian A. Feinstein, VA National Center for PTSD, VA Boston Healthcare System, Boston, Massachusetts; Brian P. Marx, Danny G. Kaloupek, and Terence M. Keane, VA National Center for PTSD, VA Boston Healthcare System; and Department of Psychiatry, Boston University School of Medicine.

Correspondence concerning this article should be addressed to Brian P. Marx, PhD, 150 South Huntington Avenue, 116B-4, Boston, MA 02130. E-mail: brian.marx@va.gov

viduals with PTSD and both ASPD and SUD would display the least reactivity, because this group likely has the most severe underlying disinhibitory traits.

## Method

### Participants

The current study consisted of secondary analyses applied to data from VA Cooperative Study #334 (Keane et al., 1998). Specifically, these analyses were conducted on data from a subsample of participants from the original study; this subsample was comprised of 926 male Vietnam Veterans who were in military service between August 1964 and May 1975. Information regarding the full sample and recruitment methods can be found elsewhere (Keane et al., 1998). Briefly, all participants were recruited through inpatient and outpatient services at 15 Department of Veterans Affairs medical centers across the United States and provided informed consent before participation. For the purposes of our secondary analyses, participants included in the subsample were assigned to one of five groups based on the results of a semistructured diagnostic interview: (a) those with current PTSD but without ASPD or SUD ( $n = 447$ ; PTSD); (b) those with current PTSD and a current comorbid SUD without ASPD ( $n = 166$ ; PTSD-SUD); (c) those with current PTSD and current comorbid ASPD without SUD ( $n = 44$ ); (d) those with current PTSD and current comorbid ASPD and SUD ( $n = 30$ ; PTSD-ASPD/SUD); and (e) a group with no mental disorder ( $n = 239$ ; No Disorder). PTSD status was based on a qualifying combat-related trauma. Disorders included in the SUD category consisted of alcohol and drug (sedative, hypnotic, anxiolytic, cannabis, stimulant, opioid, cocaine, hallucinogen/PCP, polydrug, other) abuse and/or dependence. We allowed other Axis I psychiatric comorbidities in the four PTSD groups (see Table 1).

### Measures

**Structured Clinical Interview for Diagnostic and Statistical Manual (SCID).** The SCID (Spitzer, Williams, Gibbon, & First, 1989) is a clinician administered diagnostic instrument that assesses Axis I disorders from the *DSM-III-R* (American Psychiatric Association, 1987). The SCID was used to diagnose all psychiatric disorders.

**Combat Exposure Scale (CES).** The CES (Keane et al., 1989) is a seven-item self-report measure that was developed to

assess war-zone stressors (e.g., men in one's unit killed in action, number of times surrounded by the enemy) experienced by military personnel who were deployed during the Vietnam War. The CES score ranges from 0 to 41 and is calculated by using a sum of weighted scores.

**Mississippi Scale for Combat-Related PTSD (M-PTSD).** The M-PTSD (Keane, Caddell, & Taylor, 1988) is a 35-item self-report measure used to assess combat-related PTSD in veterans. The M-PTSD score ranges from 35 to 175 and is calculated by summing responses to all items.

### Procedure

Pertinent details of the procedure are presented here, but a more complete description can be found in Keane et al. (1998). The psychophysiological challenge procedure consisted, in part, of measuring changes in HR and SC during two types of combat-related stimulus presentations. Both a standardized audiovisual presentation and idiographic imagery scripts were administered so that psychophysiological reactivity could be examined relative to stimuli that were consistent across participants and stimuli that maximized personal relevance. The standardized presentation, modeled after the study by Malloy, Fairbank, and Keane (1983), required participants to view 12 (six neutral and six combat-related) 1-min audiovisual presentations that were recorded onto videotape. The neutral stimuli consisted of photographic images of outdoor scenes that were distinctly different from Vietnam and were accompanied by classical music. The combat-related stimuli were photographic images of the Vietnam War (i.e., a helicopter assault and a firefight) that were accompanied by combat-related sounds (i.e., rotating helicopters blades, small arms fire, explosions, combatant voices). The relevant portion of the procedure began with a 5-min resting baseline, and then the six neutral stimuli were presented, followed by another 5-min recovery/baseline period, and then the six combat-related stimuli. Participants rated their level of subjective distress on a computer using a scale that ranged from 0 (no distress) to 100 (the most that could be imagined) after each presentation.

The second type of stimulus presentation required participants to listen to four imagery scripts, two with standardized neutral content and two with idiographic combat-related content (see Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Participants had each met with a mental health professional during a previous session to compose scripts portraying their two most stressful combat experiences. The neutral imagery scripts described a quiet

Table 1  
*Percentage of Current Disorders as a Function of Diagnostic Group*

Disorder	Sample <sup>a</sup>	No disorder <sup>a</sup>	PTSD <sup>a</sup>	PTSD-SUD <sup>a</sup>	PTSD-ASPD <sup>a</sup>	PTSD-ASPD/ SUD <sup>a</sup>	Group differences <sup>b</sup>
Panic disorder	9.8	0	13.3	12.7	6.1	14.3	C < P, S
Obsessive compulsive disorder (OCD)	5.0	0	6.4	7.4	4.1	8.6	C < P, S
Social Phobia	9.8	0	12.9	12.7	8.2	20	C < P, S, AS
Major depressive disorder (MDD)	27.1	0	35.7	39.2	32.7	37.1	C < P, S, A, AS

*Note.* Sample size was 254 for the No Disorder group (C), 502 for the PTSD group (P), 189 for the PTSD-SUD group (S), 49 for the PTSD-ASPD group (A), and 35 for the PTSD-ASPD/SUD group (AS). PTSD = posttraumatic stress disorder; ASPD = antisocial personality disorder; SUD = substance use disorder.

<sup>a</sup> Numbers are percentages (% yes) for all variables. <sup>b</sup> Bonferroni correction was used for post hoc comparisons of the five groups.

scene viewed from a lawn chair and a scene at the beach. Each script was recorded on audiotape for the purpose of subsequent presentation in the laboratory and consisted of four sequential 30-s periods: resting baseline, reading of the script, imagining the script, and recovery.

HR was recorded from 9-mm-diameter Sensor Medics Ag/AgCl electrodes filled with Beckman electrolyte paste and attached by adhesive collars at standard lead I (arm) sites. Electrodes were connected to a Coulbourn High Gain Bioamplifier (S75-01), and output from the amplifier was directed to a Coulbourn Tachometer (S77-26) to yield a beat-by-beat voltage that was proportional to interbeat interval. SC was measured directly by a Coulbourn Isolated Skin Conductance coupler (S71-23) using a constant 0.5-V output through 9-mm-diameter Sensor Medics Ag/AgCl electrodes filled with an isotomic paste (Fowles et al., 1981). Electrodes were attached to the hypothenar surface of the non-dominant hand, separated by 14 mm. Skin preparation and electrode placement followed published recommendations (Fridlund & Cacioppo, 1986). The HR and SC analog signals were digitized by a Coulbourn Lablinc Analog-to-Digital Converter (L25-12), which was connected to an IBM-compatible computer through a Coulbourn Lablinc Computer Interface (L1R-16). Physiological signals were sampled at 2 Hz and converted to appropriate measurement units (i.e., beats per minute for HR and microsiemens for SC).

Consistent with Keane et al. (1998), we subtracted each individual's highest 30-s mean for HR or SC during the neutral presentations from their highest 30-s mean for HR or SC during the combat-related presentations and then used these difference scores to represent physiological reactivity in subsequent analyses. For the scripted imagery, we subtracted the mean value for the two neutral script imagery periods from the mean value for the two combat script imagery periods. We subtracted the highest subjective distress value from the six neutral scenes from the highest subjective distress value from the six combat scenes to reflect distress reactivity during the standard audiovisual presentations.

## Statistical Analyses

All statistical analyses were performed using SPSS version 17.0. Descriptive statistics were computed for the entire sample and for all five groups, separately. We used analysis of variance (ANOVA) to address our central question about group differences. We used pair-wise comparisons using a Bonferroni adjustment to identify significant differences among the groups. No covariates were included in the initial analyses, but all of the analyses were rerun including variables that the groups differed on as covariates. Covariates included presence/absence for all comorbid diagnoses (panic disorder, obsessive-compulsive disorder, social phobia, major depressive disorder) and several demographic variables (age at time of testing, age at first arrival in Vietnam, annual income, and a dichotomous minority/nonminority race variable).

## Results

### Demographics

Results from initial demographic analyses for the entire sample indicated a mean age at the time of study participation of 43.28

years ( $SD = 3.86$ ), a mean age at first arrival in Vietnam of 20.41 years ( $SD = 3.38$ ), and a mean of 13.95 years ( $SD = 2.44$ ) of lifetime education. Sixty-seven percent of the sample identified as white, 19% as African American, 10% as Hispanic, and 4% as "Other." Table 2 presents the demographic characteristics of the sample as a function of diagnostic group. All four PTSD groups, on average, reported younger age at the time of testing and younger age at the time of arrival into Vietnam, fewer years of total education, and lower current annual incomes than the No Disorder group. The PTSD-ASPD/SUD group also had fewer years of total education than the PTSD only group. No differences were detected in racial composition among the five groups or between the PTSD-ASPD/SUD and PTSD-SUD groups on proportions with alcohol or drug use disorder diagnoses.

Table 1 presents the current diagnoses of comorbid mental disorders in the entire sample and then as a function of diagnostic group. The four PTSD groups did not differ significantly for diagnoses of panic disorder, obsessive-compulsive disorder, social phobia, or major depressive disorder.

### Self-Report Measures

Table 2 also presents the M-PTSD and CES total scores by diagnostic group. On the M-PTSD, the full model was significant,  $F(4, 952) = 191.08, p < .001$ . Post hoc comparisons indicated that the PTSD, PTSD-SUD, PTSD-ASPD, and PTSD-ASPD/SUD groups all had significantly higher scores than the No Disorder group and did not significantly differ from each other. The same was true of scores on the CES, and the full model was also significant,  $F(4, 954) = 57.67, p < .001$ .

We then used logistic regression to examine the degree to which the groups differed on their self reports of the two diagnostic symptom criteria that inquire about emotional and physiological distress to trauma reminders (*DSM-III-R* symptoms B4 and D6). Results showed that all four PTSD groups reported more emotional distress than the No Disorder group (standardized beta weights for PTSD, PTSD-SUD, PTSD-ASPD, and PTSD-ASPD/SUD were  $\beta = 3.76$ , OR = 42.94, 95% CI = 24.86, 74.15;  $\beta = 3.75$ , OR = 42.02, 95% CI = 22.94, 76.98;  $\beta = 3.39$ , OR = 29.52, 95% CI = 13.49, 64.60; and  $\beta = 3.68$ , OR = 39.76, 95% CI = 15.92, 99.27, respectively; all  $ps < .001$ ). The four PTSD groups did not significantly differ from one another. The same pattern was found for physiological reactivity on exposure to internal or external trauma cues ( $\beta = 3.33$ , OR = 28.00, 95% CI = 17.02, 46.06;  $\beta = 3.33$ , OR = 28.01, 95% CI = 16.02, 48.98;  $\beta = 3.25$ , OR = 25.81, 95% CI = 11.89, 56.02; and  $\beta = 3.71$ , OR = 40.67, 95% CI = 15.70, 105.37, respectively; all  $ps < .001$ ).

### Heart Rate Reactivity

Table 3 presents the analysis of covariance (ANCOVA) results for the physiological and subjective distress variables. The full model was significant for HR reactivity to the standardized audiovisual presentations,  $F(4, 903) = 11.27, p < .001$ . Post hoc comparisons indicated that the four PTSD groups were all significantly more reactive than the No Disorder group ( $p < .001, p = .001, p = .014, p = .012$ , respectively).

For HR reactivity to the imagery scripts, the full model was also significant,  $F(4, 903) = 7.11, p < .001$ . Follow-up analyses

Table 2  
*Demographic Characteristics and Self-Report Measures as a Function of Diagnostic Group*

Variable	No disorder <sup>a</sup>	PTSD <sup>a</sup>	PTSD-SUD <sup>a</sup>	PTSD-ASPD <sup>a</sup>	PTSD-ASPD/ SUD <sup>a</sup>	Group differences <sup>b</sup>
Age upon arrival in Vietnam (years)	22.28 (4.67)	19.90 (2.48)	19.84 (2.90)	19.29 (2.30)	19.03 (1.40)	C > P, S, A, AS
Total years of education	15.25 (2.70)	13.70 (2.25)	13.43 (2.15)	13.00 (1.57)	12.51 (12.08)	C > P, S, A, AS; P > AS
Current age (years)	45.05 (5.28)	42.80 (3.12)	42.70 (2.86)	42.41 (3.46)	41.77 (2.54)	C > P, S, A, AS
Current annual income (\$, in thousands)	29.27 (22.10)	14.21 (13.68)	12.28 (12.24)	9.17 (2.08)	8.80 (11.68)	C > P, S, A, AS
Minority (% yes)	31.2	32.5	36.2	40.8	40.0	—
Mississippi Scale	86.42 (14.28)	108.13 (12.53)	108.69 (12.49)	106.45 (12.38)	107.41 (12.38)	C < P, S, A, AS
Combat Exposure Scale	18.70 (10.98)	28.93 (8.24)	29.22 (8.80)	28.78 (9.20)	29.43 (8.16)	C < P, S, A, AS
Psychological distress with reminder (% yes)	6.5	74.8	74.6	67.3	73.6	C < P, S, A, AS
Physiological reactivity with reminder (% yes)	8.4	71.7	71.5	68.8	78.8	C < P, S, A, AS

*Note.* Sample size ranged from 219 to 243 for the No Disorder group (C), ranged from 406 to 459 for the PTSD group (P), ranged from 158 to 178 for the PTSD-SUD group (S), ranged from 43 to 49 for the PTSD-ASPD group (A), and ranged from 28 to 32 for the PTSD-ASPD/SUD group (AS). PTSD = posttraumatic stress disorder; ASPD = antisocial personality disorder; SUD = substance use disorder.

<sup>a</sup> Numbers under the columns for continuous variables are means followed by standard deviations in parenthesis and percentages indicated by % for dichotomous variables. <sup>b</sup> Bonferroni correction was used for post hoc comparisons of the five groups.

revealed that although the PTSD and PTSD-SUD groups were both significantly more reactive than the No Disorder group ( $ps < .001$ ), the PTSD-ASPD and the PTSD-ASPD/SUD groups did not significantly differ from the No Disorder group. Moreover, the PTSD-ASPD and PTSD-ASPD/SUD groups did not significantly differ from the PTSD and PTSD-SUD groups.

### Skin Conductance Reactivity

The full model predicting SC reactivity during the standardized audiovisual presentation was significant,  $F(4, 847) = 9.92, p < .001$ . The PTSD, PTSD-SUD, and PTSD-ASPD groups were all found to be significantly more reactive than the No Disorder group ( $p < .001, p < .001, and p = .009$ , respectively). However, the PTSD-ASPD/SUD group did not differ from the No Disorder group ( $p = .55$ ). Notably, significant differences were found within the four PTSD groups. The PTSD, PTSD-SUD, and PTSD-ASPD groups were also found to be significantly more reactive than the PTSD-ASPD/SUD group ( $p = .002, p = .014, p = .020$ , respectively).

The full model for SC reactivity to the imagery scripts was found to be significant  $F(4, 854) = 4.25, p < .05$ . Probing for group differences in SC reactivity to the imagery scripts revealed that the PTSD, PTSD-SUD, and PTSD-ASPD groups were all significantly more reactive than the No Disorder group ( $p < .001, p = .012, and p = .026$ , respectively). However, once again, the PTSD-ASPD/SUD group did not differ from the No Disorder group ( $p = .25$ ).

### Subjective Distress Reactivity

The full model for group differences on subjective distress reactivity was significant,  $F(4, 898) = 26.15, p < .001$ . The four PTSD groups all demonstrated significantly greater increases in subjective distress to the standardized audiovisual presentation than the No Disorder group (all  $ps < .001$ ). The PTSD groups did not differ from each on reported distress reactivity.

For all models of reactivity, the models remain essentially unchanged when the covariates noted above were included, suggesting that the noted above group differences are robust to statistical control of potentially confounding variables.

## Discussion

The current findings provide support for the hypothesis that externalizing psychiatric comorbidity may contribute to the variability in physiological reactivity to trauma-related stimuli among individuals with PTSD. As expected, the PTSD group without any externalizing comorbidity demonstrated significantly more HR and SC reactivity than the No Disorder group on both standardized and idiographic presentations. Partially consistent with our main hypothesis, the PTSD-ASPD and PTSD-ASPD/SUD groups did not differ from the No Disorder group in HR reactivity to the idiographic imagery task, suggesting that these individuals do not exhibit the expected physiological reactivity consistent with others who receive a PTSD diagnosis. Additionally, during the standardized presentations the PTSD-ASPD/SUD group did not differ from the No Disorder group on SC reactivity, and both groups were significantly less reactive than the PTSD, PTSD-SUD, and PTSD-ASPD groups. However, it should be noted that all of the PTSD groups were moderately more reactive than the No Disorder group in HR reactivity on the standardized presentations. Contrary to our hypotheses, the PTSD-SUD group was significantly more reactive than the No Disorder group on all physiological outcomes.

The general findings showing that the PTSD-ASPD/SUD group and, in a more limited way, the PTSD-ASPD group did not differ from the No Disorder group in terms of physiological reactivity were consistent with expectations. These results are partly consistent with findings that individuals with ASPD exhibit reduced HR and SC during exposure to a stressor when compared with non-disordered and substance dependent groups (Raine, Lencz, Bihrl, LaCasse, & Colletti, 2000). ASPD may be responsible for limited HR and SC reactivity to trauma-related stimuli despite emotional



Table 3  
*Physiological and Subjective Responses to Standardized Audiovisual Presentations and Imagery Scripts as a Function of Group*

Variable	No disorder <sup>a</sup>	PTSD <sup>a</sup>	PTSD-SUD <sup>a</sup>	PTSD-ASPD <sup>a</sup>	PTSD-ASPD/SUD <sup>a</sup>	ANOVA <i>F</i>	Group difference
Audiovisual presentation Imagery scripts	-0.07 [-0.71, 0.58] 1.71 [1.23, 2.19]	2.65 [2.17, 3.12] 3.30 [2.94, 3.65]	Heart rate reactivity 1.85 [1.08, 2.61] 3.04 [2.46, 3.61]	2.04 [0.49, 3.58] 2.33 [1.17, 3.48]	2.43 [0.59, 4.27] 2.87 [1.49, 4.24]	11.27** 7.11**	C < P, S, A, AS C < P, S
	0.22 [0.03, 0.41] 0.47 [0.35, 0.59]	0.90 [0.76, 1.03] 0.77 [0.68, 0.86]	Skin conductance 0.75 [0.53, 0.97] 0.71 [0.57, 0.86]	0.87 [0.42, 1.31] 0.82 [0.54, 1.11]	0.05 [-0.46, 0.57] 0.56 [0.22, 0.90]	9.92** 4.25*	C, AS < P, S, A C < P, S, A
Subjective distress	34.84 [31.01, 38.66]	58.08 [55.26, 60.90]	57.37 [52.78, 61.95]	57.32 [48.14, 66.49]	61.11 [50.00, 72.21]	26.15**	C < P, S, A, AS

*Note.* Sample size ranged from 219 to 239 for the No Disorder group (C), from 405 to 447 for the PTSD group (P), from 155 to 166 for the PTSD-SUD group (S), from 40 to 44 for the PTSD-ASPD group (A), and from 28 to 30 for the PTSD-ASPD/SUD group (AS). PTSD = posttraumatic stress disorder; ASPD = antisocial personality disorder; SUD = substance use disorder.  
<sup>a</sup> Numbers under the columns for continuous variables are adjusted means followed by 95% CIs in brackets. <sup>b</sup> Subjective units of distress was reported after the audiovisual presentations.  
\*  $p < .05$ . \*\*  $p < .001$ .

distress as reflected by self-report. The present study highlighted that ASPD, in combination with SUD, resulted in the lowest reactivity among individuals with PTSD. Having both disorders may indicate the greatest level of disinhibition, a potential mediator for physiological reactivity. A potential mechanism for the limited HR and SC reactivity is an underactive autonomic arousal system that increases propensity for sensation-seeking, which then leads to engagement in antisocial acts (Raine, Reynolds, Venables, Mednick, & Farrington, 1998). The electrodermal hyporeactivity found in individuals with ASPD may also be caused by attentional allocation processes that affects motivational systems (Patrick & Lang, 1999).

A recent meta-analysis of psychophysiological responding in PTSD found that both standardized and idiographic stimuli demonstrated effect sizes in the moderate range when differentiating between PTSD groups and controls (Pole, 2007). In the present study, both groups with ASPD comorbidity were significantly more reactive than the No Disorder group in the standardized HR condition, but not in the idiographic condition. Our finding is particularly interesting because Pole's (2007) meta-analysis found that idiographic stimuli were associated with the greatest differentiation for individuals with PTSD. Contrary to the observed changes in HR for standardized scripts, all groups showed an increased HR response to the idiographic imagery scripts, but the relative increase in HR reactivity was smaller for the two ASPD groups. One explanation is that antisocial individuals may be less likely to fully engage in idiographic stimuli, consistent with previous work that found an association between antisocial behavior and imagery response deficits (Patrick, Cuthbert, & Lang, 1994).

The heightened physiological reactivity shown by the PTSD-SUD group for HR response to both standardized and idiographic presentations and SC response to the standardized presentation is at least superficially inconsistent with the notion of a general externalizing disorder category linked with a diminished physiological reactivity to trauma cues. However, a widely embraced explanation for the high rates of co-occurrence between PTSD and SUD is that individuals with PTSD use substances to reduce or control their distress (Brown & Wolfe, 1994; Coffey, Stasiewicz, Hughes, & Brimo, 2006; Conrod & Stewart, 2003). Individuals who develop SUD as a result of attempting to cope with the emotional sequelae to trauma exposure may be etiologically distinct from individuals who have a primary SUD.

Furthermore, past studies that examined reactivity in individuals with SUD may not have taken into full account the potential reduced reactivity caused by comorbid ASPD. The inclusion of the PTSD-ASPD/SUD group allowed for the separation of those in the SUD group with and without ASPD. Although SUD has been linked to disinhibition (Krueger et al., 2002), it is likely that those individuals with both SUD and ASPD are more disinhibited (and, thus, less reactive to contextual stimuli) than those with either disorder on its own.

Further insight into the observed pattern of physiological reactivity may be gained, considering that the ASPD group may, in itself, display considerable heterogeneity. In Lorber's (2004) meta-analysis exploring psychophysiology related to psychopathy/sociopathy, aggression, and conduct problems, these traits are not uniform in their relation to physiological responding. In adults, aggression was related to increased HR and SC reactivity, whereas psychopathy was related to decreased reactivity on those measures.

Unfortunately, we were unable to examine either aggression or psychopathic traits using the present data set, although both constructs are related to ASPD and could help to explain variability within the ASPD group.

In contrast to the varying results for physiological indicators, the four PTSD groups were comparable in their interview-based endorsement of PTSD symptoms that index emotional and physiological distress to trauma reminders. The discrepancy between physiological responses and self-report of subjective distress underscores the importance of performing multimodal assessments (see Keane, Street, & Stafford, 2004) as well as providing further support that mind and body may express distress somewhat desynchronously (Orr & Roth, 2000).

There are three noteworthy limitations to study methods. First, the sample consists solely of male Vietnam Veterans, thereby potentially limiting the generalizability of these findings to other populations. Second, we were unable to create equal-sized, large group sizes because of the characteristics of the available sample. Third, diagnoses were based on *DSM-III-R* criteria rather than *DSM-IV* criteria. Although replication using current diagnostic criteria are warranted, previous studies have demonstrated good agreement between *DSM-III-R* and *DSM-IV* PTSD diagnoses (e.g., Schwarz & Kowalski, 1991) and good to excellent agreement for substance abuse and dependence (e.g., Grant, 1996).

Current findings echo previous demonstrations that objective physiological reactivity to trauma-related stimuli is not isomorphic with PTSD diagnostic status. We have demonstrated that some groups with a PTSD diagnosis do not exhibit the same magnitude of psychophysiological reactivity to trauma-related stimuli. These findings are important because it may impact conceptualization of the disorder, as well as temper the expectation that all individuals with a diagnosis of PTSD will respond similarly. Importantly, these findings extend previous research on the topic by suggesting that externalizing psychiatric comorbidity plays a role in physiological nonresponding.

The current findings are consistent with findings from previous work showing that it may be difficult to discriminate individuals with PTSD from those without the disorder using only psychophysiological challenge tasks (e.g., Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Blanchard, Kolb, & Prins, 1991; Keane et al., 1998). Comorbid ASPD may be one reason for low levels of responding, although it cannot explain all of the variation of reactivity. Understanding the nature of this influence may eventually make it possible to refine physiological reactivity to emotional challenges as a biological marker for PTSD. Our results are also in line with the well-accepted notion that all of the current PTSD assessment methods have inherent limitations. As such, it is standard practice to use multiple methods and measures to better inform PTSD diagnostic decisions (e.g., Weathers, Keane & Foa, 2009). Such multimethod assessment takes advantage of each method's relative strengths, overcoming the psychometric limitations of any single instrument and maximizing correct diagnostic decisions.

The results of this study also have potentially important treatment implications. Specifically, given their lack of physiological reactivity to trauma-related stimuli, individuals diagnosed with both ASPD and PTSD may be less likely to respond favorably to evidence-based treatments for PTSD, such as prolonged exposure (Foa, Hembree, & Rothbaum, 2007), that emphasize the repeated

confrontation of trauma-related memories and other stimuli to reduce pathological fear through the process of habituation (Foa & Kozak, 1986). In such cases, the use of other evidence-based treatments, such as Cognitive Processing Therapy (Monson et al., 2006; Resick & Schnicke, 1992) that purport to alleviate PTSD symptoms by addressing underlying pathological cognitions or other processes may be indicated.

Further research on the potential for some comorbid disorders to alter the symptom presentation among those with PTSD may provide important information for mental health professionals working with individuals coping with traumatic stress. Future studies that consider psychophysiological responding as predictors of PTSD onset, persistence, and treatment response should include both SUD and ASPD, their early life correlates, and the associated trait of disinhibition as potential moderators of effects. The challenge going forward is to determine why trauma-related cues apparently fail to evoke substantial physiological reactions in some individuals with PTSD, particularly those for whom ASPD is diagnosed.

## References

- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised). Washington, DC: American Psychiatric Association.
- Blanchard, E., Hickling, E., Buckley, T., Taylor, A., Vollmer, A., & Loos, W. (1996). Psychophysiology of posttraumatic stress disorder related to motor vehicle accidents: Replication and extension. *Journal of Consulting and Clinical Psychology, 64*, 742–751.
- Blanchard, E. B., Kolb, L. C., Pallmeyer, T. P., & Gerardi, R. J. (1982). A psychophysiological study of Post Traumatic Stress Disorder in Vietnam veterans. *Psychiatric Quarterly, 54*, 220–229.
- Blanchard, E. B., Kolb, L. C., & Prins, A. (1991). Psychophysiological responses in the diagnosis of posttraumatic stress disorder in Vietnam veterans. *Journal of Nervous and Mental Disease, 179*, 97–101.
- Brown, P. J., & Wolfe, J. (1994). Substance abuse and posttraumatic stress disorder comorbidity. *Drug and Alcohol Dependence, 35*, 51–59.
- Coffey, S. F., Stasiewicz, P. R., Hughes, P., & Brimo, M. L. (2006). Trauma-focused imaginal exposure with comorbid PTSD- alcohol dependent individuals: Revealing mechanisms of alcohol craving in a cue reactivity paradigm. *Psychology of Addictive Behaviors, 20*, 425–435.
- Conrod, P. J., & Stewart, S. H. (2003). Experimental studies exploring functional relations between posttraumatic stress disorder and substance use disorder. In P. Ouimette & P. J. Brown (Eds.), *PTSD and substance use disorder comorbidity* (pp. 57–72). Washington, DC: American Psychological Association.
- Foa, E. B., Hembree, E. A., & Rothbaum, B. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences: Therapist guide*. New York, NY: Oxford University Press.
- Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*, 20–35.
- Fowles, D. (2000). Electrodermal hyporeactivity and antisocial behavior: Does anxiety mediate the relationship? *Journal of Affective Disorders, 61*, 177–189.
- Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology, 18*, 232–239.
- Fridlund, A. J., & Cacioppo, J. T. (1986). Guidelines for humans electromyographic research. *Psychophysiology, 23*, 567–589.
- Grant, B. (1996). *DSM-IV, DSM-III-R, and ICD-10 alcohol and drug abuse/harmful use and dependence, United States, 1992: A nosological comparison. Alcoholism: Clinical and Experimental Research, 20*, 1481–1488.

- Iacono, W. G., Carlson, S. R., & Malone, S. M. (2000). Identifying a multivariate endophenotype for substance use disorders using psychophysiological measures. *International Journal of Psychophysiology*, *38*, 81–96.
- Ishikawa, S. S., Raine, A., Lencz, T., Bihle, S., & LaCasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology*, *110*, 423–432.
- Keane, T., Kolb, L., Kaloupek, D., Orr, S., Blanchard, E., Thomas, R., . . . Lavori, P. (1998). Utility of psychophysiology measurement in the diagnosis of posttraumatic stress disorder: Results from a department of Veteran's Affairs cooperative study. *Journal of Consulting and Clinical Psychology*, *66*, 914–923.
- Keane, T. M., Caddell, J. M., & Taylor, K. L. (1988). Mississippi Scale for Combat-Related Posttraumatic Stress Disorder: Three studies in reliability and validity. *Journal of Consulting and Clinical Psychology*, *56*, 85–90.
- Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., Taylor, K. L., & Mora, C. A. (1989). Clinical evaluation of a measure to assess combat exposure. *Psychological Assessment*, *1*, 53–55.
- Keane, T. M., Street, A. E., & Stafford, J. (2004). The assessment of military-related PTSD. In J. P. Wilson, & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (2nd ed., pp. 262–285). New York: Guilford Press.
- Kessler, R. C., Nelson, C. B., McGonagle, K. A., Edlund, M. J., Frank, R. G., & Leaf, P. J. (1996). The epidemiology of co-occurring addictive and mental disorders: Implications for prevention and service utilization. *American Journal of Orthopsychiatry*, *66*, 17–31.
- Koenen, K., Moffitt, T., Poulton, R., Martin, J., & Caspi, A. (2007). Early childhood factors associated with the development of post-traumatic stress disorder: Results from a longitudinal birth cohort. *Psychological Medicine*, *37*, 181–192.
- Krueger, R. (1999). The structure of common mental disorders. *Archives of General Psychiatry*, *56*, 921–926.
- Krueger, R., Hicks, B., Patrick, C., Carlson, S., Iacono, W., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology*, *111*, 411–424.
- Kulka, R., Schlenger, W., Fairbank, J., Hough, R., Jordan, B., Marmar, C., & Weiss, D. (1990). *Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study*. Philadelphia: Brunner/Mazel.
- Lorber, M. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, *130*, 531–552.
- Malloy, P. F., Fairbank, J. A., & Keane, T. M. (1983). Validations of a multimethod assessment of posttraumatic stress disorders in Vietnam veterans. *Journal of Consulting and Clinical Psychology*, *51*, 488–494.
- Miller, M., Greif, J., & Smith, A. (2003). Multidimensional Personality Questionnaire profiles of veterans with traumatic combat exposure: Externalizing and internalizing subtypes. *Psychological Assessment*, *15*, 205–215.
- Miller, M., Kaloupek, D., Dillon, A., & Keane, T. (2004). Externalizing and Internalizing Subtypes of Combat-Related PTSD: A Replication and Extension Using the PSY-5 Scales. *Journal of Abnormal Psychology*, *113*, 636–645.
- Miller, M., & Resick, P. (2007). Internalizing and externalizing subtypes in female sexual assault survivors: Implications for the understanding of complex PTSD. *Behavior Therapy*, *38*, 58–71.
- Monson, C. M., Schnurr, P. P., Resick, P. A., Friedman, M. J., Young-Xua, & Stevens, S. P. (2006). Cognitive Processing Therapy for veterans with military-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, *74*, 898–907.
- Orr, S., Metzger, L., Miller, M., & Kaloupek, D. (2004). Psychophysiological Assessment of PTSD. In *Assessing psychological trauma and PTSD* (2nd ed., pp. 289–343). New York: Guilford Press.
- Orr, S., & Roth, W. (2000). Psychophysiological assessment: Clinical applications for PTSD. *Journal of Affective Disorders*, *61*, 225–240.
- Patrick, C., Cuthbert, B., & Lang, P. (1994). Emotion in the criminal psychopath: Fear image processing. *Journal of Abnormal Psychology*, *103*, 523–534.
- Patrick, C., & Lang, A. (1999). Psychopathic traits and intoxicated states: Affective concomitants and conceptual links. In *Startle modification: Implications for neuroscience, cognitive science, and clinical science* (pp. 209–230). New York: Cambridge University Press.
- Pitman, R., Orr, S., Foa, D., Altman, B., de Jong, J., & Herz, L. (1990). Psychophysiological responses to combat imagery of Vietnam veterans with posttraumatic stress disorder versus other anxiety disorders. *Journal of Abnormal Psychology*, *99*, 49–54.
- Pitman, R., Orr, S., Foa, D., de Jong, J., & Claiborn, J. (1987). Psychophysiological assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry*, *44*, 970–975.
- Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: A meta-analysis. *Psychological Bulletin*, *133*, 725–746.
- Prins, A., Kaloupek, D., & Keane, T. M. (1995). Psychophysiological evidence for autonomic arousal and startle in traumatized adult populations. In M. Friedman, D. Charney, & A. Deutch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to post-traumatic stress disorder* (pp. 291–314). Philadelphia: Lippincott Williams & Wilkins.
- Raine, A., Lencz, T., Bihle, S., LaCasse, L., & Colletti, P. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Archives of General Psychiatry*, *57*, 119–127.
- Raine, A., Reynolds, C., Venables, P. H., Mednick, S. A., & Farrington, D. P. (1998). Fearlessness, stimulation-seeking, and large body size at age 3 years as early predispositions to childhood aggression at age 11 years. *Archives of General Psychiatry*, *55*, 745–751.
- Resick, P. A., & Schnicke, M. K. (1992). Cognitive Processing Therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*, 748–756.
- Schwarz, E., & Kowalski, J. (1991). Posttraumatic stress disorder after a school shooting: Effects of symptom threshold selection and diagnosis by DSM-III, DSM-III-R, or proposed DSM-IV. *The American Journal of Psychiatry*, *148*, 592–597.
- Spitzer, R. L., Williams, J. B. W., Gibson, M., & First, M. D. (1989). *Structured Clinical Interview for DSM-III-R Patient Version (SCID)*. New York: New York State Psychiatric Institute.
- Sylvers, P., Brubaker, N., Alden, S., Brennan, P., & Lilienfeld, S. (2008). Differential endophenotypic markers of narcissistic and antisocial personality features: A psychophysiological investigation. *Journal of Research in Personality*, *42*, 1260–1270.
- Taylor, J. (2004). Electrodermal reactivity and its association to substance use disorders. *Psychophysiology*, *41*, 982–989.
- Taylor, J., Carlson, S. R., Iacono, W. G., Lykken, D. T., & McGue, M. (1999). Individual differences in electrodermal responsivity to predictable aversive stimuli and substance dependence. *Psychophysiology*, *36*, 193–198.
- Weathers, F. W., Keane, T. M., & Foa, E. B. (2009). Assessment and diagnosis of adults. In E. B. Foa, T. M. Keane, M. J. Friedman, & J. A. Cohen (Eds.), *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed., pp. 23–61). New York, NY: Guilford Press.

Received May 20, 2010

Revision received November 8, 2010

Accepted November 15, 2010 ■

### **Correction to Humphreys et al. (2011)**

In the article “The Influence of Externalizing Comorbidity on Psychophysiological Reactivity Among Veterans With Posttraumatic Stress Disorder,” by Kathryn L. Humphreys, Kristen M. Foley, Brian A. Feinstein, Brian P. Marx, Danny G. Kaloupek, and Terence M. Keane (*Psychological Trauma: Theory, Research, Practice, and Policy*, Advance online publication, March 28, 2011. doi: 10.1037/a0022644), there were errors in Table 3. In the Audiovisual presentation for heart rate reactivity line, the values for PTSD should have been 2.65 [2.17, 3.12], and the values for PTSD-SUD should have been 1.85 [1.08, 2.61].

DOI: 10.1037/a0026441