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Autonomic arousal and emotion in victims of interpersonal violence: Shame proneness but not anxiety predicts vagal tone

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Abstract

The redefinition of PTSD in the DSM-5 has highlighted a range of post-traumatic affects beyond fear and anxiety. For survivors of interpersonal violence, shame has been shown to be an important contributor of self-reported symptomatology. While biological models of PTSD emphasize physiological arousal secondary to fear and anxiety, evidence suggests shame might be related to increased arousal as well. This study tested the contributions of anxiety, fear, and shame to autonomic arousal in a sample of female victims (N = 27) of interpersonal violence with PTSD. Shame proneness was the only significant correlate of autonomic arousal during a trauma-reminder paradigm. These findings indicate that shame corresponds to important indicators of changes to the autonomic nervous system, which have previously been assumed to be fear-related.

Keywords

Shame; Heart Rate Variability; Interpersonal Violence; PTSD; Complex Trauma

Described in the DSM-IV as an anxiety disorder, biological explanations of posttraumatic stress disorder (PTSD) have focused on pathological fear learning, exaggerated physiological arousal, and reduced fear modulation in response to reminders (Milad, Rauch, Pitman, & Quirk, 2006; Pole, 2007). This model, based primarily on studies of single-incident and military trauma, has been instrumental to the development of PTSD intervention (Foa, Riggs, Massie, & Yarczower, 1995). Although fear and anxiety play a central role in traumatic stress, the reach of current models has been challenged by research demonstrating that fear reactivity alone does not fully explain the heterogeneity of PTSD presentations (Lanius, Bluhm, Lanius, & Pain, 2006). Emerging evidence suggests that the relationship between trauma and symptoms may be moderated by posttraumatic reactions beyond fear (Deprince, Chu, & Pineda, 2011; Resick & Miller, 2009). In traumas where roles of dominance and subordination are enacted, such as in interpersonal violence (IPV), shame has been linked to posttraumatic symptoms (PTS; Andrews, Brewin, Rose, & Kirk, 2000; Robinaugh & McNally, 2010) including anxiety (Platt & Freyd, 2012), avoidance, and dissociation (Deprince et al., 2011). Negative trauma-related emotions such as shame are now listed as a PTSD symptom in the DSM-5 (American Psychiatric Association, 2013);

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however, to expand a fear-based biological model of PTSD, this study examines the association of fear, anxiety, and shame to physiology in a traumatized sample. Given that not all negative affects have similar biological correlates (Carver & Harmon-Jones, 2009), it is possible that physiology may function differently across different trait and state negative affects.

Shame is one in a family of "self-conscious emotions," assumed to facilitate social stability and social hierarchies (Tracy & Robins, 2007). As opposed to fear, shame involves appraisals about oneself in relationship to others. Shame is hypothesized to result from perceived social transgressions attributed to an internal, stable, and global sense of self (Tangney & Dearing, 2002). Evolutionary models posit that shame signals a threat to social status (M. Kemeny & Gruenewald, 2004). Feelings associated with shame including powerlessness, worthlessness, and desire to hide (Tangney & Dearing, 2002) may serve as signals to the boundaries of socially acceptable behavior (Deonna, Rodogno, & Teroni, 2012). Behaviors including slumped posture and downcast gaze (Tracy, Robins, & Schriber, 2009) signal submission and acceptance of one's subordinate status, to stave off aggression (Gilbert, 2000). While momentarily adaptive (de Hooge, Breugelmans, & Zeelenberg, 2008), trait shame is associated with psychopathology (Woien, Ernst, Patock-Peckham, & Nagoshi, 2003) including social anxiety (Gilbert, 2000), anger (Hejdenberg & Andrews, 2011) and poor empathy (Rangganadhan & Todorov, 2010).

Interpersonal violence (IPV) reinforces social hierarchies that engender subordination beyond that which is present in normative relationships (Dearing & Tangney, 2011). Due to the psychosocial nature of IPV, shame has been theorized to be associated with PTSD in IPV survivors (Herman, 2011; Matos & Pinto-Gouveia, 2009; Trumbull, 2003). Shame proneness (the likelihood that shame will be evoked by real or imagined social transgression) has been linked to emotion dysregulation (Orsillo, Batten, Plumb, Luterek, & Roessner, 2004), social withdrawal (Dorahy, 2010), social anxiety, and worry (Fergus, 2010). In a sample of female victims of IPV, Beck and colleagues (2011), and Street and Aria (2001) found that shame was related to symptoms of PTSD in survivors of IPV.

Perhaps uniquely among traumatic incidents, IPV constitutes a threat both to one's physical integrity and to one's evaluation of oneself as a worthwhile of social affiliation. Traditionally, the trauma exposure required to meet criteria for PTSD is conceptualized as a threat to life or physical integrity which triggers the emotions fear or anxiety. These emotions initiate organized behavioral and biological changes necessary to address this physical threat. However, physical threats such as these are not the only kinds of that are potentially dangerous. Social affiliation offers tangible survival benefits by conserving "important and often metabolically costly somatic and neural resources through the social regulation of emotion" (Beckes & Coan, 2011, pg. 976) Where fear and anxiety are signals to a threat to life or physical integrity, Kemeny and Grunewald (2004) theorize that shame signals threat to social affiliation. Shame in turn initiates organized behavioral and biological changes necessary to address this threat to one's acceptance by a social group; thus, the biological challenges arising from fear and from shame may differ.

Biological models of PTSD underscore the increased sensitivity of the autonomic nervous system (ANS). Pole's (2007) meta-analysis revealed that PTSD was associated with sympathetic nervous system (SNS) activation, indicated by higher resting heart rate (HR) and skin conductance level (SCL), and larger HR and skin conductance responses (SCR) to trauma cues. These findings are consistent with SNS elevations, but HR is under tonic inhibitory control by the parasympathetic nervous system (PNS; Berntson & Cacioppo, 1993), yielding the possibility that elevated HR is also due to diminished PNS activity.

Studies of PTSD have found reduced parasympathetic tone, measured by respiratory sinus arrthymia (RSA) at baseline and in response to trauma-related information (Cohen, Kotler, Matar, & Kaplan, 1998; Sack, Hopper, & Lamprecht, 2004), such as standardized images (Hauschildt, Peters, Moritz, & Jelinek, 2011). In PTSD, low resting RSA has been linked to physiological hyperarousal and contributes to the elevated HR observed at baseline and in response to trauma reminders (Hopper, Spinazzola, Simpson, & van der Kolk, 2006).

Phasic changes in RSA reflect the ability to adjust response to stressors. High PNS activation is theorized to be a marker for emotion regulation (Pu, Schmeichel, & Demaree, 2010) and behavioral flexibility, necessary for engaging in social interaction (Thayer & Lane, 2000). Trait negative affects, such as those in mood (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010) and anxiety disorders (Mennin, Heimberg, Turk, & Fresco, 2002) correspond to the use of ineffective emotion regulation strategies that are theoretically mediated by lower PNS tone (Shinba et al., 2008), consistent with lower RSA found in anxiety (Thayer & Lane, 2000; Friedman, 2007). However, other negative affects beyond anxiety have been associated with reductions in RSA, including anger and guilt (Bleil, Gianaros, Jennings, Flory, & Manuck, 2008).

While chronic fear-related ANS changes are well established in DSM-IV PTSD, less is known about the physiological concomitants of chronic shame. Self-esteem, related to shame proneness (Tangney & Dearing, 2002), is inversely related to resting RSA (Martens et al., 2010). Social hierarchy research has established that lower social status is associated with the presence of stress hormones indicative of SNS activation in both human community samples (Gruenewald, Kemeny, & Aziz, 2006; Rohleder, Chen, Wolf, & Miller, 2008) and among members of stigmatized groups where shame-proneness corresponds to poor health outcome (Persons, Kershaw, Sikkema, & Hansen, 2010). These endocrine findings suggest that shame may be autonomically arousing; however, autonomic correlates of shame in PTSD have not been examined.

Aberrant autonomic activity correlates with shame proneness and exposure to traumatic stress. However, there is no research on the relative contribution of shame proneness over anxiety or fear to ANS activity among trauma survivors. The relationship of shame to physiology may be especially pronounced in IPV survivors, where shame proneness mediates PTS (Andrews, Brewin, Rose, & Kirk, 2000). While the DSM-5 has added increased emphasis to negative affect in PTSD, not all negative affect has equal physiological impact. For example, some negative affect, such as anger, relates to physiological states associated with approach, and other negative affects, such as fear, relates to physiological states associated with withdrawal (Carver & Harmon-Jones, 2009).

That negative affect may be physiologically differentiated suggests that understanding the unique contribution within negative affects may be fruitful for trauma survivors. Understanding affects beyond fear and anxiety in PTSD may help generate new approaches to therapy. We predict:

(H1) Trait and state anxiety, fear, and shame will be associated with increased physiological activation, indicated by a negative correlation with RSA and a positive correlation with SCL;

(H2) Trait and state shame will contribute significant variance to physiological arousal, after accounting for the effects of anxiety and fear.

Method

Participants

Participants were 27 women seeking therapy for interpersonal trauma, aged 18 to 64 years (M= 37.5 years; SD = 13.9 range 18–64). Exclusion criteria were low levels of English literacy, presence in a violent relationship within the last 6 months, or active psychosis. No participants met exclusion criteria. The majority of participants were Caucasian (74%), and 82% reported at least some college education. The participants had varied histories of interpersonal trauma, defined as history of physical or sexual assault or abuse; the majority (52%) had experienced both physical and sexual abuse. All experienced IPV beginning in childhood, with an average first trauma exposure occurring at age 6. All participants had a probable diagnosis of PTSD, with a PCL score >44 (M = 51.61, SD = 13.5; Ruggiero, Del Ben, Scotti, & Rabalais, 2003; see AUTHOR CITATION for details on recruitment, sample, and trauma).

Materials

The *Positive and Negative Affect Schedule* (PANAS; Watson, 1988) was used to assess affect. For trait affect, participants responded on scale from 1 ("never or not often") to 5 ("very often") how often in the past month they felt a series of affect adjectives. The PANAS contains the item "ashamed." The item "scared" was used as an analogue for fear; "nervous" was used as an analogue for anxiety. Although the PANAS was designed to measure general affective domains, factor analysis has found up to nine factors (Crawford & Henry, 2004; Tellegen, Watson, & Clark, 1999). In these analyses, fear items (afraid, scared) fall on different factor than anxiety items (nervous, jittery) or the self-conscious affect items (guilty, ashamed). Despite sharing similar factor and being highly inter-correlated, work on the differing motivational properties of guilt and shame (Tangney & Dearing, 2002) make it theoretically import to distinguish these emotions.

A similar procedure was used to measure state shame: a list of adjectives was provided after the physiological task, with the prompt "please rate how much you felt each emotion while watching the slides." Items included "afraid/anxious" and "ashamed/guilty."

The *Posttraumatic Stress Disorder Checklist* (Weathers et al., 1993) is a client self-report measure of the 17 DSM-IV PTSD symptoms. Participants are asked to rate their symptoms over the past month, rated for severity and intensity on a 5-point Likert scale and then

summed to yield an overall score. PCL scores correlate highly with the gold-standard CAPS diagnostic interview (Orsillo, 2001). A modified version of the PCL rating both intensity and severity was used, to create consistency with the CAPS. Internal consistency for the scale is high (Cronbach's alpha = .94).

Physiological measures

Respiratory Sinus Arrthymia—RSA refers to the alteration of HR due to breathing (Berntson & Cacioppo, 1993). It is a reliable index of the activity of the parasympathetic nervous system (Malik & Camm, 1990), which serves to slow heart rate (HR) and return an organism to homeostasis following threat (Thayer & Lane, 2000). RSA, measured in seconds, was derived from ECG and computed by finding the difference in maximum HR during inhalation and minimum HR during exhalation. ECG data was manually inspected and artifacts where corrected or data was rejected in accordance with established methodology (Berntson et al., 1997); no participant data included indications of physiological abnormalities in ECG.

Skin Conductance—Skin Conductance (SC) is associated with changes in eccrine sweat gland activity in response to arousing stimuli and is primarily mediated by the sympathetic nervous system (Dawson, Schell, & Filion, 2000). Skin Conductance Level (SCL), which reflects tonic level of SNS activation during baseline, task, and recovery conditions.

Procedure

Resting Physiology and Slide Task—A 2 minute resting baseline was obtained for heart rate (HR, bpm) and SCL. Physiology was then continually monitored for the duration of a slide-viewing task, in which participants viewed 10 blocked slides containing trauma-related images (e.g., a man raising his fist to a woman), presented for 5 seconds each. After viewing, a 2 minute recovery period was recorded. State affect data were assessed post-recovery. (For a full explanation of procedures, see AUTHOR CITATION).

Data Analysis—Due to the small sample size and concern about type II errors, *p* values between .05 and .10 are noted as trend-level findings. Confidence intervals for r-values were estimated using 1000 bootstrap samples to guard against type I errors (Strube, 1988).

Results

Associations between demographics and study variables—Of the 27 participants, two had unusable physiology data due to equipment issues. There were no significant correlations between HR or SC and any demographic variable. There was not a significant relationship between RSA and the participants age at baseline ($r = -.309$, $p = .133$, 95% CI [−.625, .052]) or during slide viewing ($r = -.323$, $p = .115$, 95% CI [−.628, .062]), but there was a significant relationship during the recovery period ($r = -.410$, $p = .042$, 95% CI [−.665, −.049]).

Trait shame was correlated with fear ($r = .573$, p = .003, 95% CI [251, .808]) and anxiety ($r = .502$, $p = .010$, 95% CI [.118, .782]); the relationship between anxiety and fear trended

toward significance (r = .349, p < .088, 95% CI [.000, .646]). A repeated measures ANOVA was conducted to compare the effect of task on physiology. There was a significant effect of task on RSA (Wilks' Lambda = .697, $F(2,23) = 4.99$, p = .016). For SCL, Mauchly's test indicated that the assumption of sphericity had been violated, therefore a corrected value (Greenhouse-Geisser correction) of F was used. There was no effect for time on SCL ($F(2,23) = 2.83$, p = .103). Means and standard deviations are reported in Table 1.

State anxiety/fear was not associated with trait anxiety (r = .282, p = .182, 95% CI [−.068, .599]) nor with trait fear (r = −.334, p = .110, 95% CI [−.016, .638]). State shame was not associated with trait shame (r = .137, p = .524, 95% CI [−.260, .557]).

H1

Trait Shame—In partial support of H1, higher trait shame was related to lower RSA at baseline, during trauma reminders, and during recovery, indicating shame proneness was predictive of PNS withdrawal. Trait shame was not related to SCL. (See Table 2.)

State Guilt/Shame—In partial support of H1, higher state shame was associated with lower RSA at baseline (r = −.512, p = .011, 95% CI [−.724, −.293]), and during recovery (r = −.419, p = .041, 95% CI [−.688, −.188]) and the relationship approached significance during trauma reminders (r = −.391, p = .059, 95% CI [−.653, −.193]) indicating state shame was related to PNS withdrawal. State shame did not correlate with SCL during any epoch (r's = −.128 to −.025).

Trait Fear and other affects—The relationship between trait fear and RSA during baseline approached significance, but was not significant during trauma reminders or recovery. There was no relationship between SCL and fear. The relationship between trait anxiety and RSA during trauma reminders approached significance, but was not significant during baseline or during recovery. There was not a significant relationship between SCL and anxiety or between RSA, SCL, and any other trait affect. (See Table 2.)

State Fear/Anxiety—In partial support of H1, the relationship between state fear/anxiety and RSA approached significance during baseline (r = −.354, p = .089, 95% CI [−.658, −.020]) and trauma reminders (r = −.404, p = .050, 95% CI [−.652, −.048]), but not during recovery (r = −.346, p = .098, 95% CI [−.645, .033]). State fear/anxiety was not related to SCL during any epoch (r's = −.076 to −.037).

H2

Though trait and state shame were the only significant affective correlates of physiology, a series of regression analyses were performed predicting RSA from affect in epochs where fear or anxiety approached significance. Trait shame did not make a significant contribution over fear or anxiety to PNS activity during baseline, and made a marginal contribution while viewing trauma reminders. During recovery, trait shame was the only significant predictor of physiologic arousal. (See Table 3.) State shame made a significant contribution to lower RSA during baseline, but not during trauma reminders or recovery. (See Table 4.)

Post hoc analysis

Previous studies have found a relationship between PTS and reductions in RSA in other samples with PTSD. Post-hoc mediation analysis was performed to examine the role of shame in this relationship. A mediation model was not supported. Participants' PTS symptoms were significantly related to trait shame, $B = -.009$, $SE\ B = .003$, B = .486, p = .014, but there was no relationship between PTS and PNS (baseline: $B = -.001$, $SE\ B = .001$, B = −.181, p = .387; slides: $B = -.000$, $SE\ B = .001$, B = −.060, p = .776; recovery: $B = -.000$, $SE\ B = .001$, B = −.154, p = .461).

Discussion

Anxiety and fear influence the course and maintenance of PTSD, but for survivors of IPV, there is emerging evidence of the contribution of shame to symptoms. While biological models of PTSD capture the importance of physiological arousal secondary to fear and anxiety, little data has focused on the interplay between other negative affects and physiological dysregulation. This study tested the contributions of anxiety, fear, and shame to physiological arousal at baseline and in response to trauma reminders. PNS deactivation was associated with fear and shame at baseline, anxiety and shame during trauma reminders, and shame alone during recovery. Shame marginally predicted larger SNS responses when viewing trauma related information, but fear and anxiety did not.

Shame, fear, and anxiety predicted reduced PNS activation during baseline and while viewing trauma reminders. These findings confirm and extend the role of negative trait affect in baseline physiology and physiological reactivity in PTSD (Andrews, Brewin, Rose, & Kirk, 2000). Trait shame was the only predictor of RSA during recovery, indicating shame-prone individuals were less able to recover from the effects of trauma reminders. State shame was the only significant predictor of RSA during the task, and added significant variance over fear/anxiety at baseline.

This low baseline RSA in those with state shame, and failure to recover in the shame prone individuals, conforms to a profile of inflexible autonomic regulation which has been associated with failures of emotion regulation more generally (Thayer & Lane, 2000). These findings bridge a gap between several lines of research that separately connect IPV to PTS via either shame (Andrews et al., 2000) or difficulties with emotion regulation (Ehring & Quack, 2010; Goldsmith, Chesney, Heath, & Barlow, 2013; Orsillo, Batten, Plumb, Luterek, & Roessner, 2004). While traditionally clinicians have looked for symptoms such as hyper arousal, in this population, shame may indicate a failure of emotion regulation that indexes physiological dysregulation. Moreover, phasic changes in PNS activity are associated with a suite of other regulatory actions including affective processing, attention regulation and the behavioral flexibility necessary for successful social interaction (Geisler, Kubiak, Siewert, & Weber, 2013; Park, Van Bavel, Vasey, & Thayer, 2012). Shame may affect symptomatology in survivors of IPV by compromising the effectiveness of help-seeking behaviors (Black, Curran, & Dyer, 2013) or disrupting other interpersonal processes (Covert, Tangney, Maddux, & Heleno, 2003). Similarly, the tendency to withdraw inherent in shame may lead to social isolation (Kim, Thibodeau, & Jorgensen, 2011) which corresponds to lower RSA

(Grippo, Lamb, Carter, & Porges, 2007). Shame and RSA likely have reciprocal influence on one another.

It was notable that the data did not reveal an association between affect and SNS activation, as measured by SCL. This finding may be accounted for by a restriction of range of SCL responses in this highly-traumatized sample. It is also possible that the task was not evocative of psychological states associated with increased SCL, such as fear and anxiety, but rather evoked withdrawal-related affects that may not recruit the SNS.

Shame was related to self-reported trauma symptomatology and autonomic arousal, but there was no relationship between PNS tone and reported PTS. The unexpected lack of association between PTS and autonomic activation further highlights the importance of shame with respect to the physiology of this sample. Shame was the only significant correlate of SNS reactivity to trauma reminders. The distinctive relationships between shame, PTSD, and the PNS highlights the complexity of the relationship between affect, symptom, and physiology and suggests the importance of measuring trait affect in a variety of populations with PTSD, particularly as the PTSD diagnosis has been revised to incorporate negative affect (DSM-5, 2013). One possible interpretation is that the affective label of shame is more salient than fear or anxiety among trauma survivors, even if it is not necessarily their most prominent emotional experience.

This study had several limitations. The correlational nature of the analysis and lack of a comparison group limit the conclusions that can be drawn from the findings. In addition to a small sample size, this population is known to be shame-prone (Shorey et al., 2011), and other populations with a different trauma exposure profile for which guilt or fear are more salient emotions may have different responses (Beck et al., 2011). Further, while the use of the PANAS offers advantages in terms of face validity and convenience, it was not designed to measure a range of affects and there are several limitations to using a single item on the PANAS to measure trait shame and trait guilt. For example, there is reason to believe that participants may not be able to adequately differentiate a dispositional tendency towards feelings of shame from guilt using this method (See Tangney and Dearling (2002) Chapter 3 for a full discussion of the value of different measures of shame and guilt). One possible interpretation is that the affective label of shame is more salient than fear or anxiety among trauma survivors, even if it is not necessarily their most prominent emotional experience.

While the state measures suggest a similar pattern of autonomic response as the trait measures, there are several limitations to the use of the state questionnaires in this study. As noted earlier, state shame and guilt are theorized to be distinct affective states and the combination of state shame and guilt items limits our ability to interpret the contribution of state shame to physiology. Furthermore, trait and state fear where correlated, but trait and state shame where not. This may be due to the inclusion of guilt in the stare measure, but may also provide evidence that that the measurement tools may lack construct validity. These limitations highlight the need for brief self-report assessments of state and trait shame and guilt.

Overall, these findings provide support to the effort to integrate emotions other than fear and anxiety into models of PTSD (Sack et al., 2004). While shame may not be more important among trauma survivors than other affects, these data suggest it be given significant weight. These findings highlight the importance of measuring shame in this population in understanding the course and maintenance of symptoms in this group, consistent with the addition of negative trauma-related emotions such as shame to PTSD criteria in DSM-5. A purely fear-based model of PTSD overlooks other potential sources of arousal that may affect symptoms that range from startle response to social functioning. From a treatment perspective, treating hyperarousal secondary to shame may require different approaches than treating arousal caused by fear. To date there is little solid evidence of how to target and reduce shame and shame related attributions. Given the complex interpersonal and cognitive nature of shame, it may not be amenable to the gold standard treatment of Exposure Therapy. If shame represents a failure of emotion regulation, therapies that teach these skills may be more effective in targeting shame related symptomatology.

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Table 1

Descriptive Statistics for Physiology and Affect Variables.

	M	SD	Min	Max
RSA				
Baseline	0.08	0.06	.00	.21
Trauma	0.07	0.06	.00	.26
Recovery	0.08	0.06	.00	.25
SCL				
Baseline	4.13	1.77	.74	7.95
Trauma	−0.03	0.04	−.13	.09
Recovery	3.89	1.46	1.56	6.66
PANAS				
Distressed	3.16	1.24	1	5
Upset	3.60	1.55	1	5
Hostile	2.08	0.95	1	5
Irritable	3.00	1.19	1	5
Scared	3.12	1.09	1	5
Afraid	2.96	1.27	1	5
Ashamed	2.40	1.19	1	5
Guilty	2.50	1.14	1	5
Nervous	3.40	1.29	1	5
Jittery	2.56	1.19	1	5
State Affect				
Fear/Anxiety	2.67	1.04	1	4
Shame/Guilt	1.42	1.38	0	4

Note: RSA = Respiratory Sinus Arrhythmia, arbitrary units; SCL = Skin Conductance, μSiemens; PANAS = Positive and Negative Affect Schedule.

Table 2

Correlations Among Trait Affect and Physiology

	RSA			SCL		
***r*, 95% CI**	**Baseline**	**Trauma**	**Recovery**	**Baseline**	**Trauma**	**Recovery**
Distressed	.074	.026	.027	−.053	.042	.042
Lower Limit	−.270	−.306	−.361	−.440	−.373	−.377
Upper Limit	.413	.362	.400	.340	.476	.508
Upset	.185	.148	.239	−.054	.141	.105
Lower Limit	−.211	−.295	−.194	−.394	−.217	−.274
Upper Limit	.556	.545	.598	.324	.554	.576
Hostile	−.159	−.141	−.170	−.148	−.158	−.168
Lower Limit	−.464	−.445	−.505	−.487	−.511	−.507
Upper Limit	.165	.166	.169	.390	.303	.307
Irritable	.002	−.031	−.036	.094	.309	.344
Lower Limit	−.419	−.468	−.468	−.346	−.075	−.048
Upper Limit	.480	.434	.430	.541	.665	.697
Scared	−.348	−.153	−.145	−.018	.134	.135
Lower Limit	−.678	−.614	−.555	−.401	−.235	−.234
Upper Limit	.123	.292	.269	.446	.541	.540
Afraid	−.025	−.041	.059	.188	.227	.190
Lower Limit	−.382	−.397	−.319	−.150	−.176	−.235
Upper Limit	.375	.428	.468	.539	.615	.622
Ashamed	−.400*	−.427*	−.503*	−.032	.015	.006
Lower Limit	−.661	−.658	−.745	−.343	−.341	−.343
Upper Limit	−.045	−.080	−.160	.304	.372	.381
Guilty	−.194	−.255	−.234	.231	.245	.226
Lower Limit	−.519	−.569	−.567	−.108	−.138	−.167
Upper Limit	.247	.346	.234	.551	.618	.614
Nervous	−.292	−.383	−.279	.147	.039	.025
Lower Limit	−.636	−.670	−.591	−.257	−.387	−.402
Upper Limit	.183	.151	.206	.513	.509	.496

	RSA			SCL		
***r*, 95% CI**	**Baseline**	**Trauma**	**Recovery**	**Baseline**	**Trauma**	**Recovery**
Jittery	−.185	−.227	−.072	−.218	−.022	.004
Lower Limit	−.508	−.515	−.436	−.528	−.367	−.344
Upper Limit	.352	.360	.439	.125	.354	.363

Notes. N's range from 24 to 25 due to occasional missing PANAS data. RSA = Respiratory Sinus Arrhythmia, SC = Skin Conductance, PANAS = Positive and Negative Affect Schedule .

$^{*}p < .05$.

Table 3

The contribution of shame to variance in RSA throughout the task, accounting for variance contributed by fear and anxiety.

	Baseline RSA				Trauma RSA				Recovery RSA			
Variable	***B***	***SE B***	**B**	**ΔR^2**	***B***	***SE B***	**B**	**ΔR^2**	***B***	***SE B***	**B**	**ΔR^2**
Model 1				.10				.15				.08
Scared	−.019	.015	−.271		−.001	.012	−.023		−.003	.012	−.055	
Nervous	−.006	.013	−.104		−.017	.010	−.375^{+}		−.012	.010	−.260	
Model 2				.05				.10^{+}				.22*
Scared	−.009	.018	−.126		.010	.013	.192		.015	.013	.257	
Nervous	−.001	.013	−.024		−.012	.010	−.253		−.004	.010	−.082	
Ashamed	−.019	.017	−.287		−.021	.013	−.426^{+}		−.032	.013	−.620*	

$^{+}$ = $p < .1$,

* = $p < .05$.

Note: RSA = Respiratory Sinus Arrhythmia.

Table 4

The contribution of state shame to variance in RSA throughout the task, accounting for variance contributed by state fear/anxiety.

	Baseline RSA				Trauma RSA				Recovery RSA			
Variable	***B***	***SE B***	**B**	**ΔR^2**	***B***	***SE B***	**B**	**ΔR^2**	***B***	***SE B***	**B**	**ΔR^2**
Model 1				.12				.16+				.12+
Fear/Anxiety	−.022	.012	−.354		−.023	.011	−.404		−.021	.012	−.246	
Model 2				.15*				.26				.08
Fear/Anxiety	−.008	.013	−.136		−.016	.013	−.228		−.011	.013	−.185	
Shame/Guilt	−.021	.010	−.445*		−.011	.010	−.424+		−.015	.010	−.329	

+ = $p < .1$,

* = $p < .05$.

Note: RSA = Respiratory Sinus Arrhythmia.