Prospective Prediction of PTSD Symptoms Using Fear Potentiated Auditory Startle Responses

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Abstract

Background—PTSD has been most consistently associated with exaggerated physiological reactivity to startling sounds when such sounds occur in threatening contexts. There is conflicting evidence about whether startle hyperreactivity is a pre-existing vulnerability factor for PTSD or an acquired result of post-trauma neural sensitization. Until now, there have been no prospective studies of physiological reactivity to startling sounds in threatening contexts as predictors of PTSD symptoms.

Methods—One hundred and thirty-eight police academy cadets without current psychopathology were exposed to repeated 106 dB startling sounds under increasing (low, medium, or high) threat of mild electric shock while their eyeblink electromyogram, skin conductance, heart rate, and subjective fear responses were recorded. Measures of response habituation were also calculated. Following one year of exposure to police-related trauma, these participants were assessed for PTSD symptom severity.

Results—After accounting for other baseline variables that were predictive of PTSD symptom severity (age and general psychiatric distress), more severe PTSD symptoms were prospectively and independently predicted by the following startle measures: greater subjective fear under low threat, greater skin conductance under high threat, and slower skin conductance habituation.

Conclusions—These results imply that hypersensitivity to contextual threat (indexed by greater fear under low threat), elevated sympathetic nervous system reactivity to explicit threat (indexed by larger responses under high threat), and failure to adapt to repeated aversive stimuli (evidenced by slower habituation) are all unique pre-existing vulnerability factors for greater PTSD symptom severity following traumatic stress exposure. These measures may eventually prove useful for preventing PTSD.

Keywords

PTSD; psychophysiology; prospective; police; acoustic startle

Introduction

Long before posttraumatic stress disorder (PTSD) was a recognized psychiatric diagnosis, distressed trauma survivors complained about being easily startled (1). As the diagnostic criteria for PTSD has evolved, "exaggerated startle" has remained a frequently endorsed symptom of the disorder (2). Furthermore, PTSD has been associated with objective hyperstartle indicators such as larger eyeblink responses (the short latency primary index of human startle), larger electrodermal and cardiac responses (longer latency secondary startle responses), and slower habituation of electrodermal responses (a tertiary measure indexing reduced responding over repeated stimulus presentations) (3,4). The primacy of the eyeblink response and the robustness of the secondary and tertiary measure findings (4) justifies the continued examination of all of these measures in PTSD.

Exaggerated startle in PTSD was originally interpreted as resulting from either a proneness to excessive unconditioned responding to aversive stimuli (5,6) or classical conditioning of traumatic experiences with coinciding startling sounds (e.g., gunfire). However, it became clear that PTSD patients do not show elevated reactivity to all aversive stimuli (7) and that those exhibiting elevated startle reactivity have not necessarily experienced trauma involving loud sounds (3). Recent thought posits that startle hyper-reactivity in PTSD is not an abnormal response to sudden intense stimuli but rather an augmented fear response to the context in which such stimuli occur. Though the startle reflex is implemented by a simple brainstem circuit, its magnitude and the magnitude of its secondary autonomic components may be potentiated by input from brain regions activated by threat cues and fear (8,9). Under *contextual threat* (i.e., settings in which the patient anticipates an aversive event), PTSD has been consistently associated with stronger startle eyeblink responding that appears to be independent of explicit threat cues (e.g., signals indicating precisely when the shock will occur) and relatively impervious to safety cues (e.g., signals indicating that the shock will not occur) (10–13). Similar results have been observed for electrodermal responses to startling sounds (13). However, in the absence of contextual threat, exaggerated eyeblink findings in PTSD have been less consistent (6,14). Moreover, hypersensitivity to threatening contexts has been implicated in increased conditionability (i.e., more rapid learning and slower extinction of associations between previously neutral cues and aversive stimuli) (15), which lends further plausibility to contextual threat sensitivity as a vulnerability factor for developing PTSD.

It is unknown, however, whether elevated startle responding is a pre-existing vulnerability factor for PTSD or is an acquired result of either trauma exposure or the development of other PTSD symptoms. Evidence has emerged on both sides of the argument. Orr et al. (16) startled 130 Vietnam combat veterans and their non-combat exposed monozygotic twins and found that the veterans with PTSD exhibited greater heart rate responses to startling sounds than their non-exposed, genetically identical twin. This implied that the augmented heart rate responses were not pre-existing but rather acquired following trauma. Shalev et al. (17) measured startle reactivity in 218 trauma survivors within one week of their trauma exposure and again four months later, at which time they were diagnosed with or without PTSD. The PTSD group showed no startle response differences one week after trauma but then demonstrated larger heart rate responses and slower eyeblink and skin conductance habituation four months later. Griffin (18) found similar results in a study of 40 female interpersonal violence survivors who were exposed to startling sounds within one month post-trauma and then again at six months post-trauma when they were also diagnosed with or without PTSD. Neither initial eyeblink

nor heart rate responses predicted later PTSD diagnoses but such diagnoses were accompanied by elevations in both physiological measures at the six month time point. These studies favor the view that abnormal startle responding is not a pre-existing vulnerability factor for PTSD but rather emerges along with PTSD symptoms, perhaps through a process of post-trauma progressive neuronal sensitization.

Yet, there are two important limitations to the aforementioned studies. First, none of them assessed startle prior to trauma exposure. Guthrie and Bryant (19), on the other hand, measured startle-related eyeblink and skin conductance responses in firefighter trainees before they experienced duty-related trauma and found that both physiological measures prospectively predicted acute PTSD symptoms within 30 days of trauma exposure suggesting that startle may be a vulnerability factor. Second, no prospective study has manipulated contextual threat during startle assessment. If exaggerated startle actually indexes elevated fear due to contextual threat sensitivity then such threat might be necessary to reveal a prospective relationship between startle responses and PTSD. Therefore, it is unclear whether the Guthrie and Bryant results or previous failures to find a prospective relationship between startle and PTSD could be explained by the presence or absence of contextual threat.

The present study was undertaken to address both of these limitations by determining the relationship between pre-trauma startle reactivity under contextual threat and PTSD symptom severity after one year of exposure to police-related trauma. Contextual threat was established by warning cadets that they would receive a mild electric shock at a specific time during the study. Such uncontrollable but predictable aversive situations have been shown to increase generalized fear in animal models of PTSD (20). The cadets were startled as their threat of being shocked was systematically increased. Exaggerated startle responses under low and medium threat of shock were conceptualized as primarily indexing contextual threat sensitivity. Exaggerated startle responses under high threat of shock were conceptualized as primarily indexing explicit threat sensitivity. We hypothesized that more severe PTSD symptoms at 12 months would be predicted primarily by contextual threat sensitivity and secondarily by explicit threat sensitivity. We also hypothesized that PTSD symptom severity would be predicted by slower habituation of startle responding (especially slower skin conductance habituation).

Methods and Materials

Participants

Cadets were recruited from urban police academies in New York and California. The study was approved by Institutional Review Boards at the University of California, San Francisco and the San Francisco Veterans Affairs Medical Center. A Federal Certificate of Confidentiality protected participants from future subpoena and disclosure to their respective police departments. Cadets who were combat veterans or who had prior experience in law enforcement or emergency services were not accepted into the study. Among the 292 cadets who received a complete study description, provided written informed consent, and enrolled in the larger research program, several were excluded from the present study for the following reasons: meeting criteria for a current mental disorder or using medications during the initial assessment ($n = 21$), missing or corrupted psychophysiological data ($n = 33$), not completing the 12 month follow-up assessment ($n = 64$), or not reporting a traumatic event meeting criterion A1 for PTSD within the first year of police service (n = 36).[1] This resulted in a final sample of 138 officers for analysis with minimal missing data. There were no significant differences

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[1]Participants were not excluded for failing to meet the A2 criterion for PTSD because police officers tend to minimize emotional distress and because strict PTSD diagnoses were not a focus of this study.

between the final sample and the originally enrolled sample in measured variables other than those that served as the basis for exclusion.

Assessment Procedures

While in the police academy, each cadet was assessed by a trained doctoral level clinician for Axis I disorders using the *Structured Clinical Interview for DSM-IV* (SCID; 21) and for prior trauma using the *Life Stressor Checklist-Revised* (LSC-R; 22). Cadets also responded to demographic questions and completed the *Social Desirability Scale* (SDS; 23) and the *Symptom Checklist 90-Revised* (SCL-90-R; 24). Prior trauma exposure was defined as the sum of endorsed *LSC-R* life threatening event items. Social desirability bias was defined as the sum of *SDS* items endorsed in a socially desirable direction. General psychiatric distress was defined as the mean rating of all *SCL-90-R* items (i.e., the General Severity Index). These measures provided means of determining and adjusting for the potential influence of confounding variables on the subsequent development of PTSD symptoms.

The cadets also participated in our startle procedure (13,25). They were instructed to refrain from exercise, cigarettes, and coffee on the startle day and from eating for one hour prior to the procedure. Their compliance and their medication use were assessed by self report. Trained research technicians, who were blind as to the participant's psychometric status, collected the psychophysiological data. The participant's left eyeblink electromyogram (EMG) activity, skin conductance (SC) level, and heart rate (HR) were assessed during a two minute resting baseline period. Participants were fitted with headphones and told that they would hear potentially startling sounds. They were asked to sit in a chair and to keep their eyes focused (except when blinking) on a monitor a few feet in front of them. A Coulbourn Instruments Lablinc V Modular System binaurally presented 106-dB(A), 40 ms white noise bursts with 0-ms rise and fall times separated by intertrial intervals of between 30 and 50 seconds under low, medium, and high threat of shock. Under *low threat*, participants were instructed that they would not be shocked until later in the study. They were then exposed to ten startling sounds. Only their last five responses were retained. Under *medium threat*, participants were fitted with a Coulbourn Instruments Transcutaneous Aversive Finger Stimulator but were told that they would not yet be shocked. Five additional startling sounds were presented. Previous work (13,25,26) indicated that the mere placement of the finger stimulator increases threat and startle reactivity. Under *high threat*, the cadets continued to wear the finger stimulator and they were signaled that shocks were imminent as five additional startling sounds were presented followed by an annoying (but not painful) 2.5 mA shock. Each condition lasted approximately 4 minutes and was separated by about 1 minute. The medium and high threat conditions were counterbalanced to minimize carry-over effects between these conditions. Participants completed ratings of subjective fear following the resting baseline and each threat condition on a scale from 1 = none to 5 = quite a lot. All physiological signals were sampled at 2 Hz during the resting baseline and at 1000 Hz during the acoustic presentations, digitized, and stored for off-line analysis, *EMG* was measured in microvolts using three, 4-mm (sensor diameter) In Vivo Metrics Ag/AgCl surface electrodes filled with electrolyte paste. Two electrodes were placed on the left orbicularis oculi according to published specifications (27) and the ground electrode was placed on the center of the forehead. Impedance levels were kept below 10 KOhms. The EMG signal was amplified, rectified, filtered to retain the 13 to 1000 Hz range, notch filtered at 60 Hz, and smoothed by applying a 5-ms time constant, *SC* was measured in microsiemens by sending a constant 0.5 V through 9-mm (sensor diameter) InVivo Metrics Ag/AgCl electrodes filled with isotonic paste and placed on the hypothenar surface of the medial phalanges of the middle and index fingers of the nondominant hand. HR was measured in beats per minute and recorded via electrodes attached in a Type-I EKG configuration.

Human Startle Software (Coulbourn Instruments, Allentown, PA) automatically calculated mean psychophysiology at baseline and during the one second prior to each stimulus onset. It also calculated the peak post-stimulus levels within 21 to 200 milliseconds for eyeblink EMG and within 1 to 4 seconds for SC and HR. These data were inspected for potential artifact and rejected accordingly. Response scores were calculated by subtracting pre-stimulus mean values from post-stimulus peak values. No minimum response threshold was designated for any physiological measure. Participants needed at least four (of five) valid responses for each condition and for all three physiological measures to be included in the study. Individual response scores were square root transformed to reduce heteroscedasticity and skewness. Transformed response scores were averaged for each physiological measure within each threat condition. Relative habituation scores were also calculated for each physiological measure by determining the slope (b) of the regression equation $Y = bX + a$, in which Y was defined as the square root of physiological response scores 2 through 10 obtained prior to the placement of the finger stimulator and X was defined as the natural log of the corresponding trial number.

One year after the startle testing, participants completed self-report measures including the *Critical Incident History Questionnaire* (CIHQ) (28) to assess duty-related trauma exposure and the *Posttraumatic Stress Disorder Checklist* (PCL) (29) to assess current PTSD symptom severity stemming from their worst duty-related traumatic event. All index traumatic events included in the study had occurred more than one month prior to the 12 month assessment ($M = 5.4$, $SD = 3.5$ months) and were thus beyond consideration for acute stress disorder. Duty-related CIHQ items meeting criterion A1 for PTSD were tallied to derive an overall exposure score. PTSD symptom severity was defined as the sum of all PCL item ratings. Officers were financially compensated following each wave of participation.

Data Analyses

Measures assessed while the officers were in the police academy were correlated with 12 month PTSD symptom severity to identify potentially confounding variables.[2] Main hypotheses were tested using multiple regression analyses with potentially confounding variables entered in the initial steps. Concerns about type I and type II errors were balanced by following a two-stage approach recommended by Cohen and Cohen (30) in which multivariate "omnibus" tests were conducted on sets of related predictors (i.e., resting baseline; low threat, medium threat, high threat; and habituation) and only further decomposed to constituent univariate tests when the omnibus test was significant. Because this two-stage approach has been empirically shown to protect against excessively large type I error rates, a two-tailed threshold of $\alpha=.05$ for statistical significance was used for the omnibus tests without further alpha correction. We determined which startle variable was of primary importance in predicting PTSD symptom severity by conducting a stepwise multiple regression analysis with forward selection of all measures showing a univariate relationship with PTSD symptom severity. Analyses were conducted using SPSS 14.0.

Results

Characteristics of the Sample and Their Responses to the Startle Task While in the Police Academy

Demographic, psychometric, and psychophysiological characteristics of the sample during police academy training are given in Table 1. This table shows that the sample was ethnically diverse (47.1% Caucasian American, 14.5% Asian American, 7.2% African American, 15.9%

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[2]We also conducted a series of exploratory bivariate correlations between the demographic, psychometric, and startle measures obtained at baseline and subclusters of PTSD symptoms (i.e., self reported startle, intrusion, avoidance, and hyperarousal symptoms) observed at the 12 month time point. Because of limited journal space these analyses are presented as an online supplement to this article.

Latino American, 15.2% Mixed or Other) predominantly male, under 30 years old, and partially college educated. Analyses of variance revealed that the threat manipulation led to significant increases in EMG (p <.001), SC (p <.001), HR (p <.01), and subjective fear (p <.001) responses to the startling sounds.[3]

Trauma Exposure and PTSD Symptoms After One Year of Police Service

When the sample was assessed after one year of police service, they had experienced an average of 7.0 (SD = 4.1) potentially traumatic duty-related critical incidents including: encountering a dying or dead body (87.6%), threat with a deadly weapon (27.0%), witnessing an officer's injury or death (20.4%), being seriously injured (12.4%), having to shoot or kill someone (7.3%), being shot (5.1%), or being involved in a serious accident (5.1%). Nonetheless, the officers reported very low levels of PTSD symptoms. Their average PCL total score was 19.8 (SD = 5.8) on a scale that ranges from 17 to 85. Only one officer reported symptoms above the recommended cut score for full PTSD and only three others could be considered "partial" PTSD cases on the basis of having met two of the B through D criteria for PTSD. This small number of PTSD cases supported our decision to focus on predicting PTSD symptom severity rather than diagnosis.

Predictors of Total PTSD Symptom Severity After One Year of Police Service

Table 1 shows the correlations between variables assessed in the academy and total PTSD symptom severity at 12 months. Among demographic and psychometric variables, we found that cadets who were older and who had higher general psychiatric distress reported more severe PTSD symptoms one year later suggesting that these might be potentially confounding variables. Table 2 shows the results of our main hypothesis tests. After adjusting for age, initial general psychiatric distress, and prior trauma exposure, we found that 12 month PTSD symptom severity was significantly predicted by startle measures obtained under low threat, ΔR^2 =.14, F(4, 127) =5.95, p <.001; medium threat, ΔR^2 =.06, F(4, 128) =2.57, p <.05; high threat, ΔR^2 =.11, F(4, 125) =4.67, p <.01; and the habituation measures, ΔR^2 =.06, F(3, 129) =2.97, p <.05. Examination of the regression coefficients associated with the individual predictors comprising the significant omnibus tests revealed that only the following variables were predictive of 12 month PTSD symptom severity: SC and subjective fear under low threat; eyeblink EMG and subjective fear (as a trend) under medium threat; SC under high threat; and SC habituation slope.

The stepwise multiple regression analysis aimed at determining which of the significant predictors from the prior models best predicted PTSD symptom severity revealed that after adjusting for age, initial psychiatric symptom severity, and prior trauma exposure (which together accounted for 13.9% of the variance), the following measures were most predictive (in order): subjective fear under low threat (9.1% of the variance), SC responses under high threat (8.0% of the variance), and SC habituation slope (3.3% of the variance). Neither eyeblink responses under medium threat nor SC responses under low threat added significantly to this final model, which, in sum, accounted for 34.4% of the variance (Adjusted R^2 =.31) in 12 month total PTSD symptom severity.

Discussion

This study supported our hypotheses that startle variables collected under contextual threat would primarily prospectively predict elevated PTSD symptom severity. Consistent with Guthrie and Bryant (19), we found that both eyeblink and skin conductance responses predicted increased PTSD severity. In accordance with the other studies (16–18), we did not find a

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[3]See the online supplement for further details about the manipulation check including an examination of order effects.

prospective relationship between heart rate responses to startling sounds and PTSD symptom severity. Consonant with other PTSD studies involving contextual threat, PTSD was associated with greater eyeblink responses under medium threat (11–13,26) and greater skin conductance responses under low threat (13). Our results are also in line with an animal study that found that rats with pre-existing exaggerated startle showed the highest level of PTSD-like symptoms after experiencing a trauma-like laboratory stressor (32).

These findings support the view that startle-related differences pre-date the development of PTSD symptoms, perhaps because of unmeasured variables such as genetic predisposition or early neuropsychiatric impairments. Heightened subjective fear only significantly predicted PTSD symptoms at the lowest threat of shock, which suggests either overactivation of fear and/or difficulty inhibiting fear in the presence of contextual threat (13,25). It is noteworthy that elevated fear under low threat was the most important predictor in the final model and that its inclusion eliminated the predictive influence of both low and medium threat physiological responses. Thus, in effect, elevated pre-trauma fear under contextual threat explained the elevated physiological responses under the low and medium threat conditions. The fact that larger skin conductance responses under high threat explained additional PTSD symptom variance suggests an unexpected additional vulnerability factor of hyperactive sympathetic nervous system (SNS) responses to an explicit threat (33). The SNS has been implicated in the etiology of PTSD by other investigators (34) because of its potential to increase peritraumatic responding and consolidation of trauma memories. The final predictor in the model, slower skin conductance habituation, suggests a third vulnerability factor involving a failure of SNS adaptation to repeated aversive stimuli, which has also been implicated in the etiology of PTSD because of its strong heritability (35), association with conditionability (36), and robustness as a correlate of PTSD (4). Taken together, our results imply that a combination of hypersensitivity to potentially threatening contexts, heightened sympathetic nervous system reactivity to explicit threat, and failure to regulate the sympathetic nervous system response all incrementally contribute to risk for heightened PTSD symptoms.

The minor inconsistencies between our present findings and our earlier work (13) (e.g., the absence of a correlation between SC and PTSD under medium threat and the presence of such a correlation under high threat in the present study) may be related to the fact that the former study examined a sample that was both more highly exposed to trauma and displaying more severe PTSD symptoms than the present sample. Thus, this difference could reflect changes that occur as PTSD symptoms become more severe and chronic. The bigger discrepancies between our conclusion that increased startle reactivity is a pre-trauma vulnerability factor and the seemingly opposite conclusion reached by others (16–18) may be due to methodological differences between our study and prior work. For example, the Orr et al. twin study did not involve a threat manipulation. In addition, the other two studies that viewed increased startle as a post-trauma acquisition assessed startle shortly after (rather than before) the index trauma and predicted PTSD diagnosis rather than PTSD symptom severity. If one wishes to determine whether exaggerated startle is a risk factor for PTSD then startle assessment before trauma exposure is more compelling than assessment shortly after trauma. Startle responding may be altered in the immediate aftermath of a trauma in unknown ways. On the other hand, predicting PTSD diagnostic status would have been more compelling than predicting PTSD symptom severity. Our results and those of Guthrie and Bryant might pertain only to subclinical symptoms and may not extend to the actual diagnosis. We were unable to address this issue because of low rates of full PTSD in our sample. It is also unclear what proportion of this sample will spontaneously remit and what proportion will go on to have chronic PTSD symptoms. However, these issues may become resolved as we continue to follow these police officers for the next several years during which time their PTSD rates are expected to rise (37,38).

The conclusions which may be drawn from our study are limited by the nature of the sample and its reliance on self-report measures of PTSD symptoms. Police officers may differ in important ways from other populations to which one might wish to generalize these results. Self-report measures are susceptible to witting or unwitting distortions and may underestimate PTSD symptoms in a sample of early career police officers who are highly motivated to appear resilient in the face of stress. Thus, it is reassuring that we found no evidence of social desirability reporting biasing the PTSD symptom reporting. Nonetheless, the study has a number of strengths including the fact that the sample was ethnically diverse, carefully characterized, and physiologically assessed prior to their index trauma making this one of the few truly prospective studies of acoustic startle and PTSD. The study was also unique in incorporating contextual threat into the startle assessment procedures. As such, it adds to accumulating evidence that threat sensitivity may be a vulnerability factor for a range of anxiety and mood disorders (39,40). Findings from this line of investigation may eventually assist with identifying first responders at higher risk for psychopathology in order to efficiently direct limited resources for resilience training and early intervention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

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Table 1

Demographic, Psychometric, and Psychophysiological Characteristics of Police Cadets During Training and Their Correlation with Self-Reported PTSD Symptoms After One Year of Police Service

Variables Assessed During Police Training	Descriptive Statistics M (SD), N (%)	Correlation (r) Self-Reported PTSD Symptoms After One Year of Police Work
Age (years)	27.3 (4.8)	.24[**]
Education (years)	15.2 (1.5)	.00
Male Gender	120 (87.0%)	.11
Ethnic Minority	73 (52.9%)	.02
Social Desirability	9.2 (2.2)	−.08
Prior Trauma Exposure	2.4 (1.7)	.07
General Psychiatric Distress	.12 (.14)	.31[**]
Resting Baseline		
EMG (microvolts)	10.90 (13.30)	.10
SC (micromohs)	12.59 (7.37)	−.09
HR (beats per minute)	65.78 (9.79)	−.04
Subjective Fear (1 − 5)	1.22 (.54)	.08
Low Shock Threat		
EMG ($\sqrt{\Delta}$microvolts)	5.37 (3.44)	.14
SC ($\sqrt{\Delta}$micromohs)	.35 (.32)	.21[*]
HR ($\sqrt{\Delta}$ beats per minute)	.96 (1.56)	−.03
Subjective Fear (1 − 5)	1.22 (.49)	.39[**]
Medium Shock Threat		
EMG ($\sqrt{\Delta}$ microvolts)	5.84 (3.74)	.18[*]
SC ($\sqrt{\Delta}$ micromohs)	.37 (.29)	.13
HR ($\sqrt{\Delta}$ beats per minute)	1.43 (1.55)	−.02
Subjective Fear (1 − 5)	1.25 (.55)	.23[**]
High Shock Threat		
EMG ($\sqrt{\Delta}$ microvolts)	6.77 (4.25)	.16
SC ($\sqrt{\Delta}$ micromohs)	.46 (.34)	.27[**]
HR ($\sqrt{\Delta}$ beats per minute)	1.42 (1.46)	−.13
Subjective Fear (1 − 5)	1.62 (.89)	.11
Habituation Measures		
EMG Response Slope	−1.03 (1.80)	.02
SC Response Slope	−.22 (.21)	.20[*]
HR Response Slope	−.27 (1.96)	.02

Note. PTSD = Posttraumatic Stress Disorder. EMG = Eyeblink electromyogram. SC = Skin conductance. HR = Heart rate. Prior Trauma Exposure was assessed using the *Life Stressor Checklist-Revised*. General Psychiatric Distress was measured using the General Severity Index (GSI) of the *Symptom Checklist-90-Revised*. PTSD symptom severity was measured the total score of the *Posttraumatic Stress Disorder Checklist* (PCL).

[*] p <.05.

[**] p <.01.

[***] p <.001.

Table 2

Final Linear Regression Models Predicting 12 Month Total PTSD Symptom Severity After Adjusting For Age, Initial General Psychiatric Distress, and Prior Trauma Exposure

Variable	B	S.E.	β	t
Resting Baseline				
Age	.27	.11	.22	2.56[*]
General Psychiatric Distress	12.10	3.63	.29	3.33[***]
Prior Trauma Exposure	−.08	.29	−.02	−.28
Eyeblink EMG	.03	.04	.07	.89
Skin Conductance Level	−.08	.07	−.10	−1.16
Heart Rate	−.02	.05	−.03	−.33
Subjective Fear	.50	.95	.04	.52
Low Shock Threat				
Age	.19	.09	.16	2.00[*]
General Psychiatric Distress	9.42	3.39	.22	2.78[**]
Prior Trauma Exposure	.05	.26	.01	.18
Eyeblink EMG Response	.09	.14	.05	.68
SC Response	3.66	1.41	.20	2.60[**]
HR Response	−.25	.29	−.07	−.88
Subjective Fear	3.57	.94	.30	3.78[***]
Medium Shock Threat				
Age	.23	.10	.19	2.31[*]
General Psychiatric Distress	11.49	3.53	.27	3.25[***]
Prior Trauma Exposure	−.02	.27	−.01	−.09
Eyeblink EMG Response	.25	.12	.16	2.03[*]
SC Response	2.27	1.65	.11	1.37
HR Response	−.23	.30	−.06	−.77
Subjective Fear	1.48	.87	.14	1.70[+]
High Shock Threat				
Age	.21	.09	.17	2.22[*]
General Psychiatric Distress	13.84	3.48	.32	3.97[***]
Prior Trauma Exposure	.12	.27	.04	.45
Eyeblink EMG Response	.16	.11	.12	1.53
SC Response	4.99	1.36	.29	3.66[***]
HR Response	−.54	.31	−.13	−1.73[+]
Subjective Fear	.11	.54	.02	.21
Habituation Response Slope				
Age	.24	.10	.20	2.51[*]
General Psychiatric Distress	14.12	3.49	.33	4.05[***]
Prior Trauma Exposure	−.06	.27	−.02	−.22
Eyeblink EMG Response Slope	−.31	.27	−.10	−1.17

Variable	B	S.E.	β	t
SC Response Slope	6.45	2.20	.24	2.93[**]
HR Response Slope	−.03	.24	−.01	−.13
Best Predictors				
Age	.16	.09	.13	1.76[+]
General Psychiatric Distress	10.86	3.24	.25	3.35[***]
Prior Trauma Exposure	.13	.25	.04	.51
Subjective Fear (Low Threat)	3.54	.89	.30	3.98[***]
SC Response (High Threat)	5.01	1.25	.29	4.00[***]
SC Response Slope	5.03	1.97	.19	2.56[*]

Note. PTSD = Posttraumatic Stress Disorder. EMG = Electromyogram. SC = Skin Conductance. HR = Heart Rate.

[+] $p < .10$.

[*] $p < .05$.

[**] $p < .01$.

[***] $p < .001$.