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De Novo Conditioning in Trauma-Exposed Individuals With and Without Posttraumatic Stress Disorder

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Differential conditioning was assessed in 15 medication-free individuals meeting *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) criteria for chronic posttraumatic stress disorder (PTSD) and 18 trauma-exposed individuals who never developed PTSD (non-PTSD). Conditioned stimuli (CSs) were colored circles, and the unconditioned stimulus was a "highly annoying" electrical stimulus. Individuals with PTSD had higher resting heart rate (HR) and skin conductance (SC) levels and produced larger SC orienting responses. During conditioning, the PTSD group showed larger differential SC, HR, and electromyogram responses to the reinforced vs. nonreinforced stimuli (CS+ vs. CS−) compared with the non-PTSD group. Only PTSD participants continued to show differential SC responses to CS+ vs. CS− during extinction trials. Results suggest that individuals with PTSD have higher sympathetic nervous system arousal at the time of conditioning and are more conditionable than trauma-exposed individuals without PTSD.

Individuals with a current diagnosis of posttraumatic stress disorder (PTSD) consistently have been found to produce larger physiologic responses, including heart rate (HR), skin conductance (SC), and facial electromyogram (EMG), when exposed to cues reminiscent of their traumatic event(s), compared with individuals who have experienced similar events but did not develop PTSD. This increased responsivity is shown by individuals with PTSD resulting from a wide range of traumatic events, including combat (Orr, Pitman, Lasko, & Herz, 1993; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987), childhood sexual abuse (Orr et al., 1998), motor vehicle accidents (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994), and other noncombat traumas (Shalev, Orr, & Pitman, 1993).

Conditioning theory (e.g., Pitman, 1988) provides a framework for understanding the process through which trauma-related cues could acquire the capacity to evoke large emotional responses in some individuals. In this model, various cues, or conditioned stimuli (CSs), present at the time of the traumatic event become associated with the "intense fear, helplessness, or horror" generated by the traumatic event and thereby acquire the capacity to evoke strong emotional responses on subsequent occasions (*Diagnostic and Statistical Manual of Mental Disorders,* 4th ed.; *DSM–IV;* American Psychiatric Association, 1994). Heightened physiologic reactivity to trauma-related cues does not appear to result from generally increased reactivity. PTSD has not been found to be associated with heightened physiologic responsivity to generic stressors such as mental arithmetic (e.g., Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986; Orr, Meyerhoff, Edwards, & Pitman, 1998), personal imagery of stressful (nontraumatic) life events (Orr et al., 1993; Pitman et al., 1987; Shalev et al., 1993), or postural change or cold pressor challenge (Orr et al., 1998).

The conditioning model of PTSD helps to explain how PTSD might develop in trauma-exposed individuals. However, it does not explain why upon exposure to comparably severe traumatic events (i.e., unconditioned stimuli [UCSs]), some individuals develop PTSD whereas others do not. Here the concept of *conditionability* may be useful. If some PTSD symptoms may be understood as conditioned responses (CRs) to a traumatic UCS, then individuals who are more prone to acquire CRs in the first place would be more likely to develop such symptoms. It is also possible that a difference in conditionability could manifest as slower extinction of a CR, in the presence of normal acquisition. For example, generalized anxiety disorder patients, compared with nonanxious individuals, showed comparable acquisition but

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slower extinction of a conditioned SC response to angry facial expression CSs (Pitman & Orr, 1986).

A recent study of conditioning in Gulf War veterans reported enhanced conditioning to contextual but not to explicit fear cues in veterans with PTSD (Grillon & Morgan, 1999). Eyeblink response magnitude to a startle probe was used to assess CR strength; measures of autonomic activity were not obtained. In the first of two conditioning sessions separated by 4 or 5 days, veterans with PTSD showed a generalized fear response to the CS− (stimulus not paired with the UCS) and CS+ (stimulus paired with the UCS) and thereby failed to acquire a differential CR. In the second session, the PTSD group acquired a differential CR comparable to that of the non-PTSD group. These findings suggest, as noted by Grillon and Morgan, that individuals with PTSD show a heightened generalized fear response and have difficulty learning to identify safety cues.

In this study, we examined the issue of conditionability in individuals with and without PTSD. We used a differential conditioning procedure to assess the acquisition and extinction of autonomic and corrugator EMG responses to emotionally neutral stimuli. An attempt was made to make the UCS, a mild electric shock, comparably aversive across individuals by asking each to set a level that was "highly annoying, but not painful." This is important because a group difference in unconditioned response (UCR) magnitude could confound interpretation of larger CRs, more rapid acquisition, or slower extinction, because such effects might be expected from a stronger UCR.

Method

Participants

The sample consisted of 24 men and 9 women with exposure to various traumatic events including combat experiences in Vietnam (n = 15), war-related nursing in Vietnam (n = 8), physical assault (n = 2), firefighting or emergency medical technician (EMT) experiences (n = 7), or motor vehicle accident (n = 1). Potential participants were recruited from the population of active and former Veterans Affairs (VA) outpatients, Vet Center clients, mailings to Vietnam nurse veterans, Professional Firefighters of New Hampshire, posted notices at EMT stations, and advertisements in the media. Each research candidate underwent a diagnostic interview for the presence of Axis I mental disorders using the Structured Clinical Interview for *DSM–IV* (First, Spitzer, Gibbon, & Williams, 1994). No candidate met *DSM–IV* criteria for organic mental disorder, schizophrenia, or current manic syndrome. On the basis of the Clinician-Administered PTSD Scale (Blake et al., 1995), participants who met *DSM–IV* criteria for current PTSD were classified into the PTSD group (10 men, 5 women); those who did not meet criteria for current or past PTSD were classified into the non-PTSD group (14 men, 4 women). Eight individuals in the PTSD group (53%) had one or more current comorbid Axis I disorders as follows: four major depression, one cyclothymia, one dysthymia, two panic without agoraphobia, one panic with agoraphobia, one agoraphobia without history of panic disorder, one specific phobia, and one hypochondriasis. Two of the non-PTSD participants (11%) had one or more current Axis I disorders as follows: one dysthymia, one undifferentiated somatoform, and one alcohol abuse. Participants were free from psychoactive medications and drugs at the time of psychophysiologic testing, as determined from self-report and urine drug screens.

Measures

Psychometric

The psychometric measures included the Mississippi Scale for Combat-Related PTSD or its civilian version (Keane, Caddell, & Taylor, 1988), Impact of Event Scale (Horowitz, Wilner, & Alvarez, 1979), State–Trait Anxiety Inventory (Spielberger, Gorsuch, & Lushene, 1970), Beck Depression Inventory (Beck, Rush, Shaw, & Emery, 1979), and Symptom Checklist–90–Revised (Derogatis, 1983). One of three scales was used to assess the amount of trauma exposure, depending on the nature of the traumatic experience. Combat veterans completed a Combat Exposure scale (Keane et al., 1989), Vietnam nurse veterans completed the Women's War-Time Stressor Scale (Wolfe, Brown, & Furey, 1993), and firefighter–EMTs completed a scale developed at our laboratory for this study. The latter instrument consisted of 11 items that identified potential job-related stressors, which were rated on 5-point scales according to how often they had been experienced. Scores from the various trauma exposure scales were transformed so that the range of possible scores was the same for each scale (i.e., 0–100). The amount of trauma exposure was not assessed in the 3 participants who each experienced a single traumatic event (i.e., a physical assault or motor vehicle accident).

Psychophysiologic

A Coulbourn Modular Instrument System (Allentown, PA) was used to record physiologic analog signals, which included SC, HR, and left corrugator EMG. For EMG, the skin was abraded, and 4-mm (sensor diameter) Sensor Medics Ag/AgCl electrodes were filled with electrolyte paste and placed over the corrugator muscle site according to published specifications (Fridlund & Cacioppo, 1986). The EMG was amplified by a Coulbourn High Gain Bioamplifier (S75-01), filtered to retain the 90- to 250-Hz frequency range, and integrated by a Coulbourn Contour Following Integrator (S76-01) with a 300-ms time constant; this corresponds to a setting of 500 on the integrator dial. SC was measured directly by a Coulbourn Isolated Skin Conductance coupler (S71-23) using a constant 0.5 V through 9-mm (sensor diameter) Sensor Medics Ag/AgCl electrodes placed on the hypothenar surface of the participant's nondominant hand in accordance with published guidelines (Fowles et al., 1981). The SC electrodes were separated by 14 mm, as determined by the width of the adhesive collar. HR was recorded by standard limb electrocardiogram leads connected to a High Gain Bioamplifier (S75-01) inputting to a Coulbourn Tachometer (S77-26). Physiologic analog signals were digitized by a Coulbourn Lablinc Analog to Digital Converter (L25-12). An IBM-compatible computer system was used for sampling and storing the digitized physiologic signals.

Procedure

The experimental session took place in a humidity- and temperature-controlled, sound-attenuated room, connected by wires to an adjoining laboratory in which the experimental apparatus was located. The participant was seated in a comfortable armchair and monitored by an unobtrusive video camera. The CS+ and CS− were represented by two differently colored 6-in. (15.2-cm) diameter circles, randomly selected for each participant from four options (red, blue, white, or green). The colored CSs were computer generated and displayed on a monitor positioned 4 ft (1.2 m) in front of the participant. The UCS was a 500-ms electric shock previously determined by the participant to be "highly annoying but not painful," delivered through electrodes attached to the second and third fingers of the dominant hand, and generated by a Coulbourn Transcutaneous Aversive Finger Stimulator (E13-22), which was isolated from line current and used a 9-V dry cell battery attached to an adjustable step-up transformer.

On arrival at the laboratory, the participant was reminded that a mild electric stimulus would be used and that, if necessary, he or she would be free to terminate the experiment at any time. Physiologic recording electrodes and those for administering the electrical UCS were then attached. Prior to setting the UCS level, the technician gave the following instructions:

> For this experiment, you will set your own level of electric stimulation. You should choose a level that is highly annoying but not painful. I will start the stimulation at a very low level and gradually increase the level until you say "stop." The level that you set will then be used throughout the remainder of the experiment.

The technician then proceeded to set the UCS level and noted the final dial setting of the transformer (ranging from 0.2 to 4.0 mA); this provided a measure of the UCS intensity selected by the participant.

Once the UCS level was established, the participant was given the following instructions:

> This experiment will consist of a baseline period followed by three phases. During the baseline period, which will last 5 min, we will check our instruments and you should try to relax. At the end of this period, you will see "Begin Phase I" displayed on the monitor. During this phase, two different colored circles will be presented on the monitor. You should sit quietly and look at each colored circle as it is presented. At the end of the period, "Begin Phase II" will appear on the monitor. During this phase, the colored circles will be presented again, and some of them will be followed by the electrical stimulus. Again, you should sit quietly and look at each colored circle as it is presented. At the end of Phase II, "Begin Phase III" will appear on the monitor. During this phase, you will see more colored circles. However, you will no longer receive any electrical stimulation. Please continue to sit quietly and look at each colored circle as it is presented. It is important that you watch the screen at all times. Do you have any questions?

When the participant was ready to proceed, the technician left the room and activated the computer, which took over the administration of the experiment. There was a 5-min baseline recording period during which the dependent measures were sampled at 2 Hz. The habituation phase (Phase I) consisted of five presentations each of the to-be CS+ and CS− in pseudorandom order (i.e., there were no more than two consecutive presentations of the same stimulus type). The CS duration was 8 s, and the intertrial interval was 20 ± 5 s, determined at random by the computer. During the acquisition phase (Phase II) a 500-ms shock pulse occurred immediately following each CS+ offset. There were five presentations of each stimulus type. The extinction phase (Phase III) consisted of 10 nonreinforced presentations of the CS+ and CS−. The dependent physiologic measures were sampled at 10 Hz, beginning 2 s prior to CS onset and ending 6 s following CS offset.

At the completion of the extinction phase, the technician reentered the participant's room, removed the physiologic recording electrodes, and proceeded with the debriefing. As part of the debriefing, participants were asked, "Were you able to predict when the shock would occur?" They were then asked to identify the particular color of the circle paired with the shock, if this information was not spontaneously given.

Response Scores

Because SC has been the most useful physiologic variable in the principal investigator's prior conditioning work and because of its wide application in the kind of human aversive conditioning used here, it was the measure of primary interest. An SC response score for each CS interval was calculated by subtracting the mean level for the 2 s immediately preceding CS onset from the highest value among those recorded during the 8-s CS interval. An SC response score for the interval containing the UCR was calculated by subtracting the average SC level within 6–8 s following CS onset, from the maximum increase in SC level during the 0.5–6.5-s interval following CS offset (which corresponded to the onset of the 0.5-s UCS). Data reduction for HR and corrugator EMG paralleled that for SC.

Results

Demographics, Psychometrics, Debriefing, Resting Physiologic Levels, and UCS Level

Means, standard deviations, and results of *t*-test comparisons between the PTSD and non-PTSD groups for the various demographic and psychometric measures, the UCS level set by the participant, and resting physiologic levels are presented in Table 1. The groups did not differ in age, education level, or amount of trauma exposure. Overall, the PTSD group showed significantly more PTSD-specific and general psychopathology than the non-PTSD group. Also, the PTSD group had higher resting HR and SC levels, and selected a lower level for the UCS, than the non-PTSD group.

Results of the debriefing indicated that 13 of 13 PTSD and 12 of 14 non-PTSD participants correctly identified the relationship between the CS+ and shock (Fisher's exact probability test, $p = .48$, two-tailed). Because collection of these data was added during the performance of the study, they were not available for the first 6 (2 PTSD, 4 non-PTSD) participants.

Conditioning Procedure

The group mean SC, HR, and corrugator EMG response scores for the CS intervals of CS+ and CS− trials during the habituation, acquisition, and extinction phases are presented in Figure 1A. Analyses of variance for repeated measures (ANOVARs) were separately conducted for the three phases. The ANOVAR model included three variables: diagnosis (PTSD, non-PTSD), which was analyzed as a between-subjects effect; stimulus type (CS+, CS−), which was analyzed as a within-subjects effect; and trials, which served as the repeated measure. The Trials variable contained 5 levels (5 CS+ and 5 CS−) for the habituation and acquisition phases and 10 levels for the extinction phase. The ANOVAR results for the diagnosis and stimulus variables are presented in Table 2; significant effects involving the trials variable are presented in the text. All significance levels reported for analyses that included the trials variable reflect the Greenhouse–Geisser correction for sphericity.

Habituation Phase

As can be seen in Table 2, the PTSD group produced significantly larger SC responses to the CSs compared with the non-PTSD group. No other main effects or interactions for SC, HR, or EMG responses during the habituation phase reached statistical significance.

An estimate of SC orienting response (OR) magnitude was obtained by averaging each individual's response to the first presentation of the CS+ and the CS−. The mean SC OR was significantly larger in the PTSD group ($M = .37$, $SD = .29$), than in the non-PTSD group ($M = .12$, $SD = .22$), $t(31) = 2.8$, $p = .008$.

Table 1
Group Means, Standard Deviations, and Results of t Tests for the Demographic and Psychometric Measures, and Resting Physiologic Levels

	PTSD (n = 15)		Non-PTSD (n = 18)		t tests	
Measure	M	SD	M	SD	$t(31)$	p
Age	46.8	8.8	45.2	11.5	0.4	.67
Education	14.5	2.8	14.9	1.9	−0.5	.62
Trauma exposure (0–100)	51.9	25.2	42.5	17.4	1.2	.24
Impact of Event Scale						
Intrusion (0–35)	20.3	9.4	7.0	8.8	4.1	<.001
Avoidance (0–40)	24.1	9.9	6.6	8.8	5.3	<.001
Mississippi Scale (35–175)	112.3	21.2	68.0	14.1	6.9	<.001
CAPS total score (0–136)	75.2	20.7	3.9	5.7	13.7	<.001
STAI						
Trait (20–80)	54.6	13.3	29.6	6.8	6.8	<.001
State (20–80)	48.5	17.3	28.6	8.4	4.2	<.001
SCL-90-R, GSI (0–4)	1.5	1.0	0.4	0.3	4.5	<.001
BDI (0–63)	17.6	11.5	5.8	5.3	3.7	<.001
Level of UCS (0–4)	1.9	0.7	2.6	1.1	−2.3	.03
Physiologic resting levels						
Skin conductance (μS)	6.8	4.1	4.1	2.8	2.3	.03
Heart rate (BPM)	77.9	12.9	66.2	7.3	3.3	.002
Corrugator EMG (μV)	4.3	2.1	3.5	1.9	1.1	.27

Note. In cases where the civilian version of the Mississippi Scale was administered, only 35 items were scored (i.e., those that are also included in the combat version). Because of unavailable trauma exposure data for 3 participants, the t-test comparison had 28 degrees of freedom. PTSD = posttraumatic stress disorder; CAPS = Clinician-Administered PTSD Scale; STAI = State–Trait Anxiety Inventory; SCL-90-R = Symptom Checklist–90–Revised; GSI = Global Severity Index; BDI = Beck Depression Inventory; UCS = unconditioned stimulus; BPM = beats per minute; EMG = electromyogram.

Acquisition Phase

CS interval responses. As can be seen from Figure 1A and Table 2, the PTSD group showed larger SC, HR, and EMG responses to CS+ versus CS− trials, compared with the non-PTSD group (Diagnosis × Stimulus interactions). The groups also showed different patterns of SC responding across trials, as indicated by a significant Diagnosis × Stimulus × Trials interaction, $F(4, 124) = 3.8$, $p = .02$. The difference in SC response to CS+ versus CS− trials increased over trials for the PTSD group, whereas it initially increased and then decreased for the non-PTSD group. The PTSD group also produced overall larger SC responses to CS+ and CS− acquisition trials.

The significant stimulus effects for SC and EMG responses were explored by using separate ANOVARs for each group. These analyses revealed a significantly larger mean SC response to the CS+ versus CS− trials for both the PTSD, $F(1, 14) = 31.0$, $p < .001$, and non-PTSD, $F(1, 17) = 7.5$, $p = .01$, groups. The PTSD group also showed a larger mean EMG response to CS+ versus CS− trials, $F(1, 14) = 7.7$, $p = .02$, whereas the non-PTSD group did not ($F < 1$). Separate analyses for HR data revealed a larger mean HR response to CS+ versus CS− trials for the PTSD, $F(1, 14) = 7.7$, $p = .02$, but not the non-PTSD, $F(1, 17) = 1.7$, $p = .21$, group. Aside from a significant Trials main effect for HR, $F(4, 124) = 4.4$, $p = .01$, no other main effects or interactions were significant.

UCS interval responses. Group mean SC, HR, and EMG responses during the UCS intervals of CS+ and CS− acquisition phase trials are presented in Figure 1B. An examination of the data across both groups showed that the shock UCS produced a measurable UCR for each of the physiologic measures. The SC, HR, and EMG responses to the UCS were significantly larger than responses during the UCS intervals of nonreinforced trials, all $Fs(1, 31) > 8.4$, $ps < .01$. The magnitudes of the SC and EMG UCRs were comparable in the PTSD and non-PTSD groups as shown by nonsignificant Diagnosis × Stimulus interactions, $Fs(1, 31) \leq 2.8$, $ps > .10$. However, the PTSD group produced a larger mean HR UCR compared with the non-PTSD group, $F(1, 31) = 12.1$, $p = .002$.

Extinction Phase

The PTSD group produced larger SC responses to the CSs during the extinction phase. This was not simply a result of larger responses to CS+ trials, as the PTSD group's mean SC response to CS− trials was also significantly larger than that of the non-PTSD group, $F(1, 31) = 4.8$, $p = .04$. As can be seen from Table 2, the combined group data showed clear evidence of larger SC responses to CS+ versus CS− trials, but no differences for HR or EMG responses. Greater differential conditioning in the PTSD group was evident in their larger mean SC response to CS+ versus CS− trials (Diagnosis × Stimulus interaction), compared with the non-PTSD group. Separate ANOVARs indicated that the PTSD group showed a significantly larger mean SC response to CS+ versus CS− extinction trials, $F(1, 14) = 10.6$, $p = .006$, whereas the non-PTSD group did not ($F < 1$). The PTSD group continued to show evidence of a conditioned SC response even into later extinction trials, as indicated by a significant Diagnosis × Stim-

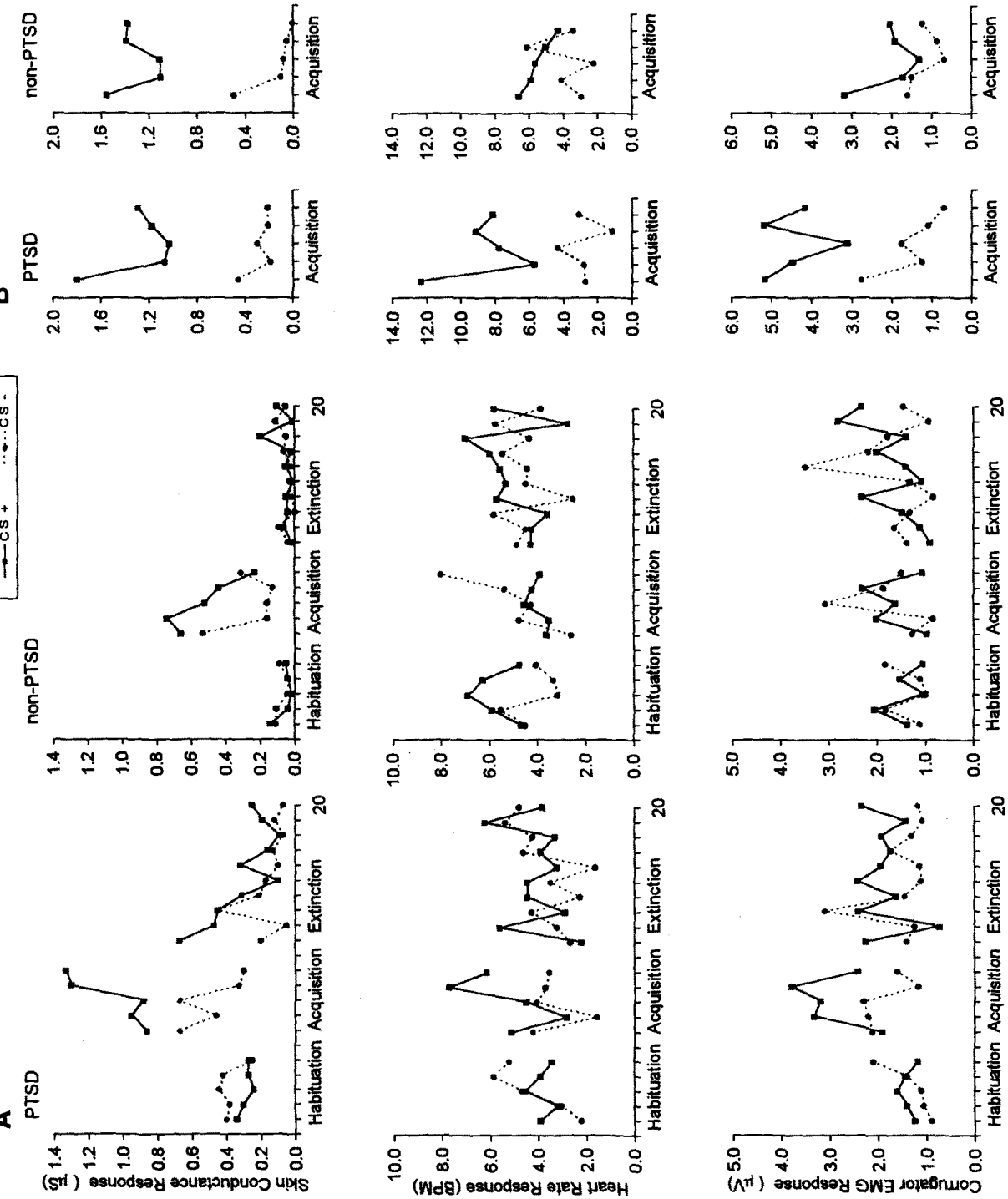

Figure 1. Posttraumatic stress disorder (PTSD; $n = 15$) and non-PTSD ($n = 18$) group mean skin conductance; heart rate; and corrugator electromyogram (EMG) response scores for the conditioned stimulus (CS) intervals (A) of CS+ and CS− trials during the habituation, acquisition, and extinction phases, and the unconditioned stimulus intervals (B) of CS+ and CS− trials during the acquisition phase. BPM = beats per minute.

Table 2
ANOVA Results for CS Interval Data

	ANOVA					
	Diagnosis		Stimulus		Diagnosis × Stimulus	
Experimental phase	$F(1, 31)$	p	$F(1, 31)$	p	$F(1, 31)$	p
Habituation						
Skin conductance (μS)	10.5	.003	3.9	.06	1.7	.20
Heart rate (BPM)	<1	*ns*	<1	*ns*	2.2	.15
Corrugator EMG (μV)	<1	*ns*	<1	*ns*	<1	*ns*
Acquisition						
Skin conductance (μS)	4.4	.04	35.5	<.001	5.1	.03
Heart rate (BPM)	<1	*ns*	<1	*ns*	7.3	.01
Corrugator EMG (μV)	<1	*ns*	4.4	.04	7.0	.01
Extinction						
Skin conductance (μS)	8.9	.006	12.0	.002	9.2	.005
Heart rate (BPM)	<1	*ns*	<1	*ns*	<1	*ns*
Corrugator EMG (μV)	<1	*ns*	1.7	.20	1.0	.31

Note. Diagnosis effect = PTSD versus non-PTSD; stimulus effect = CS+ trials versus CS− trials; ANOVA = analysis of variance; CS = conditioned stimulus; BPM = beats per minute; EMG = electromyogram; PTSD = posttraumatic stress disorder.

ulus interaction, $F(1, 14) = 5.5$, $p = .03$; their mean SC response for the last five CS+ trials was larger than that for the last five CS− trials.

Analyses of Covariance (ANCOVA)

An ANCOVA for repeated measures was used to test the possibility that the PTSD group's larger differential SC responses to CS+ versus CS− trials during the acquisition and extinction phases could be attributed to heightened physiologic arousal level or responsivity, or the tendency to report a higher amount of trauma exposure. Potential covariates included SC resting level, pretrial SC level during the acquisition phase, SC OR, and trauma exposure score. The pretrial SC level during the acquisition phase was calculated by averaging the 2-s pretrial SC levels across all CS+ and CS− acquisition trials. The mean pretrial SC level was significantly higher in the PTSD ($M = 8.0$ μS, $SD = 4.4$, than in the non-PTSD group ($M = 4.6$ μS, $SD = 3.3$), $t(31) = 2.5$, $p = .02$. The three physiologic covariates, but not trauma exposure, showed a significant relationship with the differential SC response to CS+ versus CS− acquisition trials, $F(2, 29) \geq 3.4$, $ps < .05$; only the mean SC OR (habituation phase) showed a significant relationship with the differential SC response to extinction trials, $F(2, 29) = 3.8$, $p = .03$. The covariate regression lines were parallel in all cases.

Results of ANCOVAs for the acquisition phase revealed that the Diagnosis × Stimulus × Trials interaction remained significant after adjusting for SC resting level, $F(4, 120) = 5.6$, $p = .002$, pretrial SC level, $F(4, 120) = 4.6$, $p = .008$, and trauma exposure, $F(4, 108) = 3.5$, $p = .02$, and nearly so after adjusting for SC OR, $F(4, 120) = 2.7$, $p = .06$. The overall magnitude of the differential SC response to CS+ versus CS− trials differed between the PTSD and non-PTSD groups after adjusting for trauma exposure, as revealed by a significant Diagnosis × Stimulus interaction, $F(4, 27) = 4.7$, $p = .04$, but did not differ after adjusting for SC resting level, $F(1, 30) = 1.9$, $p = .18$, pretrial SC level, $F(1, 30) < 1$, and SC OR, $F(1, 30) < 1$. Results of the ANCOVA for the extinction phase revealed that the differential SC response to CS+ versus CS− trials (i.e., the Diagnosis × Stimulus interaction) remained significantly larger in the PTSD group after adjusting for SC resting level, $F(1, 30) = 5.5$, $p = .03$, pretrial SC level, $F(1, 30) = 5.0$, $p = .03$, and trauma exposure, $F(1, 27) = 11.6$, $p = .002$; there was a trend toward a difference after adjusting for the mean SC OR, $F(1, 30) = 2.9$, $p = .10$.

Correlational Analyses

Pearson correlations were computed among the various indices of SC activity because SC appeared to be the most informative physiologic channel and because it has been commonly used in studies of human conditioning. As can be seen in Table 3, resting SC level showed significant positive relationships with mean SC OR (habituation phase), UCR, and differential CR during acquisition. The mean SC OR magnitude showed positive correlations with the mean UCR and differential CRs during acquisition and extinction. The mean UCR was positively correlated with the differential CR during acquisition.

Discussion

The findings of this study provide evidence that a de novo aversively conditioned electrodermal response to a previously neutral CS was acquired more strongly and was more resistant to extinction in individuals with PTSD, compared with trauma-exposed individuals without the disorder. Greater conditionability as observed here provides one explanation for why some individuals might acquire the intense and persistent negative emotional responses that characterize PTSD, whereas other individuals might not, when they appear to have been exposed to comparably stressful events. In the present study, the PTSD group showed stronger conditioning than the non-PTSD group even though individuals in both groups had previously set presumably comparable levels for

Table 3
Pearson Correlations Among Selected Skin Conductance Variables

SC measure	1	2	3	4	5	6
1. Resting level	—	.48**	.38*	−.38*	.46**	.26
2. OR		—	.39*	−.13	.61***	.40*
3. UCR			—	−.01	.52**	.07
4. Habituation differential				—	−.14	−.18
5. Acquisition differential					—	.21
6. Extinction differential						—

Note. SC = skin conductance; Resting level = mean during 5-min rest period; OR = orienting response, or averaged response to first presentation of the CS+ and the CS− during the habituation phase; UCR = averaged unconditioned response for CS+ trials during the acquisition phase; habituation, acquisition, and extinction differentials = the averaged CS interval response to CS+ trials minus the averaged CS interval response to CS− trials for the habituation, acquisition, and extinction phases, respectively; CS = conditioned stimulus.
* $p < .05$. ** $p < .01$. *** $p < .001$.

the UCS (i.e., levels that were "highly annoying"). The pattern of SC responses shown by the PTSD group (i.e., SC response magnitudes that became increasingly larger across CS+ acquisition trials) was strikingly different from that of the non-PTSD group. Anticipation of the shock UCS seemed to produce increasingly strong emotional responses over trials for individuals with PTSD. Thus, fearfulness increased for the PTSD group as the CS+ acquired signal value for predicting the UCS, whereas fearfulness decreased as the UCS became more predictable for individuals without PTSD. This may represent a fundamental difference in the way that individuals with and without PTSD emotionally process threat cues.

The PTSD group showed a higher level of arousal than the non-PTSD group at the onset of the study, manifested in elevated SC and HR levels during the rest phase and larger SC ORs during habituation trials. This heightened arousal may reflect an increased sensitivity to threatening contexts or aversive stimulation, which could have contributed to the PTSD group's stronger differential conditioning. Research using nonclinical samples has found that anxious individuals show a greater relative increase in the number of CRs in a context made threatening by the possibility of receiving mild electric shocks, compared with a nonthreatening context (Spence, Farber, & Taylor, 1954). Other nonclinical research has reported that individuals who show larger ORs tend to condition more strongly (e.g., Zeiner & Schell, 1971); however, not all studies have observed such a relationship (e.g., Öhman & Bohlin, 1973a).

In the present study, correlational analyses indicated that higher resting SC levels were associated with larger SC ORs during habituation trials and larger differential SC responses to CS+ versus CS− trials during acquisition, and that larger SC ORs were predictive of larger differential SC responses during acquisition and extinction. The results of ANCOVAs that adjusted for SC level and OR magnitude revealed that the magnitude of the differential SC response during acquisition did not differ between the PTSD and non-PTSD groups, whereas it did differ in the unadjusted analyses. Thus, the PTSD group's higher level of arousal may explain their overall larger differential SC responses to CS+ versus CS− acquisition trials. However, two of the key group differences observed in the present study do not appear to be attributable to heightened arousal. The pattern of SC response magnitudes that became increasingly larger across CS+ acquisition trials, and the larger magnitude differential response during extinction trials shown by the PTSD group, persisted even after adjusting for SC level and OR magnitude.

The present findings suggest that individuals with PTSD show heightened conditionability (i.e., under comparable conditions individuals with PTSD acquire a larger and more persistent autonomic differential response to an aversive CS, compared with nonpsychiatric trauma-exposed individuals who never developed PTSD). The origin of this heightened conditionability is not clear. However, it does not appear that the PTSD group's heightened conditionability can be attributed to better cognitive learning of the relationship between the CS+ and UCS. Results of the debriefing indicated that upon being asked, nearly all participants accurately identified this relationship. Furthermore, an examination of the SC findings during acquisition (see Figure 1) indicates that both groups quickly showed a differential SC response to CS+ versus CS− trials; this differential response was apparent by the second presentation of each stimulus type.

There remains a possibility that the PTSD group's heightened conditionability resulted from a greater sensitivity to the aversive UCS. The PTSD and non-PTSD groups produced comparably large SC responses to the UCS, suggesting that the UCS was equally aversive for the two groups. However, the UCS produced a larger HR response in the PTSD group, thereby suggesting that the UCS was not experienced in precisely the same way by the two groups. It remains to be seen whether individuals with PTSD will also show heightened conditioning to an appetitive UCS, and whether this conditionability extends to nonphysiologic measures of emotion.

The present PTSD group's stronger differential conditioning to an explicit fear cue indicates that the CS+ and CS− were readily discriminated (i.e., the PTSD participants had no difficulty recognizing the CS− as a safety signal). This finding stands in contrast to that of Grillon and Morgan (1999), whereby individuals with PTSD produced a generalized fear response to the CSs and failed to acquire a differential CR in the first of two sessions. These discrepant findings could simply reflect idiosyncrasies of the two PTSD samples. Alternatively, it may be that autonomic measures, particularly SC, provide a more sensitive index of an aversive CR in humans than does eyeblink startle response magnitude. It is also possible, as suggested by Grillon and Morgan, that autonomic measures and startle magnitude assess different emotional pro-

cesses. The present findings highlight the importance of including autonomic indices in studies that assess conditioned emotional responses. Furthermore, they support the inclusion of multiple autonomic indices, given that SC and HR produced different patterns of results.

The pattern of electrodermal results shown by the present PTSD group (i.e., a higher resting level, larger ORs to initial presentations of the CSs, larger responses to the CSs during extinction trials, and stronger differential conditioning) is suggestive of increased emotionality. Emotionality has been variously conceptualized in terms such as anxiety, neuroticism, and electrodermal arousal or lability. McFarlane (1988) has reported higher neuroticism scores in firefighters who developed PTSD. Individuals identified as being more emotional have typically been found to condition more strongly. For example, clinically anxious individuals showed slower extinction of a conditioned SC response to angry facial expressions, compared with nonanxious individuals (Pitman & Orr, 1986). In nonclinical samples, Öhman and Bohlin (1973b) and Hugdahl, Fredrikson, & Öhman (1977) observed that individuals with high levels of electrodermal arousal showed better aversive conditioning. Eyeblink conditioning, using an air puff UCS, has been found to be stronger in individuals with a high score on a self-report measure of trait anxiety, compared with those with lower anxiety scores (Spence & Farber, 1953; Spence et al., 1954; Spence & Taylor, 1951). Also, acquisition of a conditioned eyeblink response was found to be faster in college undergraduates who scored high on a measure of obsessive–compulsive behavior, compared with individuals with lower scores (Tracy, Ghose, Stecher, McFall, & Steinmetz, 1999). These various findings suggest that the heightened differential conditioning observed in the present study's PTSD group may not reflect a unique feature of PTSD; rather, it may be a common feature of all anxiety disorders. However, in light of the sparse literature that has examined conditioning in clinically anxious individuals, this possibility remains to be tested.

The PTSD and non-PTSD groups in the present study produced comparably large SC responses to the UCS. However, the PTSD group's HR responses to the UCS were larger than those of the non-PTSD group. Individuals with PTSD have also been found to produce larger HR responses to brief auditory stimuli of high (Orr, Lasko, Shalev, & Pitman, 1995; Orr, Solomon, Peri, Pitman, & Shalev, 1997) and moderate intensity (Orr, Lasko, Metzger, & Pitman, 1997), even though SC response magnitudes did not differ from those of the non-PTSD groups. This increased HR responsivity has been interpreted as evidence of an abnormal defensive response in individuals with PTSD (e.g., Orr, Solomon, et al., 1997). The present results indicate that increased defensive responding is not restricted to acoustic stimuli. There may be a generally heightened sensitivity to noxious stimuli in individuals with PTSD, such that any aversive UCS will produce a larger defensive response.

During the acquisition phase, the PTSD group also produced larger HR and corrugator EMG responses to the CS+ versus CS−, compared with the non-PTSD group. Taken together, the HR and EMG findings suggest that the presence of the CS+ produced a more negatively valenced emotional response in individuals with PTSD. However, once the threat of shock was removed (extinction phase), and the CS+ no longer served as an explicit signal for the shock, the PTSD group's HR and EMG responses to CS+ presentations no longer differed from those of the non-PTSD group. This suggests that the increased negative emotional valence produced by the CS+ was moderated by the presence of actual threat in the PTSD group. In contrast, the PTSD group's increased SC responses to CS+ versus CS− trials persisted through the extinction phase and appeared to represent a conditioned emotional response that was resistant to safety information. The mechanism or process responsible for these enduring electrodermal responses may be the same one that causes individuals who develop PTSD to show the characteristically heightened and persisting reactivity to trauma-related cues, even with the knowledge that a recurrence of the traumatic event is unlikely.

The heightened conditionability observed in the present study could represent a pretrauma trait. If so, then it might serve as a vulnerability marker for PTSD and be used to identify individuals at increased risk for developing PTSD upon exposure to a traumatic event. On the other hand, heightened conditionability could be an acquired response disposition resulting from exposure to trauma or the development of PTSD. Shors, Weiss, and Thompson (1992) observed that following exposure to a stressor (i.e., low-intensity inescapable tail shock), rats conditioned more rapidly and acquired larger CRs. Exposure to trauma frequently has been found to be greater and more severe in individuals with PTSD, compared with individuals without the disorder (e.g., Green, Grace, Lindy, Gleser, & Leonard, 1990; Kulka et al., 1990). This increased exposure to traumatic events might cause a heightened sensitivity to aversive stimuli, which could lead to stronger and more lasting CRs. However, the present findings do not support the position that stronger conditionability in PTSD is a direct consequence of exposure to more severe trauma. The PTSD group continued to show significantly larger conditioned SC responses than the non-PTSD group after adjusting for their somewhat higher trauma exposure scores. Even so, it is possible that the present study's use of three different trauma exposure scales to capture the different types of traumatic events experienced by participants might not have provided a sufficiently sensitive index of trauma exposure, which could have minimized group differences. This possibility could be addressed in future research by recruiting within a specific trauma population for a given study, thereby allowing for the use of a single measure of trauma exposure. Alternatively, it may be possible to develop new measures of trauma exposure that would be universally applicable to a wide range of traumatic events.

An important direction for future research will be to design studies than can specifically address alternative explanations, such as pretrauma trait versus acquired response disposition, for the heightened autonomic conditioning shown by individuals with PTSD. For example, conditionability could be prospectively assessed in individuals likely to be exposed to traumatic events, and again following exposure to a traumatic event. Heightened conditionability could then be tested as a predictor for the development of PTSD, for its emergence following exposure to a traumatic event, or for both.

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