

Journal of Abnormal Psychology

Exposure to Violence and Nonassociative Learning Capability Confer Risk for Violent Behavior

Suzanne Estrada, Cassidy Richards, Dylan G. Gee, and Arielle Baskin-Sommers

Online First Publication, June 25, 2020. <http://dx.doi.org/10.1037/abn0000579>

CITATION

Estrada, S., Richards, C., Gee, D. G., & Baskin-Sommers, A. (2020, June 25). Exposure to Violence and Nonassociative Learning Capability Confer Risk for Violent Behavior. *Journal of Abnormal Psychology*. Advance online publication. <http://dx.doi.org/10.1037/abn0000579>

Exposure to Violence and Nonassociative Learning Capability Confer Risk for Violent Behavior

Suzanne Estrada, Cassidy Richards, Dylan G. Gee, and Arielle Baskin-Sommers
Yale University

A substantial body of research demonstrates that experiences of trauma are associated with disruptions in learning processes. Specifically, research shows altered nonassociative and associative learning in individuals who report traumatic experiences. The combination of trauma and altered learning also confers risk for negative health and mental health outcomes. One subtype of trauma that receives less attention in terms of its association with learning processes is exposure to violence (ETV)—witnessing violence, hearing gunfire, and/or being the victim of violence. Preliminary evidence shows that ETV is related to disruptions in nonassociative and associative learning processes, but these studies did not use direct and objective measures of learning. Additionally, research documents a robust relationship between ETV and violent behavior, but there has been no work examining whether it is the combination of elevated levels of ETV and learning patterns that poses a risk for engagement in violent behavior. In the present study, 164 participants completed two auditory basic learning tasks, one measuring nonassociative learning (habituation) and another measuring associative learning acquisition (classical conditioning), while skin conductance was recorded. Results indicate that individuals with higher ETV display a decreased likelihood of physiological habituation, but ETV is unrelated to associative learning acquisition. Further, the combination of higher ETV and nonhabituation predicts a greater number of violent crimes. These findings suggest that, for those with higher ETV, variations in capability for nonassociative learning may confer risk for violent behavior.

General Scientific Summary

Exposure to violence has reached epidemic status in the United States. This exposure places some people at risk for perpetrating violence. Results from the present study indicate that exposure to violence is associated with a failure to habituate to repeatedly presented nonaffective stimuli and that this pattern is a risk marker for elevated engagement in violent crime.

Keywords: habituation, classical conditioning, learning, exposure to violence, violence

Supplemental materials: <http://dx.doi.org/10.1037/abn0000579.supp>

Approximately 70% of youth and 50–60% of adults in the United States report exposure to at least one type of trauma in their lifetime (Finkelhor, Turner, Shattuck, & Hamby, 2013; Substance Abuse & Mental Health Services Administration, 2020). For some individuals, trauma curtails functioning, often resulting in psychosocial, mental health, and physical health problems (see van der Kolk, 2007 for review), and its costs exceed over \$450 billion annually (Gilad & Gutman, 2019). Therefore, it is essential that

research identifies processes related to traumatic experiences that elevate the risk for problematic functioning.

One well-studied process found in the trauma literature is learning. From a cognitive neuroscience perspective, basic learning can be parsed into subcomponents, such as nonassociative and associative learning. Habituation, a nonassociative process, is defined by the decrement in response shown to a stimulus repeatedly presented over time (Rankin et al., 2009). Achievement of habituation often is assessed objectively using physiological measures (e.g., skin conductance to gauge level of arousal, neuroimaging to evaluate level of neural reactivity). Classical conditioning, a process of associative learning, involves repeatedly pairing cues to produce a new learned response (see Lonsdorf et al., 2017 for review). A combination of objective physiological and task performance measures is employed to evaluate successful learning acquisition. Moreover, these two learning processes can be interrelated such that nonassociative learning shapes how associations are formed and/or influences how associative inputs are translated into behavior (Thorwart & Livesey, 2016). Across numerous stud-

 Suzanne Estrada, Cassidy Richards,  Dylan G. Gee, and  Arielle Baskin-Sommers, Department of Psychology, Yale University.

This study was approved by the Human Investigation Committee at Yale University (1408014485). No submitted or published articles report on part or all of this data set. None of the authors have a conflict of interest.

Correspondence concerning this article should be addressed to Suzanne Estrada, Department of Psychology, Yale University, P.O. Box 208205, New Haven, CT 06520. E-mail: suzanne.estrada@yale.edu

ies, trauma, broadly construed, results in slower nonassociative learning and in disruptions to associative learning (see Lissek & van Meurs, 2015 for review).

A meta-analysis comparing over 1,000 adults with and without posttraumatic stress disorder showed slower skin conductance habituation slopes to startling sounds (Pole, 2007). In terms of associative learning, using a broad measure of trauma exposure, Jovanovic and colleagues (2014) reported that young children with higher trauma exposure showed poor discrimination of conditioned stimuli. Similarly, Ayers, White, and Powell (2003) demonstrated that combat veterans had impaired conditioning compared to noncombat veterans. Furthermore, there is evidence that subtypes of trauma differentially impact basic learning (Machlin, Miller, Snyder, McLaughlin, & Sheridan, 2019; McLaughlin, Sheridan, & Lambert, 2014).

There is a long history of parsing subtypes of trauma. The rationale for this approach is particularly clear in the trauma-related learning literature. Atypical associative learning is related to maltreatment (McLaughlin et al., 2016) and experiences of deprivation in children (Sheridan et al., 2018). However, across studies of single types of trauma, there is evidence that traumas characterized by threat, such as physical abuse and domestic violence, are more likely associated with disruptions in learning, particularly in emotion contexts, than those characterized by deprivation (Machlin et al., 2019; McLaughlin et al., 2014). Interestingly, one subtype of trauma characterized as threat-related has received less specific attention in terms of learning processes—that is, exposure to violence (ETV).

ETV encompasses witnessing and/or being the victim of acts such as assaults, shootings, and robberies, as well as hearing gunfire in one's community (DeCou & Lynch, 2017). It typically excludes domestic violence, media violence, and the perpetration of violence. In the United States, approximately 30% of youth report exposure to violence (Finkelhor, Turner, Ormrod, & Hamby, 2010). Furthermore, between 80% and 100% of residents in poor, urban communities report being exposed to violence (Bender & Roberts, 2009; Stein, Jaycox, Kataoka, Rhodes, & Vestal, 2003). Such exposure is associated with adverse consequences, including increased risk for mental health problems such as posttraumatic stress disorder (Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009), persistent academic underachievement (Borofsky, Kellerman, Baucom, Oliver, & Margolin, 2013), and lifelong physical health issues (Ford & Browning, 2014; Wright et al., 2004). Perhaps one of the strongest documented relationships is between ETV and engagement in violent behavior (Baskin & Sommers, 2014; Fowler et al., 2009). However, the association between ETV and learning processes remains understudied.

Findings from available research on ETV and learning are equivocal. There is some evidence that repeatedly witnessing community violence results in less arousal to violence, suggesting that individuals become desensitized to it through habituation (Aiyer, Heinze, Miller, Stoddard, & Zimmerman, 2014; Gaylord-Harden, So, Bai, & Tolan, 2017; Ng-Mak, Salzinger, Feldman, & Stueve, 2004). However, other research indicates that these individuals report increased arousal (Gaylord-Harden, Bai, & Simic, 2017; Gaylord-Harden, Cunningham, & Zelencik, 2011). Critically, these studies employed only a very general construct of learning (e.g., habituation/desensitization vs. nonhabituation/hy-

perarousal), which is then measured indirectly through participant self-reports of arousal. One study on ETV did use a validated associative learning task, but in the context of harm learning, and found that ETV did not impact the ability to correctly learn about agents' harm preferences (Siegel, Estrada, Crockett, & Baskin-Sommers, 2019). Importantly, though, this effect was specific to a harm context and cannot be generalized to other contexts or to more basic learning processes. Therefore, research on the association between ETV and learning processes is needed, especially in light of both the frequency and impact of ETV.

Moreover, despite the discussion of a robust connection between ETV and violence due to a purported "learning effect" (e.g., desensitization, social learning, social modeling; Huesmann & Kirwil, 2007; Ng-Mak, Salzinger, Feldman, & Stueve, 2002), little empirical research directly measures learning processes. Guerra, Huesmann, and Spindler (2003) interpreted the mediating role of fanaticizing and normalizing beliefs about violence but not how these beliefs are learned. Thus, beliefs are used as proxies for social learning. Similarly, Gaylord-Harden and colleagues (2017) found that self-reported hyperarousal, which they interpreted as an indicator of nonhabituation, mediates the association between ETV and aggressive behavior. However, again, an indirect measure of learning was used. Additionally, across the ETV-violence literature, there are inconsistencies in the associations between ETV and learning, as well as between ETV and violence. For instance, as noted above, some studies related ETV to desensitization (i.e., habituation) and others supported the opposite (i.e., nonhabituation).

Importantly, there is evidence that the level of ETV matters for the connection between ETV and violence. Some studies showed that only high levels of ETV are connected to violent behavior (Baskin & Sommers, 2014). Further, though not directly measuring learning processes, theories and empirical studies suggest that the level of arousal or type of learning (habituation vs. nonhabituation) differentially predicts violence outcomes among those exposed to violence (Gaylord-Harden et al., 2017; Gaylord-Harden, Dickson, & Pierre, 2016; Scarpa, Tanaka, & Chiara Haden, 2008). These studies suggest that the connection between ETV and violence is moderated by certain physiological responses. Thus, individual differences in ETV and learning capability may put some more at risk for violence than others. Ultimately, then, the question becomes this: For whom is the association between ETV and violence the strongest?

The present study examined the relationship between ETV and subcomponent learning processes. We administered two auditory learning tasks while skin conductance was collected. The first task measured nonassociative learning (i.e., habituation) by repeating a series of identical tones. The second task measured associative learning (i.e., classical conditioning) by presenting tones previously heard during the nonassociative learning task and novel tones never heard before. The implementation of nonaffective auditory learning tasks made it possible to explore whether ETV was associated with disruptions in learning at the most fundamental level. The use of two tasks allowed for an examination of the specific ways in which multiple, potentially interrelated learning processes were associated with ETV. Additionally, we examined the extent to which ETV and learning processes represented risk markers for engagement in real-world violent behavior. As a follow-up analysis for any ETV-learning findings, we examined

the extent to which specific learning processes moderated the association between ETV and the number of violent crime charges. Finally, for all analyses, we examined the robustness of any relationships by considering additional factors (i.e., demographics, environmental experiences, psychological factors) that also relate to ETV, learning, and/or violence.

Method

Participants

Participants were recruited through posted flyers around New Haven County, Connecticut, a high-crime region of the United States. A prescreen phone interview and in-person assessment materials were used to exclude individuals who were younger than 18 or over 75; who had performed below the fourth-grade level on a measure of reading (Wilkinson, 1993); who scored below 70 on a measure of IQ (Zachary, 1986); who had diagnoses of schizophrenia, bipolar disorder, or psychosis, not otherwise specified (First, Williams, Karg, & Spitzer, 2015); or who had a history of certain medical problems (e.g., auditory impairment, loss of consciousness greater than 30 min, seizures). These exclusions were necessary to ensure participants' comprehension of the materials and ability to perform the tasks. All participants provided written informed consent and earned \$10/hr. The experimental protocol was approved by the Yale University Human Investigation Committee. A total of 164 adults were included in the study (see Table 1 for sample characteristics and zero-order correlations; see online Supplemental Table 1 for additional participant information).

Measures

Exposure to violence. Exposure to violence was measured using a 13-item scale that assesses lifetime exposure to violent events, not including media violence or perpetration of violence (Burnside & Gaylord-Harden, 2019; Selner-O'Hagan, Kindlon, Buka, Raudenbush, & Earls, 1998). Items documented both experienced and observed violence, including "Have you ever been hit, slapped, punched, or beaten up?" and "Have you seen someone else get attacked with a weapon like a knife or bat?" Participants responded to each item based on a dichotomous choice (yes/no). If yes was selected, participants selected the age range of their first experience (under 6 years old, 6–11 years old, 12–17 years old, 18–23 years old, 24–29 years old, or 30 years old or older). A total score was calculated using a sum of all 13 items (ETV score). In this sample, 87.8% experienced at least one exposure to violence in their lifetime, and 44.5% experienced over four (the median) different exposures to violence in their lifetime. The average age range of first exposure was between 12 and 17 years old, with 61.5% reporting their first exposure in this age range. ETV is sometimes decomposed into two subcomponents: experienced (i.e., direct victimization) and observed (i.e., witnessing) violence (Buka, Stichick, Birdthistle, & Earls, 2001). Analysis of the relationships between these subcomponents and learning processes is provided in the online Supplemental Materials.

Violent crime. All participants were asked if they ever committed a crime; if affirmative, participants provided the types of crimes they committed. These crimes were coded as violent (e.g., murder, assault, weapon) or nonviolent (e.g., theft, drug posses-

sion) and separated based on juvenile versus adult crimes. This self-report was confirmed using the State of Connecticut Department of Correction charge database, which documents adult (18 and older) charges. Most self-reported crimes were documented in the official database. However, if participants self-reported committing a crime as an adult (18 or older) that was not listed in the database, we added their self-report to the count given that people can commit a crime and never be charged for it. The total number of violent crimes committed as an adult was summed to create a total violent crime score. Overall, 39.6% of the sample had committed at least one violent crime.

Robustness analyses. We used robustness analyses to statistically isolate the effects of ETV above and beyond the effects of demographics, environmental experiences, and psychological factors. The following variables were considered in supplemental analyses: age, self-reported race (White, non-White), gender (male, female), perceived neighborhood disorder, childhood maltreatment, trait anxiety, Axis I psychopathology, Axis II Cluster B psychopathology, lifetime PTSD, and resting skin conductance levels (see online Supplemental Materials).

Habituation and conditioning task. Tones for the habituation, conditioning, and manipulation checks were produced using Audacity software and presented binaurally through a set of headphones. Data were collected in five phases: rest, habituation, conditioning, and two posttask manipulation checks following the habituation and conditioning phases, respectively (see Figure 1). For all experimental task phases (i.e., not rest), each trial consisted of a fixation cross (500 ms) followed by a blank screen. Tones were presented while participants viewed the blank screen. Each trial was 1,050 ms total, and a variable interval of 10 to 20 s occurred between trials.

Rest. To assess baseline skin conductance reactivity, participants were shown a fixation cross for 4 min. Instructions were to look at the screen without moving.

Habituation. In the habituation phase, each participant was randomly assigned to hear one of three frequency tones (1,000 Hz, 1,500 Hz, or 2,000 Hz) 20 times (1-s duration, 25-ms rise and fall times, 65-dB intensity). Tones were randomly assigned to ensure that skin conductance responses reflected differences in habituation, not differences in reactivity to certain tone frequencies. Participants were instructed that they did not have to respond to the tones.

Posthabituation manipulation check. We assessed reaction time (RT) and accuracy to previously heard tones and novel tones in order to assess behavioral discrimination of the tones presented in the habituation phase. Participants were presented with six tones in pseudorandom order. Three of these tones were identical to the tone presented in the habituation phase, while three were novel (100 Hz, 1-s duration, 25-ms rise and fall times, 65-dB intensity). Participants were instructed to press one button upon hearing the tone they had previously heard and to press a different button upon hearing the novel tone.

Conditioning. After a 3-min rest period, participants completed a conditioning task. In order to parse associative learning to familiar and novel stimuli, a total of three different tones were used as the conditioned stimuli (conditional stimulus; CS). One of these tones was identical to the tone previously used in the habituation phase (either 1,000 Hz, 1,500 Hz, or 2,000 Hz depending on which was randomly chosen for the participant for use in the habituation

Table 1
Sample Characteristics and Zero-Order Correlations Among Key Variables

Variable	<i>n</i>	<i>M</i>	<i>SD</i>	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24		
Demographics																													
1. Age	164	40.24	13.50	1																									
2. Race	164			-.02	1																								
Non-White (0)	91																												
White (1)	73																												
3. Gender	164			-.02	.13	1																							
Male (0)	114																												
Female (1)	50																												
Exposure to violence																													
4. Total	164	4.53	3.50	.13	.01	-.14	1																						
5. Witnessing	164	2.81	2.30	.13	-.08	-.16*	.95*	1																					
6. Victimization	164	1.72	1.48	.10	.13	-.13	.88*	.70*	1																				
Task variables																													
7. Habituation status	164			-.01	-.03	.10	-.18*	-.17*	-.17*	1																			
No (0)	58																												
Yes (1)	106																												
8. Average first trial of habituation (habitua-tors)	106	7.72	5.66	.01	-.01	-.11	-.12	-.15	-.04	1																			
9. Average number of habituation periods (habitua-tors)	106	1.99	1.06	-.06	-.02	.08	-.05	-.02	-.09	-.30*	1																		
10. Average conditioning score (SCR)	164	.59	1.05	-.22*	.44*	.03	-.10	-.13	-.04	-.04	.16	-.18	1																
11. Familiar CS+ difference score (SCR)	164	.75	1.44	-.19*	.41*	.06	-.07	-.08	-.03	-.06	.13	-.16	.95*	1															
12. Novel CS+ difference score (SCR)	164	.43	.88	-.23*	.39*	.04	-.13	-.17*	-.05	-.01	.19	-.18	.85*	.63*	1														
13. Habituation phase change (interbeat interval)	161	-.51	2.71	.22*	-.12	-.04	-.18*	-.21*	-.11	-.10	-.03	.03	-.07	-.03	-.10	1													
14. Average conditioning score (interbeat interval)	159	.26	45.70	.06	.11	.02	-.05	-.09	.03	-.04	-.06	.11	.10	.11	.06	.06	1												
15. Familiar CS+ difference score (interbeat interval)	159	-.56	55.39	.12	.12	.01	.03	-.02	.10	-.12	-.05	.10	.10	.10	.08	-.07	.84*	1											
16. Novel CS+ difference score (interbeat interval)	159	1.09	54.07	-.02	.08	-.01	-.11	-.14	-.05	.02	-.06	.10	.07	.08	-.03	.17*	.83*	.39*	1										
Violent behavior																													
17. Violent crime count	164	.80	1.79	.21*	-.15	-.17*	.21*	.16*	.23*	-.02	-.18	-.17	-.02	-.01	-.04	.08	.03	.02	.04	1									
Covariates																													
18. Resting skin conductance	164	.41	.17	-.09	.29*	-.01	-.02	-.07	.06	-.01	.18	-.19	.23*	.20*	.21*	-.02	.01	-.02	.04	-.01	1								
19. PND	164	32.85	10.23	.02	-.10	-.07	.16*	.15	.14	.02	-.10	.02	-.17*	-.17*	-.14	-.04	-.04	-.04	-.11	.08	-.10	1							
20. CTQ-SF	164	45.97	17.13	.07	.10	.02	.17*	.07	.29*	.14	-.02	-.09	-.09	-.11	-.03	.18*	-.05	-.03	-.06	.06	-.03	.26*	1						
21. Trait anxiety	164	.03	1.00	.01	.18*	.07	.08	.03	.15	.17*	.05	-.21*	.05	.06	-.06	-.02	-.04	.00	.11	.01	.24*	.46*	1						
22. Any Axis I disorder	164			.11	.08	-.09	.14	.12	.15	.07	.00	-.00	-.08	-.07	-.08	-.04	-.03	-.06	.01	.16*	.06	.08	.00	.24*	1				
No (0)	28																												
Yes (1)	136																												
23. Any Axis II Cluster B disorder	164			.18*	-.12	-.10	.32*	.29*	.32*	-.09	-.13	.04	-.15	-.13	-.19*	.15	-.01	-.05	-.04	.13	-.11	.08	.27*	.10	.10	1			
No (0)	106																												
Yes (1)	58																												
24. Lifetime PTSD	164			.00	.24*	.11	.12	.06	.16*	-.03	.08	.09	.04	.04	.03	.12	-.04	-.09	-.02	.02	.06	.04	.30*	.30*	.18*	-.01	1		
No (0)	141																												
Yes (1)	23																												

Note. Correlations including race, sex, habituation status, any Axis I disorder, any Axis II Cluster B disorder, and lifetime PTSD used Spearman's ρ ; all other correlations used Pearson's r . SCR = skin conductance response; CS = conditioned stimulus; PND = perceived neighborhood disorder total score; CTQ-SF = Childhood Trauma Questionnaire—Short Form total score; PTSD = posttraumatic stress disorder.
* $p < .05$.

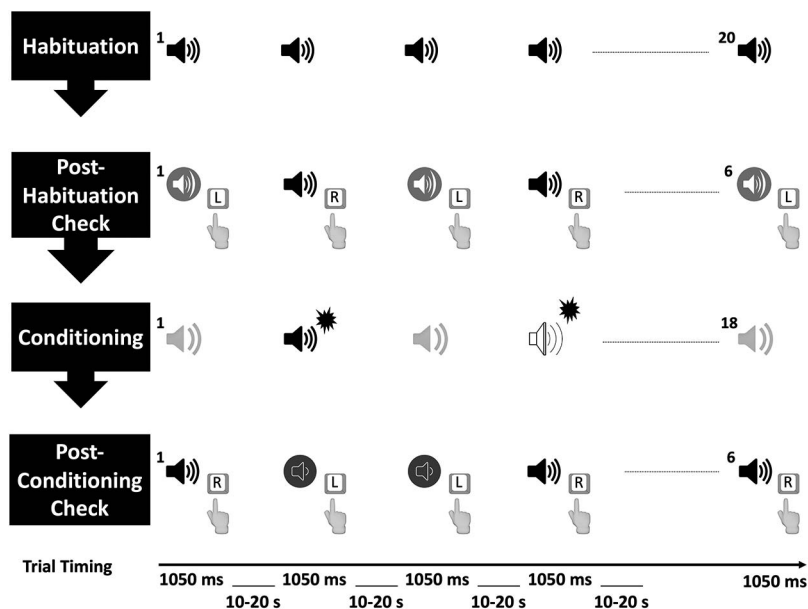


Figure 1. Schematic for habituation and conditioning task. Participants completed a 4-min rest period followed by a habituation task in which 20 identical tones were presented sequentially. Then, participants completed the posthabituation check task in which they pressed one button for previously heard tones and another button for novel tones. Following a 3-min break, participants completed a conditioning task where previously heard and novel tones served as CS⁺ (familiar CS⁺ and novel CS⁺), and another novel tone served as the CS⁻. Finally, participants completed a postconditioning check task in which they pressed one button for the tone heard in the habituation phase and another button for a novel tone. Each trial for all phases was 1,050 ms, and the intertrial interval for all phases was between 10 and 20 s.

phase; 1-s duration; 25-ms rise and fall times; 65-dB intensity), and two of the tones were novel and not used during the habituation phase (either 1,500 Hz and 2,000 Hz, 1,000 Hz and 2,000 Hz, or 1,000 Hz and 1,500 Hz depending on which were not used in the habituation phase; 1-s duration; 25-ms rise and fall times; 65-dB intensity). Each of the three CS tones was presented six times (18 tones total) in pseudorandom order. Following 12 of the CS tones, a startle probe (50-ms rise and fall times, 102-dB intensity) was presented as the unconditioned stimulus (UCS). The startle probe occurred 0 ms after the presentation of the CS and was equally distributed across the two CS types (familiar CS⁺ and novel CS⁺). The familiar CS⁺ and the novel CS⁺ were 100% reinforced (Lonsdorf et al., 2017). For the third CS tone (CS⁻), none of the six presentations were reinforced (i.e., they were never followed by the UCS).

Postconditioning manipulation check. We measured RT and accuracy to the tone used in the habituation phase and to novel tones in order to assess behavioral discrimination of the tone presented in the habituation phase across all phases of the task. Participants were presented with six tones in pseudorandom order. Three of these tones were identical to the tone presented in the habituation phase (and the conditioning phase), while three were novel (3,000 Hz, 1-s duration, 25-ms rise and fall times, 65-dB intensity). Participants were instructed to press one button upon hearing the tone they had heard previously in both the habituation and conditioning phases and to press a different button upon hearing the novel tone.

Psychophysiological recording and analysis. Skin conductance response (SCR) was measured with Ag-AgCl electrodes

from the thenar and hypothenar eminences of the nondominant hand. Biopac's GEL101 isotonic electrode paste was applied to each electrode so that humidity of the skin under the metal electrode did not augment skin conductance recording (Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures, 2012). Data were recorded through Biopac's MP160 and BioNomadix acquisition units. All data were preprocessed using the Psycho-Physiological Modeling suite (PsPM, Version 4.0.2) for MATLAB (available at pspm.sourceforge.net).

Rest. Data were preprocessed using the dynamic causal modeling (DCM) framework for spontaneous skin conductance fluctuations (Bach, Daunizeau, Kuelzow, Friston, & Dolan, 2011). DCM uses a canonical SCR function and an amplitude threshold (0.01 μ S) to estimate the frequency of the fluctuations that reach the specified amplitude threshold during the 4-min rest phase.

Habituation. Data were preprocessed using the general linear model (GLM) framework for evoked SCRs (see Bach, Friston, & Dolan, 2013), which convolved the canonical SCR function with a time derivative, determined the fit of the predicted response to the actual SCR waveform, then reconstructed the estimated SCR and outputted the peak amplitude of that response during the specified time window (1-4 s after stimulus onset) for each trial separately (Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures, 2012). Habituation was operationalized as a binary (yes/no) measure using a criterion threshold of the presence or absence of two consecutive trials that fell one-half

standard deviation below the participant's median SCR across the habituation phase.¹

Conditioning. Data were preprocessed using the DCM framework for evoked SCR. The model's forward approach took into account skin conductance responding on previous trials to estimate the most likely sudomotor nerve activity given the actual skin conductance waveform (Staib, Castegnetti, & Bach, 2015). DCM accurately discriminates CS⁺ from CS⁻ trials in a conditioning task (Staib et al., 2015). To operationalize conditioning, the amplitude of the SCR was averaged across trials within each trial type (familiar CS⁺, novel CS⁺, CS⁻). We calculated two metrics of conditioning: the average conditioning score (i.e., average of the difference between the two CS⁺ types and the CS⁻) and the type-based difference conditioning scores (i.e., SCR to the familiar CS⁺ minus the CS⁻; SCR to the novel CS⁺ minus the CS⁻).

Manipulation checks. Trials were blocked by type (familiar, novel) for each manipulation check phase. Then, we computed measures of average RT and accuracy across trials within each block.

SCR validity check. We measured heart rate variability (i.e., interbeat interval; Mueller, Sperl, & Panitz, 2019) during the habituation and conditioning tasks to assess convergent validity for the SCR measure (Tomarken, 1995). See the online Supplemental Methods for information about how heart rate variability was measured and processed.

Results

Task Effects

Table 1 reports basic task statistics. We conducted a repeated measures GLM with trial number (1 through 20) as a within-subjects factor to examine SCR across all 20 trials of the habituation phase. There was no effect of trial number on SCR, $F(19, 3097) = .54, p = .948, \eta^2 = .00, 95\% \text{ CI for } \eta^2 [.00, .00]$. We conducted a repeated-measures GLM with trial type difference score (familiar CS⁺ - CS⁻, novel CS⁺ - CS⁻) as a within-subjects factor for SCR for the conditioning phase. The effect of trial type was significant for SCR, $F(1, 163) = 13.44, p < .001, \eta^2 = .08, \text{ CI for } \eta^2 [.02, .16]$, such that learning acquisition was stronger to the familiar CS⁺ ($M = .75 \mu\text{s}, SD = 1.44$) compared to the novel CS⁺ ($M = .43 \mu\text{s}, SD = .88$).

We examined performance on the two postphase checks to ensure both successful task manipulation and participant explicit awareness of the familiar tone across phases. Two paired-samples t tests were conducted to compare RT and accuracy to familiar and novel tones in the posthabituation check and postconditioning check phases. In the posthabituation check phase, participants were significantly faster, $t(163) = -4.65, p < .001, 95\% \text{ CI of the difference } [-.23, -.09]$, and more accurate, $t(163) = 2.36, p = .020, 95\% \text{ CI of the difference } [.01, .07]$, for familiar tones compared to novel tones (RT for familiar: $M = 1.57 \text{ s}, SD = .54$; RT for novel: $M = 1.72, SD = .55$; accuracy for familiar: $M = 93.5\%, SD = .19$; accuracy for novel: $M = 89.8\%, SD = .22$). In the postconditioning check phase, participants also were significantly faster, $t(162) = -2.43, p = .016, 95\% \text{ CI of the difference } [-.24, -.02]$, and more accurate, $t(162) = 3.47, p = .001, 95\% \text{ CI of the difference } [.03, .10]$, for familiar tones compared to novel tones (RT for familiar: $M = 1.75 \text{ s}, SD = .57$; RT for novel: $M =$

$1.89, SD = .63$; accuracy for familiar: $M = 95.1\%, SD = .17$; accuracy for novel: $M = 89.0\%, SD = .23$).

What Is the Relationship Between ETV and Learning Processes?

Habituation. A binary logistic regression was conducted to predict habituation (yes/no) based on the level of ETV score (z -scored). The overall model was significant, $\chi^2(1) = 5.28, p = .022$, explained 4.4% of the variance (Nagelkerke R^2) in habituation, and correctly classified 63.4% of the cases. ETV score significantly predicted decreased likelihood of habituation ($B = -.38, SE = .17, OR = .69, p = .023, 95\% \text{ CI for } OR [.49, .95]$; see Figure 2).² These results were robust against all covariates entered separately and simultaneously into the model (see online Supplemental Table 2).

Conditioning. We conducted a simple linear regression with ETV score (z -scored) predicting average conditioning score to examine the effect of ETV score on average learning acquisition. ETV score did not predict average conditioning score, $B = -.11, SE = .08, t(163) = -1.27, p = .206, 95\% \text{ CI for } B [-.27, .06]$. Next, we ran a repeated-measures GLM to examine the effect of ETV score on learning acquisition to familiar and novel tones, using trial type difference score (familiar CS⁺ - CS⁻, novel CS⁺ - CS⁻) as a within-subjects factor and ETV score (z -scored) as a continuous, between-subjects factor. This analysis showed that neither the main effect of ETV score, $F(1, 162) = 1.61, p = .206, \eta^2 = .01, 95\% \text{ CI for } \eta^2 [.00, .06]$, nor the interaction between trial type and ETV score were significant, $F(1, 162) = .06, p = .803, \eta^2 = .00, 95\% \text{ CI for } \eta^2 [.00, .02]$.

We conducted a linear regression with ETV score (z -scored), habituation status (yes/no; mean-centered) and their interaction as predictors of the average conditioning score to understand how completion of the habituation process may interact with ETV to predict learning acquisition. Neither the main effects of ETV score, $B = -.11, SE = .09, t(160) = -1.29, p = .200, 95\% \text{ CI for } B [-.28, .06]$, habituation status, $B = -.14, SE = .18, t(160) = -.81, p = .417, 95\% \text{ CI for } B [-.49, .21]$, nor their interaction were significant, $B = -.04, SE = .17, t(160) = -.25, p = .807, 95\% \text{ CI for } B [-.39, .30]$. A repeated-measures GLM with trial type difference score (familiar CS⁺ - CS⁻, novel CS⁺ - CS⁻) as a within-subjects factor, ETV score (z -scored) as a continuous, between-subjects factor, and habituation status (yes/no; mean-centered) as a categorical, between-subjects factor also revealed that none of the main effects, ETV score: $F(1, 160) =$

¹ While previous research operationalized a binary habituation measure as two consecutive responses that fall below a predetermined threshold, the determination of that threshold varies across studies (e.g., .001, .002, .005; e.g., Clark, Siddle, & Bond, 1992; Herpertz et al., 2001; Lykken, Iacono, Haroian, McGue, & Bouchard, 1988), and there is no gold-standard threshold. Given these issues, the current study used a data-driven approach to operationalize a binary habituation measure using each individual's data to determine an adequate level of deviation from the median SCR. However, examination of a habituation criterion threshold of .001 reveals that roughly the same number of participants habituate compared to using our data-driven threshold, indicating that our threshold approximates other thresholds used in the extant literature.

² The validity check corroborated these results. ETV predicted significantly lower interbeat interval (i.e., faster heart beats) across the 20 trials (see online Supplemental Results).

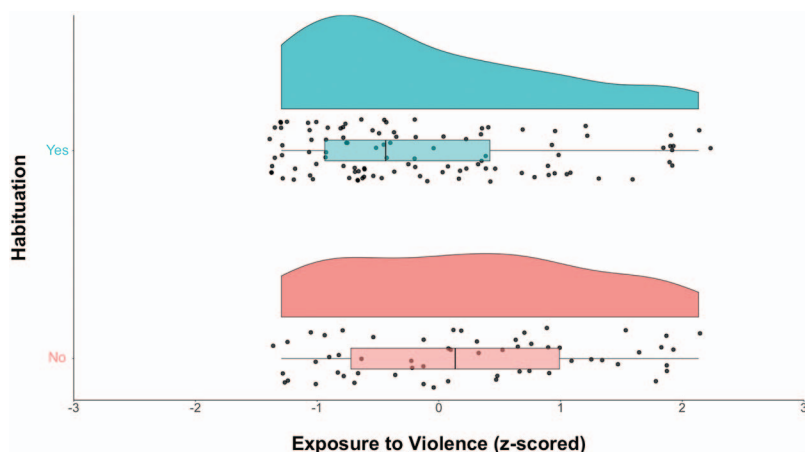


Figure 2. ETV predicts decreased likelihood of habituation. Density plots show distribution of ETV scores for those who did (blue [light gray]) and those who did not (red [dark gray]) habituate. Larger peaks indicate higher concentration of participants at that level of ETV. Scatter plots demonstrate the distribution of participants who did and did not habituate by level of ETV. Box plots reflect minimum, first quartile, median, third quartile, and maximum values. Density, scatter, and box plots indicate that ETV is associated with decreased habituation (red). See the online article for the color version of this figure.

1.66, $p = .200$, $\eta^2 = .01$, 95% CI for η^2 [.00, .06]; habituation status: $F(1, 160) = .66$, $p = .417$, $\eta^2 = .00$, 95% CI for η^2 [.00, .05], or higher-order interactions (Trial Type \times ETV Score, Trial Type \times Habituation Status, and Trial Type \times ETV Score \times Habituation Status) were significant (all $ps > .141$; see Figure 3).³ Finally, for all conditioning analyses, no suppression effects (i.e., ETV did not predict conditioning metrics) were found when covariates were entered into the model separately and simultaneously (see online Supplemental Tables 3–6).⁴

Do ETV and Habituation Capability Confer a Risk Marker for Violent Behavior?

We conducted a negative binomial regression with ETV score (z-scored), habituation status (yes/no; mean-centered), and the interaction between them as predictors of violent crime count to determine whether habituation moderated the relationship between ETV and violence. The omnibus model test was significant, likelihood ratio $\chi^2(3) = 21.79$, $p < .001$. The main effects of both ETV score ($B = .32$, $SE = .15$, $p = .032$, 95% Wald CI for B [.03, .62]) and habituation status ($B = -.78$, $SE = .32$, $p = .015$, 95% Wald CI for B [-1.41, -.15]) were significant. There also was a significant interaction between ETV score and habituation status ($B = .55$, $SE = .28$, $p = .048$, 95% Wald CI for B [.004, 1.09]) such that the relationship between ETV and violence was stronger for those who did not habituate ($B = .87$, $SE = .23$, $p < .001$, 95% Wald CI for B [.41, 1.33]) compared to those who did ($B = .32$, $SE = .15$, $p = .032$, 95% Wald CI for B [.03, .62]; see Figure 4). These results were robust against covariates entered separately and simultaneously into the model (see online Supplemental Table 7).

Discussion

Exposure to violence is one subtype of trauma that adversely affects a substantial proportion of U.S. residents. Previous theory and research suggest that learning may be disrupted in individuals

exposed to violence and that learning capability combined with ETV may be important for determining who will be most vulnerable to engaging in violence themselves following violence exposure. Results of the current study indicate that those exposed to more violence displayed disruptions in some, but not all, basic learning processes.

ETV was associated with decreased likelihood of habituation. Consistent with research in other forms of threat-related trauma, such as child maltreatment and witnessing domestic violence (Dube et al., 2003; Gerardi, Keane, Cahoon, & Klauminzer, 1994; Kendall-Tackett, 2000; van der Kolk, 1989), it appears that individuals exposed to a greater number of violent events maintain levels of arousal to repeatedly presented stimuli, rather than become desensitized to them. Though previous work demonstrated the tendency of individuals with higher ETV toward chronic hyperarousal in affective contexts, the present study found that chronic hyperarousal to repeatedly presented stimuli also occurs in nonaffective contexts, suggesting that fundamental, basic learning processes may be altered as a result of ETV.

By contrast, ETV was not related to associative learning acquisition, regardless of whether learned information was familiar or novel. This finding adds to a small body of literature that suggests that ETV is unrelated to disruptions in associative learning, broadly (Siegel et al., 2019). However, this pattern diverges from work indicating that other violent life stressors (e.g., childhood maltreatment, combat) impact associative learning acquisition

³ All conditioning analyses were corroborated by the validity check. Neither ETV score nor the ETV Score \times Habituation Status interaction was associated with average conditioning score or difference scores for familiar and novel tones when learning was indexed using the interbeat interval (see online Supplemental Results).

⁴ We attempted several transformations to reduce skewness and kurtosis of the conditioning scores (log transformation, reciprocal, cube root). Results for all conditioning analyses remained nonsignificant regardless of the transformation performed.

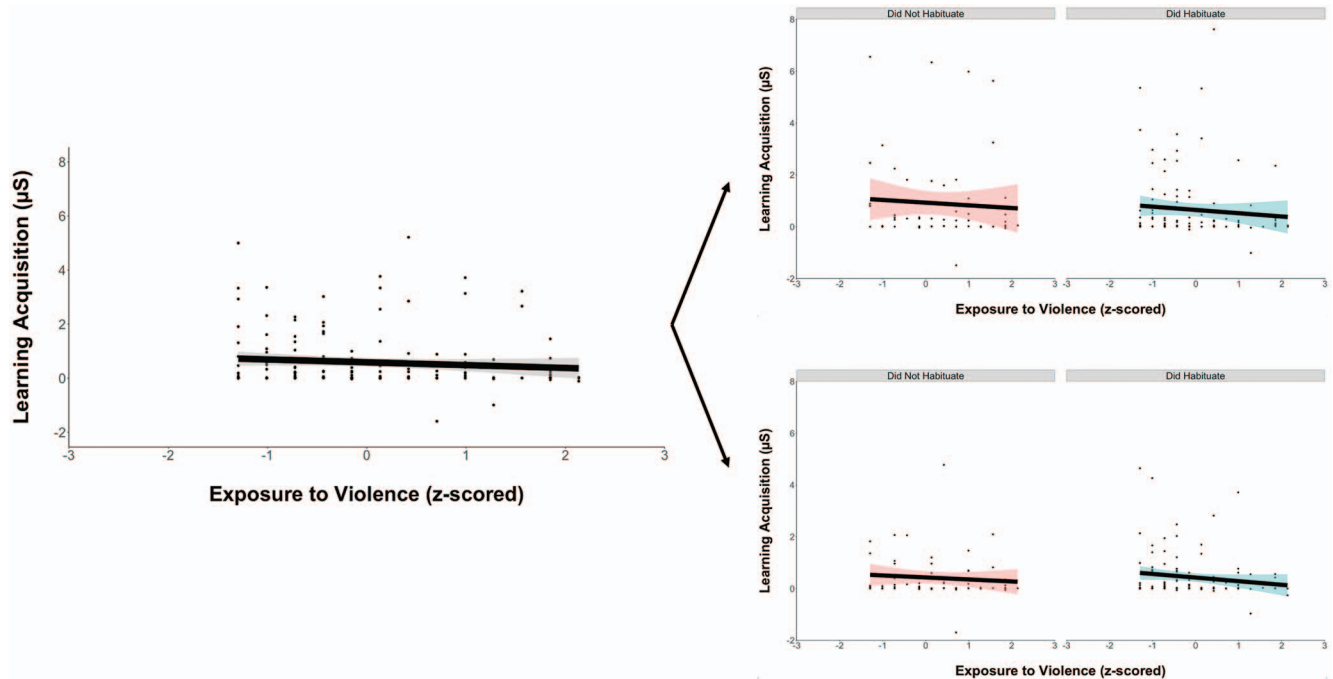


Figure 3. ETV did not impact associative learning acquisition. ETV was not associated with overall learning acquisition (left panel). Scatter plot shows distribution of conditioning acquisition scores by level of ETV. ETV was not associated with learning to familiar (top right panel) and novel tones (bottom right panel), regardless of habituation status. Left panel (red [dark gray]) represents those who did not habituate; right panel (blue [light gray]) represents those who did habituate. Error bands represent 95% confidence intervals. See the online article for the color version of this figure.

(Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007; Harms, Shannon Bowen, Hanson, & Pollak, 2018; McLaughlin et al., 2016; Orr et al., 2000; Peri, Ben-Shakhar, Orr, & Shalev, 2000), suggesting that distinct subtypes of life stressors may alter associative learning mechanisms in different ways. While ETV commonly co-occurs with other forms of life stress (i.e., polyvictimization), previous research often does not separate out the impact of these experiences on specific learning processes (Jovanovic et al., 2014). Understanding the ways in which ETV and other forms of life stress, particularly violent life stress, result in similar and distinct expressions will allow for greater accuracy and specificity in identifying and targeting the processes that contribute to the adverse consequences of ETV.

An especially serious consequence of ETV is engagement in violent behavior. It is clear that ETV is strongly associated with violence (see main effect of ETV score predicting violent crime count and Baskin & Sommers, 2014). However, for those with higher levels of ETV, learning capability also appears to influence the frequency of violence: Those with higher levels of ETV who *did not* habituate had the greatest number of violent crime charges. Individuals who display chronic hyperarousal to repeated stimuli may not notice when stimuli are no longer novel and respond appropriately (i.e., habituate; Kimble et al., 2014). In the context of ETV, maintained physiological arousal may result in increased processing of threat-related information (Shields, Larson, Swartz, & Smith, 2011; van der Kolk, 1989). This difficulty in downregulating arousal may drain cognitive and emotional capacities such

that individuals struggle to consider non-stress-related information (Aupperle, Melrose, Stein, & Paulus, 2012; Weems, Saltzman, Reiss, & Carrion, 2003). For example, maintained arousal is related to decreased processing of inhibitory inputs, such as pain (Garfinkel & Critchley, 2016). Thus, increased arousal combined with inattention to inhibitory cues may produce a feedback loop that reinforces both over detection of threatening information, like ETV, and the failure to mitigate the consequences of that information. As a result of this loop, there is an increase in violence perpetration (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988). Results from the present study suggest that the combination of ETV and learning capability is a more nuanced risk marker for violence than ETV alone.

A few limitations should be noted. First, our measure of ETV did not document the type of exposure, limiting our ability to disentangle whether certain exposures were better accounted for by other subtypes of trauma. However, in the present study, the correlation between ETV and CTQ-SF was small, suggesting different experiences were tapped, and analyses controlling for CTQ-SF supported robust associations between ETV and learning. Second, our study did not directly address the temporal order between ETV and learning and whether nonassociative and associative learning prospectively predicted engagement in violence. We cannot establish if basic learning processes promote opportunities for violence exposure and mechanistically impact the relationship between ETV and violent behavior. Third, we did not address whether learning from nonaffective stimuli (e.g., tones)

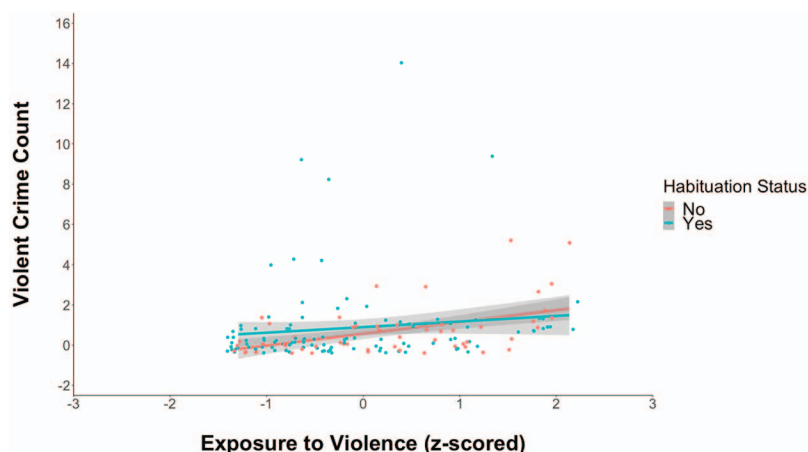


Figure 4. ETV interacts with habituation status to impact violence. For those who did not habituate (red [dark gray]), the relationship between ETV and violence is stronger than for those who did habituate (blue [light gray]). Scatter plot represents the relationship between ETV and violent crime count, and the colors of the dots designate habituation status (did not habituate in red [dark gray], did habituate in blue [light gray]). Error bands represent 95% confidence intervals. See the online article for the color version of this figure.

differs from affective stimuli (e.g., faces, threat, harm). It is unknown whether learning is disrupted across situations or if there are situation-specific disruptions in learning depending on context. Fourth, we only examined associative learning acquisition; thus, we cannot rule out the effect of ETV on other aspects of associative learning (e.g., extinction, reversal learning; Byrd, Loeber, & Pardini, 2014; Guthrie & Bryant, 2006). While two studies have shown no relationship between ETV and associative learning (the present study and Siegel et al., 2019), continuing to examine specific learning processes systematically can uncover the impact of ETV on processes fundamental to the human experience.

The negative effect of ETV on individuals and communities is undeniable. The results of the present study suggest that ETV produces specific disruptions in learning and that differences in nonassociative learning capability contribute to propensities toward engagement in violent behavior. A focus on basic processes might be used to improve societal well-being and, importantly, the well-being of those living in environments with high rates of violence. Ultimately, progress in understanding the connection between ETV and learning favors the advancement of targeted policies and interventions that can alleviate at least some of the burden associated with living under such extreme and harsh conditions.

References

- Aiyer, S. M., Heinze, J. E., Miller, A. L., Stoddard, S. A., & Zimmerman, M. A. (2014). Exposure to violence predicting cortisol response during adolescence and early adulthood: Understanding moderating factors. *Journal of Youth and Adolescence*, *43*, 1066–1079. <http://dx.doi.org/10.1007/s10964-014-0097-8>
- Allen, J. (2007). Photoplethysmography and its application in clinical physiological measurement. *Physiological Measurement*, *28*(3), R1–R39. <http://dx.doi.org/10.1088/0967-3334/28/3/R01>
- Armstrong, K. A., & Khawaja, N. G. (2002). Gender differences in anxiety: An investigation of the symptoms, cognitions, and sensitivity towards anxiety in a nonclinical population. *Behavioural and Cognitive Psychotherapy*, *30*, 227–231. <http://dx.doi.org/10.1017/S1352465802002114>
- Aupperle, R. L., Melrose, A. J., Stein, M. B., & Paulus, M. P. (2012). Executive function and PTSD: Disengaging from trauma. *Neuropharmacology*, *62*, 686–694. <http://dx.doi.org/10.1016/j.neuropharm.2011.02.008>
- Ayers, E. D., White, J., & Powell, D. A. (2003). Pavlovian eyeblink conditioning in combat veterans with and without post-traumatic stress disorder. *Integrative Physiological & Behavioral Science*, *38*, 230–247. <http://dx.doi.org/10.1007/BF02688856>
- Bach, D. R., Daunizeau, J., Kuelzow, N., Friston, K. J., & Dolan, R. J. (2011). Dynamic causal modeling of spontaneous fluctuations in skin conductance. *Psychophysiology*, *48*, 252–257. <http://dx.doi.org/10.1111/j.1469-8986.2010.01052.x>
- Bach, D. R., Friston, K. J., & Dolan, R. J. (2013). An improved algorithm for model-based analysis of evoked skin conductance responses. *Biological Psychology*, *94*, 490–497. <http://dx.doi.org/10.1016/j.biopsycho.2013.09.010>
- Baskin, D. R., & Sommers, I. B. (2014). Exposure to community violence and trajectories of violent offending. *Youth Violence and Juvenile Justice*, *12*, 367–385. <http://dx.doi.org/10.1177/1541204013506920>
- Bender, J. A., & Roberts, M. C. (2009). Exposure to violence, perceived peer relationships, and corresponding psychological sequelae. *Journal of Child and Family Studies*, *18*, 350–355. <http://dx.doi.org/10.1007/s10826-008-9237-7>
- Bennett, S., Farrington, D. P., & Huesmann, L. R. (2005). Explaining gender differences in crime and violence: The importance of social cognitive skills. *Aggression and Violent Behavior*, *10*, 263–288. <http://dx.doi.org/10.1016/j.avb.2004.07.001>
- Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., . . . Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child Abuse & Neglect*, *27*, 169–190. [http://dx.doi.org/10.1016/S0145-2134\(02\)00541-0](http://dx.doi.org/10.1016/S0145-2134(02)00541-0)
- Blechert, J., Michael, T., Vriends, N., Margraf, J., & Wilhelm, F. H. (2007). Fear conditioning in posttraumatic stress disorder: Evidence for delayed extinction of autonomic, experiential, and behavioural responses. *Behaviour Research and Therapy*, *45*, 2019–2033. <http://dx.doi.org/10.1016/j.brat.2007.02.012>

- Borofsky, L. A., Kellerman, I., Baucom, B., Oliver, P. H., & Margolin, G. (2013). Community violence exposure and adolescents' school engagement and academic achievement over time. *Psychology of Violence, 3*, 381–395. <http://dx.doi.org/10.1037/a0034121>
- Buka, S. L., Stichick, T. L., Birdthistle, I., & Earls, F. J. (2001). Youth exposure to violence: Prevalence, risks, and consequences. *American Journal of Orthopsychiatry, 71*, 298–310. <http://dx.doi.org/10.1037/0002-9432.71.3.298>
- Burnside, A. N., & Gaylord-Harden, N. K. (2019). Hopelessness and delinquent behavior as predictors of community violence exposure in ethnic minority male adolescent offenders. *Journal of Abnormal Child Psychology, 47*, 801–810. <http://dx.doi.org/10.1007/s10802-018-0484-9>
- Byrd, A. L., Loeber, R., & Pardini, D. A. (2014). Antisocial behavior, psychopathic features and abnormalities in reward and punishment processing in youth. *Clinical Child and Family Psychology Review, 17*, 125–156. <http://dx.doi.org/10.1007/s10567-013-0159-6>
- Chemtob, C., Roitblat, H., Hamada, R. S., Carlson, J. G., & Twentyman, C. T. (1988). A cognitive action theory of post-traumatic stress disorder. *Journal of Anxiety Disorders, 2*, 253–275. [http://dx.doi.org/10.1016/0887-6185\(88\)90006-0](http://dx.doi.org/10.1016/0887-6185(88)90006-0)
- Clark, B. M., Siddle, D. A., & Bond, N. W. (1992). Effects of social anxiety and facial expression on habituation of the electrodermal orienting response. *Biological Psychology, 33*(2–3), 211–223. [http://dx.doi.org/10.1016/0301-0511\(92\)90033-Q](http://dx.doi.org/10.1016/0301-0511(92)90033-Q)
- Crouch, J. L., Hanson, R. F., Saunders, B., Kilpatrick, D. G., & Resnick, H. (2000). Income, race/ethnicity, and exposure to violence in youth: Results from the National Survey of Adolescents. *Journal of Community Psychology, 28*, 625–641. [http://dx.doi.org/10.1002/1520-6629\(200011\)28:6<625::AID-JCOP6>3.0.CO;2-R](http://dx.doi.org/10.1002/1520-6629(200011)28:6<625::AID-JCOP6>3.0.CO;2-R)
- DeCou, C. R., & Lynch, S. M. (2017). Assessing adult exposure to community violence: A review of definitions and measures. *Trauma, Violence, & Abuse, 18*, 51–61. <http://dx.doi.org/10.1177/1524838015590590>
- Dube, S. R., Felitti, V. J., Dong, M., Chapman, D. P., Giles, W. H., & Anda, R. F. (2003). Childhood abuse, neglect, and household dysfunction and the risk of illicit drug use: The adverse childhood experiences study. *Pediatrics, 111*, 564–572. <http://dx.doi.org/10.1542/peds.111.3.564>
- Duits, P., Cath, D. C., Lissek, S., Hox, J. J., Hamm, A. O., Engelhard, I. M., . . . Baas, J. M. (2015). Updated meta-analysis of classical fear conditioning in the anxiety disorders. *Depression and Anxiety, 32*, 239–253. <http://dx.doi.org/10.1002/da.22353>
- Eitle, D., & Turner, R. J. (2002). Exposure to community violence and young adult crime: The effects of witnessing violence, traumatic victimization, and other stressful life events. *Journal of Research in Crime and Delinquency, 39*, 214–237. <http://dx.doi.org/10.1177/002242780203900204>
- Finkelhor, D., Ormrod, R. K., & Turner, H. A. (2009). Lifetime assessment of poly-victimization in a national sample of children and youth. *Child Abuse & Neglect, 33*, 403–411. <http://dx.doi.org/10.1016/j.chiabu.2008.09.012>
- Finkelhor, D., Turner, H., Ormrod, R., & Hamby, S. L. (2010). Trends in childhood violence and abuse exposure: Evidence from 2 national surveys. *Archives of Pediatrics & Adolescent Medicine, 164*, 238–242. <http://dx.doi.org/10.1001/archpediatrics.2009.283>
- Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2013). Violence, crime, and abuse exposure in a national sample of children and youth: An update. *Journal of the American Medical Association Pediatrics, 167*, 614–621. <http://dx.doi.org/10.1001/jamapediatrics.2013.42>
- First, M. B., Williams, J., Karg, R. S., & Spitzer, R. L. (2015). *Structured Clinical Interview for DSM-5—Research Version*. Arlington, VA: American Psychiatric Association.
- Ford, J. L., & Browning, C. R. (2014). Effects of exposure to violence with a weapon during adolescence on adult hypertension. *Annals of Epidemiology, 24*, 193–198. <http://dx.doi.org/10.1016/j.annepidem.2013.12.004>
- Fowler, P. J., Tompsett, C. J., Braciszewski, J. M., Jacques-Tiura, A. J., & Baltes, B. B. (2009). Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. *Development and Psychopathology, 21*, 227–259. <http://dx.doi.org/10.1017/S0954579409000145>
- Garfinkel, S. N., & Critchley, H. D. (2016). Threat and the body: How the heart supports fear processing. *Trends in Cognitive Sciences, 20*, 34–46. <http://dx.doi.org/10.1016/j.tics.2015.10.005>
- Gaylord-Harden, N. K., Bai, G. J., & Simic, D. (2017). Examining a dual-process model of desensitization and hypersensitization to community violence in African American male adolescents. *Journal of Traumatic Stress, 30*, 463–471. <http://dx.doi.org/10.1002/jts.22220>
- Gaylord-Harden, N. K., Cunningham, J. A., & Zelencik, B. (2011). Effects of exposure to community violence on internalizing symptoms: Does desensitization to violence occur in African American youth? *Journal of Abnormal Child Psychology, 39*, 711–719. <http://dx.doi.org/10.1007/s10802-011-9510-x>
- Gaylord-Harden, N. K., Dickson, D., & Pierre, C. (2016). Profiles of community violence exposure among African American youth: An examination of desensitization to violence using latent class analysis. *Journal of Interpersonal Violence, 31*, 2077–2101. <http://dx.doi.org/10.1177/0886260515572474>
- Gaylord-Harden, N. K., So, S., Bai, G. J., & Tolan, P. H. (2017). Examining the effects of emotional and cognitive desensitization to community violence exposure in male adolescents of color. *American Journal of Orthopsychiatry, 87*, 463–473. <http://dx.doi.org/10.1037/ort0000241>
- Gerardi, R. J., Keane, T. M., Cahoon, B. J., & Klauminzer, G. W. (1994). An in vivo assessment of physiological arousal in posttraumatic stress disorder. *Journal of Abnormal Psychology, 103*, 825–827. <http://dx.doi.org/10.1037/0021-843X.103.4.825>
- Gilad, M., & Gutman, A. (2019). *The tragedy of wasted funds and broken dreams: An economic analysis of childhood exposure to crime and violence* (U of Penn, Inst for Law & Econ Research Paper No. 19–37). <http://dx.doi.org/10.2139/ssrn.3458626>
- Guerra, N. G., Huesmann, L. R., & Spindler, A. (2003). Community violence exposure, social cognition, and aggression among urban elementary school children. *Child Development, 74*, 1561–1576. <http://dx.doi.org/10.1111/1467-8624.00623>
- Guthrie, R. M., & Bryant, R. A. (2006). Extinction learning before trauma and subsequent posttraumatic stress. *Psychosomatic Medicine, 68*, 307–311. <http://dx.doi.org/10.1097/01.psy.0000208629.67653.cc>
- Hanson, R. F., Borntager, C., Self-Brown, S., Kilpatrick, D. G., Saunders, B. E., Resnick, H. S., & Amstadter, A. (2008). Relations among gender, violence exposure, and mental health: The national survey of adolescents. *American Journal of Orthopsychiatry, 78*, 313–321. <http://dx.doi.org/10.1037/a0014056>
- Harms, M. B., Shannon Bowen, K. E., Hanson, J. L., & Pollak, S. D. (2018). Instrumental learning and cognitive flexibility processes are impaired in children exposed to early life stress. *Developmental Science, 21*(4), e12596. <http://dx.doi.org/10.1111/desc.12596>
- Herpertz, S. C., Wenning, B., Mueller, B., Qunaibi, M., Sass, H., & Herpertz-Dahlmann, B. (2001). Psychophysiological responses in ADHD boys with and without conduct disorder: Implications for adult antisocial behavior. *Journal of the American Academy of Child & Adolescent Psychiatry, 40*, 1222–1230. <http://dx.doi.org/10.1097/00004583-200110000-00017>
- Hill, H. M., & Jones, L. P. (1997). Children's and parents' perceptions of children's exposure to violence in urban neighborhoods. *Journal of the National Medical Association, 89*, 270–276.
- Huesmann, L. R., & Kirwil, L. (2007). Why observing violence increases the risk of violent behavior in the observer. In D. J. Flannery, A. T. Vazsonyi, & I. D. Waldman (Eds.), *The Cambridge handbook of violent*

- behavior and aggression* (pp. 545–570). Cambridge, England: Cambridge University Press. <http://dx.doi.org/10.1017/CBO9780511816840.029>
- Hwang, M. J., Zsido, R. G., Song, H., Pace-Schott, E. F., Miller, K. K., Lebron-Milad, K., . . . Milad, M. R. (2015). Contribution of estradiol levels and hormonal contraceptives to sex differences within the fear network during fear conditioning and extinction. *BMC Psychiatry, 15*, 295. <http://dx.doi.org/10.1186/s12888-015-0673-9>
- Inslicht, S. S., Metzler, T. J., Garcia, N. M., Pineles, S. L., Milad, M. R., Orr, S. P., . . . Neylan, T. C. (2013). Sex differences in fear conditioning in posttraumatic stress disorder. *Journal of Psychiatric Research, 47*, 64–71. <http://dx.doi.org/10.1016/j.jpsychires.2012.08.027>
- Jovanovic, T., Nylocks, K. M., Gamwell, K. L., Smith, A., Davis, T. A., Norrholm, S. D., & Bradley, B. (2014). Development of fear acquisition and extinction in children: Effects of age and anxiety. *Neurobiology of Learning and Memory, 113*, 135–142. <http://dx.doi.org/10.1016/j.nlm.2013.10.016>
- Kendall-Tackett, K. A. (2000). Physiological correlates of childhood abuse: Chronic hyperarousal in PTSD, depression, and irritable bowel syndrome. *Child Abuse & Neglect, 24*, 799–810. [http://dx.doi.org/10.1016/S0145-2134\(00\)00136-8](http://dx.doi.org/10.1016/S0145-2134(00)00136-8)
- Kimble, M., Boxwala, M., Bean, W., Maletsky, K., Halper, J., Spollen, K., & Fleming, K. (2014). The impact of hypervigilance: Evidence for a forward feedback loop. *Journal of Anxiety Disorders, 28*, 241–245. <http://dx.doi.org/10.1016/j.janxdis.2013.12.006>
- Lissek, S., & van Meurs, B. (2015). Learning models of PTSD: Theoretical accounts and psychobiological evidence. *International Journal of Psychophysiology, 98*(3), 594–605. <http://dx.doi.org/10.1016/j.ijpsycho.2014.11.006>
- Lonsdorf, T. B., Menz, M. M., Andreatta, M., Fullana, M. A., Golkar, A., Haaker, J., . . . Merz, C. J. (2017). Don't fear 'fear conditioning': Methodological considerations for the design and analysis of studies on human fear acquisition, extinction, and return of fear. *Neuroscience and Biobehavioral Reviews, 77*, 247–285. <http://dx.doi.org/10.1016/j.neubiorev.2017.02.026>
- Lykken, D. T., Iacono, W. G., Haroian, K., McGue, M., & Bouchard Jr., T. J. (1988). Habituation of the skin conductance response to strong stimuli: A twin study. *Psychophysiology, 25*, 4–15. <http://dx.doi.org/10.1111/j.1469-8986.1988.tb00949.x>
- Machlin, L., Miller, A. B., Snyder, J., McLaughlin, K. A., & Sheridan, M. A. (2019). Differential associations of deprivation and threat with cognitive control and fear conditioning in early childhood. *Frontiers in Behavioral Neuroscience, 13*, 80. <http://dx.doi.org/10.3389/fnbeh.2019.00080>
- McLaughlin, K. A., Sheridan, M. A., Gold, A. L., Duys, A., Lambert, H. K., Peverill, M., . . . Pine, D. S. (2016). Maltreatment exposure, brain structure, and fear conditioning in children and adolescents. *Neuropsychopharmacology, 41*, 1956–1964. <http://dx.doi.org/10.1038/npp.2015.365>
- McLaughlin, K. A., Sheridan, M. A., & Lambert, H. K. (2014). Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. *Neuroscience and Biobehavioral Reviews, 47*, 578–591. <http://dx.doi.org/10.1016/j.neubiorev.2014.10.012>
- Mueller, E. M., Sperl, M. F. J., & Panitz, C. (2019). Aversive imagery causes de novo fear conditioning. *Psychological Science, 30*, 1001–1015. <http://dx.doi.org/10.1177/0956797619842261>
- Ng-Mak, D. S., Salzinger, S., Feldman, R., & Stueve, A. (2002). Normalization of violence among inner-city youth: A formulation for research. *American Journal of Orthopsychiatry, 72*, 92–101. <http://dx.doi.org/10.1037/0002-9432.72.1.92>
- Ng-Mak, D. S., Salzinger, S., Feldman, R. S., & Stueve, C. A. (2004). Pathologic adaptation to community violence among inner-city youth. *American Journal of Orthopsychiatry, 74*, 196–208. <http://dx.doi.org/10.1037/0002-9432.74.2.196>
- Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Peri, T., & Pitman, R. K. (2000). De novo conditioning in trauma-exposed individuals with and without posttraumatic stress disorder. *Journal of Abnormal Psychology, 109*, 290–298. <http://dx.doi.org/10.1037/0021-843X.109.2.290>
- Peri, T., Ben-Shakhar, G., Orr, S. P., & Shalev, A. Y. (2000). Psychophysiological assessment of aversive conditioning in posttraumatic stress disorder. *Biological Psychiatry, 47*, 512–519. [http://dx.doi.org/10.1016/S0006-3223\(99\)00144-4](http://dx.doi.org/10.1016/S0006-3223(99)00144-4)
- Pole, N. (2007). The psychophysiology of posttraumatic stress disorder: A meta-analysis. *Psychological Bulletin, 133*, 725–746. <http://dx.doi.org/10.1037/0033-2909.133.5.725>
- Rankin, C. H., Abrams, T., Barry, R. J., Bhatnagar, S., Clayton, D. F., Colombo, J., . . . Thompson, R. F. (2009). Habituation revisited: An updated and revised description of the behavioral characteristics of habituation. *Neurobiology of Learning and Memory, 92*, 135–138. <http://dx.doi.org/10.1016/j.nlm.2008.09.012>
- Ross, C., & Mirowsky, J. (1999). Disorder and decay: The concept and measurement of perceived neighborhood disorder. *Urban Affairs Review, 34*, 412–432. <http://dx.doi.org/10.1177/107808749903400304>
- Sayers, B. M. (1971). The analysis of cardiac interbeat interval sequences and the effects of mental work load. *Proceedings of the Royal Society of Medicine, 64*, 707–710. <http://dx.doi.org/10.1177/003591577106400702>
- Scarpa, A., Tanaka, A., & Chiara Haden, S. (2008). Biosocial bases of reactive and proactive aggression: The roles of community violence exposure and heart rate. *Journal of Community Psychology, 36*, 969–988. <http://dx.doi.org/10.1002/jcop.20276>
- Selner-O'Hagan, M. B., Kindlon, D. J., Buka, S. L., Raudenbush, S. W., & Earls, F. J. (1998). Assessing exposure to violence in urban youth. *Journal of Child Psychology and Psychiatry, 39*, 215–224. <http://dx.doi.org/10.1111/1469-7610.00315>
- Sheridan, M. A., McLaughlin, K. A., Winter, W., Fox, N., Zeanah, C., & Nelson, C. A. (2018). Early deprivation disruption of associative learning is a developmental pathway to depression and social problems. *Nature Communications, 9*, 2216. <http://dx.doi.org/10.1038/s41467-018-04381-8>
- Shields, M. R., Larson, C. L., Swartz, A. M., & Smith, J. C. (2011). Visual threat detection during moderate- and high-intensity exercise. *Emotion, 11*, 572–581. <http://dx.doi.org/10.1037/a0021251>
- Shields, N., Nadasen, K., & Pierce, L. (2009). A comparison of the effects of witnessing community violence and direct victimization among children in Cape Town, South Africa. *Journal of Interpersonal Violence, 24*, 1192–1208. <http://dx.doi.org/10.1177/0886260508322184>
- Siegel, J. Z., Estrada, S., Crockett, M. J., & Baskin-Sommers, A. (2019). Exposure to violence affects the development of moral impressions and trust behavior in incarcerated males. *Nature Communications, 10*, 1942. <http://dx.doi.org/10.1038/s41467-019-09962-9>
- Society for Psychophysiological Research Ad Hoc Committee on Electrodermal Measures. (2012). Publication recommendations for electrodermal measurements. *Psychophysiology, 49*, 1017–1034. <http://dx.doi.org/10.1111/j.1469-8986.2012.01384.x>
- Spielberger, C. D. (1983). *Manual for the State-Trait Inventory STAI (Form Y)*. Palo Alto, CA: Mind Garden.
- Staib, M., Castegnetti, G., & Bach, D. R. (2015). Optimising a model-based approach to inferring fear learning from skin conductance responses. *Journal of Neuroscience Methods, 255*, 131–138. <http://dx.doi.org/10.1016/j.jneumeth.2015.08.009>
- Stein, B. D., Jaycox, L. H., Kataoka, S., Rhodes, H. J., & Vestal, K. D. (2003). Prevalence of child and adolescent exposure to community violence. *Clinical Child and Family Psychology Review, 6*, 247–264. <http://dx.doi.org/10.1023/B:CCFP.0000006292.61072.d2>
- Substance Abuse and Mental Health Services Administration. (2020). *Trauma*. Retrieved from <https://www.integration.samhsa.gov/clinical-practice/trauma>

- Thorwart, A., & Livesey, E. J. (2016). Three ways that non-associative knowledge may affect associative learning processes. *Frontiers in Psychology, 7*, 2024. <http://dx.doi.org/10.3389/fpsyg.2016.02024>
- Tomarken, A. J. (1995). A psychometric perspective on psychophysiological measures. *Psychological Assessment, 7*, 387–395. <http://dx.doi.org/10.1037/1040-3590.7.3.387>
- van der Kolk, B. (1989). Psychobiology of the trauma response. In B. Lerer & S. Gershon (Eds.), *New directions in affective disorders* (pp. 443–446). New York, NY: Springer. http://dx.doi.org/10.1007/978-1-4612-3524-8_95
- Van Der Kolk, B. (2007). The developmental impact of trauma. In L. J. Kirmayer, R. Lemelson, & M. Barad (Eds.), *Understanding trauma: Integrating biological, clinical, and cultural perspectives* (pp. 224–241). New York, NY: Cambridge University Press. <http://dx.doi.org/10.1017/CBO9780511500008.016>
- Weems, C. F., Saltzman, K. M., Reiss, A. L., & Carrion, V. G. (2003). A prospective test of the association between hyperarousal and emotional numbing in youth with a history of traumatic stress. *Journal of Clinical Child and Adolescent Psychology, 32*, 166–171. http://dx.doi.org/10.1207/S15374424JCCP3201_15
- Welsh, G. S. (1956). Factor dimensions A and R. In G. S. Welsh & W. G. Dahlstrom (Eds.), *Basic readings on the MMPI in psychology and medicine* (pp. 264–281). Minneapolis, MN: University of Minnesota Press.
- Wilkinson, G. S. (1993). *The Wide Range Achievement Test: Manual* (3rd ed.). Wilmington, DE: Wide Range Inc.
- Wright, R. J., Mitchell, H., Visness, C. M., Cohen, S., Stout, J., Evans, R., & Gold, D. R. (2004). Community violence and asthma morbidity: The Inner-City Asthma Study. *American Journal of Public Health, 94*, 625–632. <http://dx.doi.org/10.2105/AJPH.94.4.625>
- Zachary, R. A. (1986). *Shipley Institute of Living Scale: Revised manual*. Los Angeles, CA: Western Psychological Services.

Received December 6, 2019

Revision received April 4, 2020

Accepted April 30, 2020 ■