The Fear Response in Dissociative Identity Disorder: a Fear Potentiated Startle Study.

by

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A thesis submitted in partial fulfillment of the requirements for the degree of

B.S. Cognitive & Brain Sciences

Tufts University

April 21, 2017

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Acknowledgements

I wish to express the utmost appreciation to Dr. Lauren A. M. Lebois, PhD, for her direct role and guidance throughout the process. Without Lauren, this undertaking would not have been possible. Thank you to Dr. Lisa Shin and Dr. Elizabeth Race for their valuable input and for their supervision. Thank you also to the study participants for the time, to members of the Neurobiology of Fear Laboratory and the Dissociative Disorders and Trauma Research Program for their contributions, and to Dr. Tanja Jovanovic, Dr. Seth Norrholm, and Antonia Seligowski for ASR training.

Special thanks to the laboratory members working directly on this research: Lauren Lebois, PhD, Cara Bigony, BA, Jonathan Wolff, BS, Sherry Winternitz, MD, Kerry Ressler, MD, PhD, and Milissa Kaufman, MD, PhD.

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Abstract

The overgeneralization of conditioned fear and the impaired ability to extinguish fear are potential mechanisms underlying alterations in arousal associated with trauma-spectrum disorders. Classic posttraumatic stress disorder (PTSD) is associated with an exaggerated acoustic startle response (ASR) and a failure to extinguish the fear response in a fear-potentiated startle (FPS) paradigm. Dissociative identity disorder (DID), a trauma-spectrum disorder linked with chronic childhood abuse and trauma, presents hallmark symptoms including derealization, depersonalization, and intrusive thoughts, which contrast some of the arousal-based symptoms of PTSD. To date, one study has examined ASR and pathological dissociation and found that dissociative experiences in individuals with borderline personality disorder were linked with an attenuated ASR (Ebner-Premier, 2005). We aimed to replicate and extend these findings in an ongoing study of women with histories of childhood abuse and a diagnosis of DID (N = 12; age M = 45.58). Participants completed a self-report battery, reporting high levels of arousal-based symptoms and pathological dissociation, and an FPS paradigm measuring facial electromyography (EMG), heart rate, and skin conductance. Based on button-box responses recorded during the experiment, we found that participants successfully learned to discriminate between the danger and the safety signals. Participants also displayed lower overall FPS compared to what is seen in the literature for both non-clinical and PTSD patients, though levels of pathological dissociation were correlated with greater FPS. This could indicate that increased DID symptomology is associated with severe comorbid PTSD symptoms, while dissociation in general correlates with a dampened autonomic response. Our findings will help provide a basic understanding of the fear response in DID, informing treatment development to manage physiological symptoms.

The Fear Response in Dissociative Identity Disorder: a Fear Potentiated Startle Study.

Trauma-Spectrum Disorders

Trauma-spectrum disorders arise from coping mechanisms to traumatic events that cause chronic behavioral and physiological symptoms and affect a multitude of demographics within the population. The most well-known trauma-spectrum disorder, posttraumatic stress disorder (PTSD), carries an estimated lifetime prevalence of 6.8% (Kessler, 2005). Several other disorders can develop as responses to trauma, such as PTSD with dissociative symptoms and dissociative identity disorder (DID), but they are not nearly as studied as PTSD.

PTSD is a response to a traumatic experience, or chronic trauma, that results in hallmark symptoms such as hyperarousal, hypervigilance, re-experiencing episodes, and avoidance behaviors (American Psychiatric Association, 2013). In addition to the "overactive" symptoms such as hypervigilance and hyperarousal that are common in someone with PTSD, an individual who has experienced certain trauma may also have symptoms of dissociation. Pathological dissociation manifests itself in numerous ways and can affect an individual's memory, sensory experience, and identity (Dell, 2009). Two common symptoms, derealization and depersonalization, reflect this altered perception of experience and are defining indicators in the diagnosis of PTSD with dissociative symptoms (Lanius et al., 2014). Derealization is the sense that some aspect(s) of the individual's environment has become distorted, and depersonalization is the sense of being disconnected from one's body (Dell, 2009). Other types of dissociative symptoms include dissociative fugue or amnesia, intrusive thoughts, and trance-like episodes (Dell, 2009).

While many individuals who have undergone trauma may have dissociative experiences, the diagnosis of dissociative identity disorder (DID) describes individuals who have experienced severe chronic childhood maltreatment and abuse. DID is a trauma-spectrum disorder that gives rise to DID-specific symptoms, often in tandem with some classic symptoms of PTSD such as hyperarousal (APA,

2013). In fact, comorbidity rates of PTSD in individuals with DID have been reported to be 80-100% (Armstrong, 1990).

The dissociative symptoms in DID may originate as a defense mechanism whereby the victim mentally distances himself or herself from the trauma. Repeated neglect eventually results in a fragmentation of the mind, or various personality states (Dell, 2009). In particular, the symptom of partially dissociated intrusions is DID-specific and describes the "switching" from one personality state to another, which may be provoked by certain triggers or generally aversive events (Dell, 2009).

Based on research, clear patterns are emerging as biological markers of PTSD. The biomarkers of dissociation, however, are still largely unknown. Investigating the physiological symptoms of trauma-spectrum disorders is a crucial step towards understanding the etiology of the disorders, informing potential treatments, and directing future research regarding the neural and physiological correlates of DID and other trauma-spectrum disorders.

Biomarkers of Trauma-Spectrum Disorders

The study of physiological symptoms associated with trauma-spectrum disorders is typically done through measuring conductance of facial muscles (EMG), heart rate (HR), Electrodermal activity (EDA), systolic blood pressure, and diastolic blood pressure (Pole, 2007). The association between symptoms and their respective physiological patterns has allowed researchers to understand more about the physical responses of a person with classic PTSD. The DSM-V criteria for PTSD reflect a cyclical autonomic dysfunction that gives rise to a dichotomy of autonomic symptoms, alternating between "states of heightened sympathetic arousal" (e.g. panic and hypervigalence) and states of "parasympathetic dominance" (e.g. withdrawal and flattened affect) (Scaer, 2001). States that are driven by the sympathetic branch of the Autonomic Nervous System (ANS), or the system's "gas pedal," would result in physiological outputs such as higher HR, increased EDA, and increased blood pressure

(Sherin, 2011). The parasympathetic branch of the ANS, commonly thought of as the "break pads," could moderate a lower HR and reduced EDA (D'Andrea et al., 2013).

Many studies have attempted to map this cyclic ANS dysfunction with PTSD. For example, several investigations have found a strong connection between elevated HR and PTSD (Cohen et al., 1997; Hopper, et al., 2006; Sack et al., 2004). In another study, patients with PTSD showed higher resting HR and diastolic blood pressure than healthy controls did, though both groups exhibited similar systolic blood pressure (Buckley & Kaloupek, 2001).

Debate is ongoing regarding the terms of ANS dysfunction in traumatized individuals. While aroused states are traditionally thought of as being driven by the sympathetic system, a diminished parasympathic tone could also contribute to overall dysregulation (Tan et al., 2011). Studies have suggested that the elevated HR in PTSD may be mediated by reduced parasympathetic activity rather than elevated sympathetic activity (Hopper et al., 2006; Sack et al., 2004). Through examining a specific psychophysiological pattern, researchers thus continue to augment our understanding of the mechanisms of PTSD dysfunction. The exact role and characteristic pattern of the autonomic system in PTSD, beyond "dysregulation," still remains unclear.

Similarly to the arousal-based symptoms of classic PTSD, several dissociative symptoms are also likely mediated by the ANS. In contrast to classic PTSD symptoms, however, the distinctive symptoms of dissociation reflect a reduced emotional experience and are correlated with a "blunting" of the ANS (Ebner-Premier, 2005). Scaer (2001) associates the hyperactive aspects of PTSD to a state of sympathetic arousal, and links dissociation with a primarily parasympathetic tone. In a study of dissociative states, Reinders et al. (2012) found that "trauma-identity states" characterized by physical reactions such as the freeze, flight, or fight response, are likely facilitated by a dominant sympathetic nervous system, while the dorsal vagal branch of the parasympathetic nervous system likely mediates the more behaviorally numb states (Reinders et al., 2012).

Griffin et al. (1997) classified participants who were victims of rape as high versus low dissociators based on an interview assessing the dissociative experiences that the participant may have had during their trauma, or levels of peritraumatic dissociation. By measuring EDA and HR while participants spoke about their trauma, Griffin and colleagues found lower physiological activity in the high dissociation group, even among those who had classic PTSD symptoms, which was the majority of the group (Griffin et al., 1997).

Studies on the neurobiology of dissociation have pointed to several key brain structures and pathways that might mediate dissociative responses. Notably, the thalamus plays a central role in sensory integration and may contribute to the immobility response of individuals exposed to trauma (McKinnon et al., 2016). After repeated trauma, this "defensive mind-body state" becomes learned and repeats in the presence of trauma-cues (Kozlowska et al., 2015).

Decreased physiological activity may be correlated with dissociation in some cases, but the comorbidity of classic PTSD symptoms and DID-specific symptoms in an individual who has experienced trauma makes it difficult to definitively point to a specific type of ANS dysregulation, even for particular symptoms or measures.

Acoustic Startle Response

One way to measure physiological output and to establish patterns that might give insight into the functioning of the autonomic nervous system is through recording an Acoustic Startle Response (ASR). The ASR measures the reflex startle of a participant to an auditory burst of noise. The startle is measured through electromyogram (EMG) recordings of eye muscle conductivity in response to the startle probe (Jovanovic et al., 2005; Jovanovic et al., 2006). An exaggerated ASR is a well-established physiological correlate of PTSD (Orr et al. 1995; Shalev et al. 1992) The ASR is an ideal way to study the fear response due to the fact that the startle circuit is connected with the amygdala, a structure that plays a key role in trauma-spectrum disorder pathology (Davis et al., 1993).

The ASR has been studied in individuals with DID compared to individuals with other dissociative disorders and a group of non-diagnosed controls. Dale et al. (2008) focused on prepulse inhibition (PPI) of the ASR, which refers to the attenuation of the response to a strong stimulus, or the pulse, when it is preceded by a weaker stimulus, or the prepulse. The inhibition of the pulse reflects an automatic attentive process that focuses on the initial stimulus and thus quiets the processing of stimuli that follow immediately after. Participants with DID displayed an increased PPI compared to the other two groups, which could indicate a voluntary shift in attention away from an unpleasant stimulus (Dale et al., 2008). Those with DID also exhibited reduced habituation to the stimuli, a reaction that has been linked with hypervigilance and therefore more classic PTSD-like symptomology (Orr et al., 2002).

Ebner-Premier and colleagues (2005) also studied ASR and dissociation in humans, but were interested primarily in affective dysregulation and dissociation in borderline personality disorder (BPD). Their data showed that the presence of high dissociative experiences during the startle task was correlated with a reduced startle response when compared to the data of individuals with low dissociative experiences during the task and to controls. These findings highlight the dampening effect that dissociative thoughts, feelings, and experiences might have on an individual's psychophysiology during a threatening, fear-eliciting experience.

Fear-Potentiated Startle

The overgeneralization of conditioned fear and the impaired ability to extinguish fear are potential underlying mechanisms of dysfunction associated with trauma-spectrum disorders, and are well studied in classic PTSD (Norrholm et al., 2011). A fear-potentiated startle (FPS) model, based on Pavlovian conditioning, teaches participants to fear an expected and predictable cue. This isolates the fear response, whereas paradigms without explicit fear signals tend to produce more generalized anxiety than isolated fear responses (Grillon, 2002).

The paradigm consists of two phases: an acquisition, or conditioning, phase, and then an extinction phase. During fear acquisition, a neutral stimulus, or the conditioned stimulus (CS), is paired with a naturally aversive stimulus, the unconditioned stimulus (US). Examples of unconditioned stimuli include an abrupt tone, a mild shock, or an aversive blast of air (Lissek et al., 2005). If the pairing is successful and fear is learned, a presentation of the CS alone then elicits the unconditioned fear response, or a startle (Jovanovic et al., 2005). The fear extinction phase involves a de-coupling of the CS from the US by presenting the CS repeatedly without the US. Conditioned fear has been extinguished once the presence of the CS alone no longer provokes the paired fear response (Jovanovic & Norrholm, 2011).

This paradigm is widely used in animal and human research to investigate fear mechanisms, due in part to the fact that responses can be recorded noninvasively via multiple measures. As the exaggerated startle is one of the most commonly reported symptoms of PTSD (Pole, 2007), the startle task is thus frequently used to study individuals who have experienced trauma. A number of neuroimaging studies show a reduced ability of the prefrontal cortex to inhibit amygdala activity in the mind of someone with PTSD in response to an aversive stimulus, thus corroborating the use of the FPS in studying trauma (Rauch et al., 2006; Shin et al., 2005). A meta-analysis of fear-extinction studies found that PTSD symptoms generally correlate with increased EMG activity, elevated HR, higher EDA, and slower EDA habituation (Metzger et al., 1999). Orr et al. (2002) also reported that studies recording physiological data in response to startling tones have found higher mean HR, larger EDA magnitudes, and slower EDA habituation rates in individuals with PTSD. In an FPS paradigm, participants with PTSD show an amplified startle response during acquisition phases and a diminished ability to extinguish the fear during the extinction phase compared to controls (Jovanovic & Norrholm, 2011).

The aversive probe leads to an exaggerated startle response in someone who has PTSD; the same paradigm when applied to dissociation can serve to characterize the physiological basis of dissociative symptoms further, and elucidate potential differences between classic-PTSD reactions and dissociation-specific reactions. Based on the fact that dissociation is generally associated with detaching oneself from the threatening environment and a dampening of the ANS, exposing a dissociative individual to an aversive stimulus might result in other defense mechanisms. There has yet to be a study on a FPS paradigm and individuals with DID.

The Present Study

The current investigation will be the first study to examine how people with DID react to a FPS paradigm. The results of Ebner-Premier and colleagues (2005) on ASR and dissociative individuals with BPD suggested that higher dissociative experiences were associated with reduced startle, but these results need replicating within a population of individuals with DID and other dissociative disorders. The results of Dale and colleagues (2008) also point to the ability of individuals with DID to shift attention away from an unpleasant stimulus, which could be reflected as an attenuated startle.

Assessing the responses of patients with DID to the startle paradigm is especially interesting given that DID symptoms include those of PTSD. The etiology of DID is connected with chronic childhood maltreatment, and patients often express arousal-based symptoms such as hypervigilance and intense flashbacks. The PTSD response to the startle paradigm has been well studied and correlates with an exaggerated startle response. In contrast, preliminary evidence suggests that dissociative symptoms might reflect a dampened output. Yet, individuals with DID were found to display reduced habituation, which is a marker of hypervigilance and classic PTSD symptoms (Orr et al., 2002). An exaggerated or diminished fear potentiated startle compared to what is expected will help

us begin to understand the mechanisms that lead to their symptoms and will lend more insight into the autonomic functioning of individuals with dissociative disorders.

We analyzed the physiological responses of our participants during a fear conditioning and extinction startle paradigm using EMG as a measure of eye muscle activity in response to an airblast. We hypothesized that throughout acquisition, participants would learn to discriminate between the danger signal and safety signal and startle more to the danger signal. We expected to see this pattern both cognitively, through their responses on a button box indicating their expectation of the US, and physiologically, through the EMG startle data. We also predicted that participants would maintain a learned discrimination between the danger and safety signals at the beginning of the extinction session, demonstrated by an increased startle to the danger signal. Throughout extinction, after repeated presentations of the danger signal without reinforcement, successfully extinguished fear would be reflected by an equal startle to the safety and the danger signals.

Based on the literature and avoidance-type symptoms of the disorder, we expected to see an overall dampened autonomic output for DID patients in response to an aversive stimulus. We anticipated that higher levels of pathological dissociation might correlate with lower EMG startle values during acquisition and extinction. Alternatively, we thought it also possible that the startle task might lead the participants with DID to exhibit high levels of physiological arousal due to more classic PTSD symptoms.

Methods

Participants

Participants were recruited as part of a larger study on the neurobiology of traumatic dissociation at McLean Hospital in Belmont, Massachusetts. All 12 participants were women (*M* Age = 45.56, *SD* = 14.15), had a history of chronic childhood maltreatment, and met diagnostic criteria for DID as assessed by a trained clinician using the Structured Clinical Interview for DSM-IV Dissociative

Disorders. All participants self-identified as white. In terms of ethnicity, two participants identified as Hispanic or Latino, with the remaining majority of participants identifying as non-Hispanic or as belonging to another ethnicity. Two thirds of the sample were taking a variety of medications including benzodiazepines, antidepressants, anticonvulsants, antihistamines, sedatives and antipsychotics. Only four of our participants were not taking any medications. Exclusion criteria included history of or current psychosis and major medical illnesses as assessed by the intake of patient history, and current or within the past month substance or alcohol use disorder. Before participating in the study, all individuals were briefed and provided written informed consent that was approved by the McLean Hospital Institutional Review Board. Participants completed the psychological assessment, the physiological behavioral task, and other components of the larger study over seven hours, typically spread out over two days that spanned no longer than one week. Participants also completed a breathalyzer and a urine test to account for potential interactions with drugs. Participants received $50 compensation for the fear potentiated startle portion of the study.

Measures

Participants completed a battery of self-report and interview measures that assessed PTSD and DID pathology, as well as childhood and adult trauma history. The two primary questionnaires isolated in this thesis are the PTSD Checklist for DSM-5 (PCL-5) (Weathers et al., 1993; Weathers et al., 2013) to index PTSD symptoms and the Multidimensional Inventory of Dissociation (MID) (Dell, 2009) to measure dissociative symptoms. The data of both these self-report questionnaires were collected through REDCap software on a laboratory computer.

The PCL-5 consisted of 20 items that evaluated the 20 PTSD symptoms listed in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5). Each question was answered on a 4-point scale (0 = "Not at all," 1 = "A little bit," 2 = "Moderately," 3= "Quite a bit," 4 = "Extremely") and provided both a total symptom severity score as well as 4 subscales that pertained

to the PCL-5 symptom clusters (Cluster B = Intrusions; Cluster C = Avoidance Symptoms; Cluster D = Negative Thoughts or Feelings; Cluster E = Hyperarousal and Reactivity). Analyses on the PCL-5 have suggested a cut-off point of 38 for the total symptom score (Weathers, 1993), and have found the PCL-5 to possess consistency, reliability, and validity (Blevins et al., 2015).

The MID evaluated a variety of dissociative symptoms and generated scale scores as well as categorization into the following diagnoses: DID, PTSD, dissociative disorder not otherwise specified (DDNOS), and BPD. Of the 218 items, 168 were for dissociation and 50 were for validity. The test used an 11-point Likert scale to complete each item, and assessed for 23 major symptoms of dissociation. Two of the symptom subscales that are discussed here are the depersonalization and derealization cluster and the partially dissociated intrusions cluster. These were chosen on the basis that depersonalization and derealization are hallmark dissociative experiences that are typically evaluated in the majority of dissociation literature, and partially dissociated intrusions serve as a DID-specific measure that has the potential to impact performance on the behavioral task. The MID has also been found to demonstrate strong reliability and validity (Dell, 2006).

Fear Conditioning & Extinction Task Design

The fear conditioning and extinction behavioral task was based on a paradigm developed by Norrholm and colleagues (2006). An FPS paradigm was used to elicit the ASR, measured in terms of a facial electromyography conductivity. The task consisted of first an acquisition phase, followed by a ten-minute break and then an extinction phase. Figure 1 displays a visualization of trial types in both acquisition and extinction. In acquisition, the airblast, or the unconditioned stimulus (US), was paired with shape A to serve as the danger signal (A+), leaving the unpaired shape B to be the safety signal (B-). Shape A was a blue square, and shape B was a purple triangle. Shape A can also be called the conditioned stimulus, as it was the stimulus that the participant was conditioned to fear. Since it had the airblast paired with it, Shape A was the CS+ while Shape B was the CS-. The stimuli are referred

to as A+ and B- throughout to distinguish them during extinction, where they become A and B without the presence of an airblast.

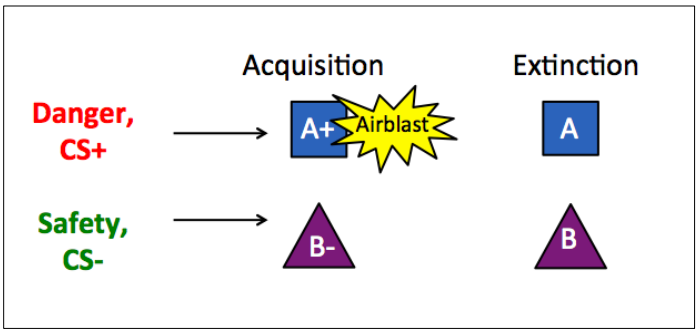

Figure 1. Stimulus Pairing. The airblast, or the unconditioned stimulus (US), is paired with shape A and not shape B. Shape A comes to represent the danger signal (CS+), and shape B represents the safety signal (CS-).

The acquisition phase began with habituation, during which the two shapes were presented to the participant without the air blast or tones. The shapes were displayed on the screen using SuperLab software (SuperLab, Cedrus Corporation, San Pedro, California). After the habituation block, three experimental blocks were presented with counterbalanced trials. One experimental block consisted of four presentations of each stimulus type: the reinforced conditioned stimulus (A+), a nonreinforced stimulus (B-), and the acoustic probe alone ("Noise Alone", NA) (See Figure 2).

The stimuli within a block were presented at randomized intervals between 9-22 seconds, and the colored shapes remained on the screen for 6s. The 180-dB noise probe was presented for 40ms, with the 140-p.s.i. airblast (US) delivered to the larynx 500ms later, lasting for 100ms. The airblast was released by a compressed air tank that was operated by a solenoid switch (Norrholm et al., 2006). Figure 3 displays the timeline of the different stimuli within the acquisition phase. Four extinction blocks with four trials of each stimulus type (NA, A, and B) were presented, but without the reinforcement of the US.

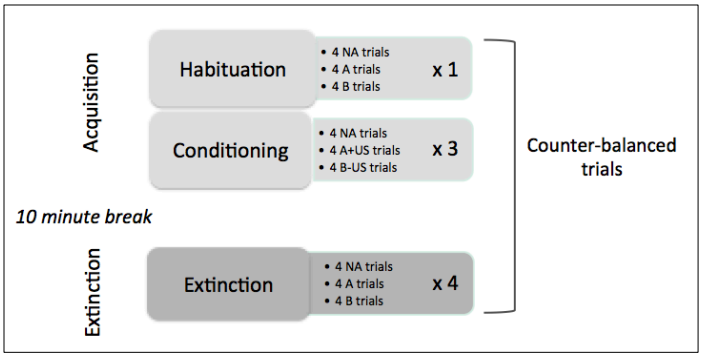

Figure 2. Paradigm Structure. NA trials are Noise Alone, whereas A or B trials are presented either with the airblast (A+) or without (B-) during acquisition. All trials were presented without the airblast (A or B) during extinction.

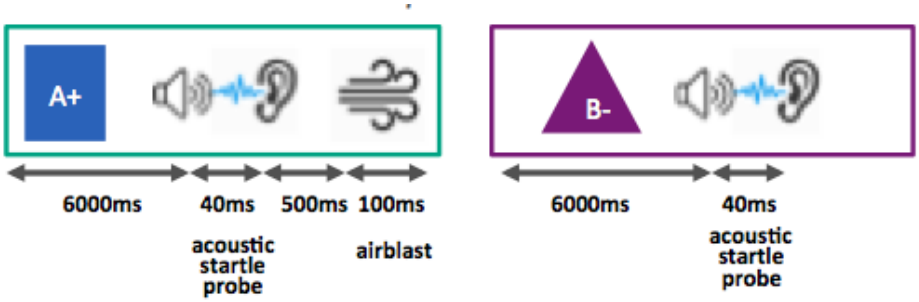

Figure 3. Startle Probe Timeline. The startle is measured in response to the acoustic probe in anticipation of the airblast.

Procedure

Participants first finished the questionnaires and then a urine screen and breathalyzer test to rule out the interaction of any substances. The participant completed the task alone in a small room with a closed door, with the equipment leading in through a hole in the wall. The research team was on the other side of the wall supervising the data collection on the screens and could hear the participant at all times. Before the task began, research personnel applied electrodes to record facial electromyography activity (EMG), electrocardiogram activity (ECG), and electrodermal activity (EDA) through the galvanic skin response (GSR). Two EL504 EMG electrodes were attached under the

participant's right eye on the orbicularis oculi muscle after the researcher exfoliated the area with Lemon Prep scrub and applied Redux paste electrolyte gel. A ground electrode was placed behind the right ear. Two EL507 GSR pads were attached to the participant's non-dominant hand with additional Isotonic paste. Three EL504 ECG electrodes were placed on the left forearm, right ribcage, and right clavicle to record heart rate, with additional Redux paste gel. The participant wore a vest with an airblast nozzle and headphones over the ears. The physiological data were recorded with Biopac MP150 (Biopac Systems, Aero Camino, California).

The participants were instructed to watch shapes presented on the computer screen and judge whether or not they thought a blast would be delivered after each shape. They were informed that blasts would follow some shapes and not others, and that there would also be some tones in the earphones. They recorded their predictions (i.e. unconditioned stimulus expectancy ratings) on a button box during the experiment (see Figure 4) (SuperLab, Cedrus Corporation, San Pedro, California). A rating of "1" meant that the airblast was expected, a rating of "0" meant the participant was unsure, and a rating of "-1" meant that the participant did not expect an airblast to follow the shape.

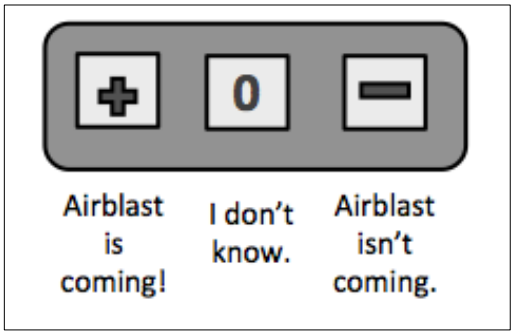

Figure 4. Response Button Box. The pad on which participants recorded their expectancy ratings of the air blast following shape presentation.

The fear acquisition phase lasted for twenty minutes and was followed by a ten-minute break. During this break, research personnel asked the participants to rate their discomfort with the blast and the ear tones the first time each occurred, as well as potential dissociative experiences during the task.

After the break, the participants began the fear extinction phase. The participants were informed that the structure of the second phase was the same as that of the first, and were instructed to continue recording their expectancy ratings on the button box. Unbeknownst to the participant at the start of the extinction phase, no airblast would occur during this phase of the experiment. After extinction, the presence of dissociative experiences was again assessed.

EMG Processing and Statistical Analysis

Startle Response Data.

The EMG startle response data were sampled at 1000Hz. Using BioPac, we applied an amplifier gain of 5000. The data were further preprocessed using MindWare software (MindWare Technologies, Ltd., Gahanna, OH). According to the methods of similar studies (e.g. Jovanovic et al., 2010), we filtered the EMG signal with a low frequency cutoff of 28Hz and a high frequency cutoff of 500Hz. We used the peak amplitude of the eye muscle contraction 20-200ms after the acoustic startle probe as the measure of the fear response. EMG data were used to consider both startle magnitude and difference scores. Participant responses from the button box were used as a measure of US expectancy.

Statistical Analysis.

FPS difference scores (DS) were calculated with the EMG data for acquisition and extinction to account for the fact that repeated exposure to a startle probe can lead to an attenuation of the startle, or habituation (Norrholm et al., 2008). For a given acquisition block, the difference score was the average startle magnitude to the CS minus the average startle to the NA trials of that same block (e.g. ACQ1 DS = [Average startle to A+ in ACQ1] - [Average startle to NA in ACQ1]). For a given

extinction block, the difference score was the average startle magnitude to the CS minus the average startle to the NA trials of the entire extinction phase (e.g. EXT1 DS = [Average startle to A in EXT1] - [Average startle to all NA trials in EXT]).

We ran a paired-samples t-test to analyze US expectancy data from the response button box. To consider the EMG data, we used a repeated measures analysis of variance (ANOVA) to analyze the EMG difference scores by stimulus type (NA, CS+, or CS-), by block (CSH, ACQ1, ACQ2, ACQ3 for acquisition, and EXT1, EXT2, EXT3, and EXT4 for extinction), and a block x trial type interaction. We also ran bivariate correlational statistics with standardized Z-scores of several questionnaires. All analyses were performed with SPSS 24.0 for Mac (SPSS, Chicago, Illinois), with $\alpha = .05$.

Results

Descriptive Statistics

The participants were administered a variety of questionnaires. Table 1 includes the descriptive statistics of the PCL-5 questionnaire, each PCL-5 cluster score, the Multidimensional Inventory of Dissociation (MID) total score, and the scores from the derealization, depersonalization, and the 11 Partially Dissociated Intrusion subscales of the MID.

The scores indicate high levels of clinical pathology in our participants. The average PCL-5 total symptom score of 47.75 was larger than the suggested clinical cutoff of 38 (Weathers, 1993), and the average MID score of 51.03 was also above the range for potential dissociative pathology combined with PTSD, which is 31-40 (Dell, 2012). Figure 5 displays the relationship between the MID and PCL-5 scores for each participant, and reveals a positive correlation between the scores, $r = 0.58$, $p < .049$.

Table 1
Descriptive Statistics for Study Population

	N	Minimum	Maximum	Mean	Std. Dev.
Age	12	23.00	61.00	45.58	14.15
PCL-5 Total Symptom Severity Score	12	16.00	66.00	47.75	16.16
PCL-5 Cluster B Severity Score	12	4.00	20.00	12.50	5.04
PCL-5 Cluster C Severity Score	12	1.00	8.00	5.00	2.80
PCL-5 Cluster D Severity Score	12	5.00	25.00	17.58	6.32
PCL-5 Cluster E Severity Score	12	5.00	19.00	12.67	3.92
MID Mean Score	12	6.30	82.90	51.03	19.33
MID Depersonalization and De-realization Subscale Score	12	6.70	81.70	50.91	21.40
Average of 11 Partially Dissociated Intrusions Scores	12	0.04	0.97	0.60	0.27

Acquisition

US Expectancy.

At each presentation of the CS, participants rated their expectation that the US, the airblast, would follow. A paired-samples t-test showed that, for the final block of acquisition (ACQ3), participants accurately classified A+ as a signal for the airblast (M = .85, SD = .27), and B- as the safety signal (M = -.56, SD = .72), $t(11) = 5.30$, $p < .001$.

Figure 6 displays the response pad data collected from participants during the acquisition phase. This data serves as a confirmation that the paradigm was successful and that participants learned that A+ was the danger signal, and that B+ was the safety signal.

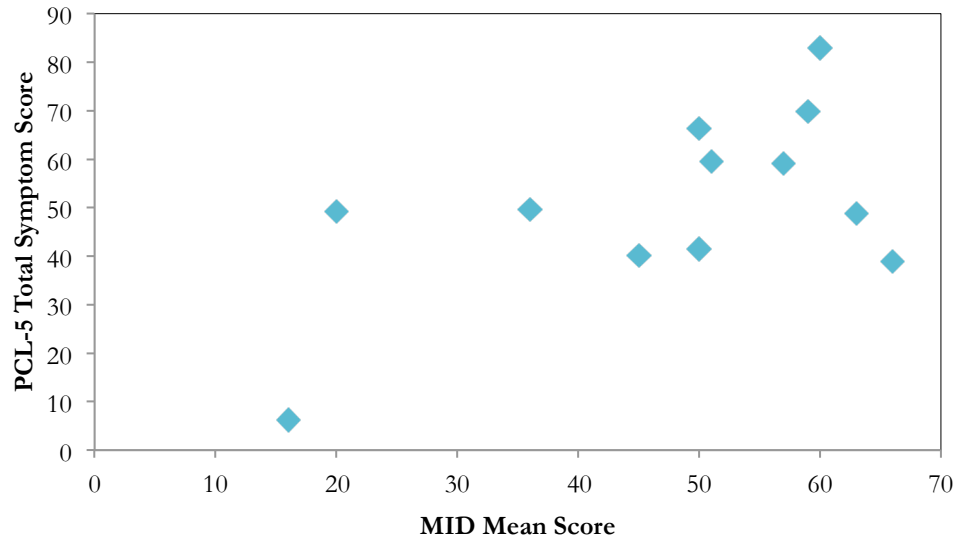

Figure 5. MID vs. PCL-5. The correlation between the MID Mean Score, which measures pathological dissociation, and the PCL-5 Total Symptoms Score, which measures PTSD symptoms, for each participant, $r = 0.58$, $p = .049$.

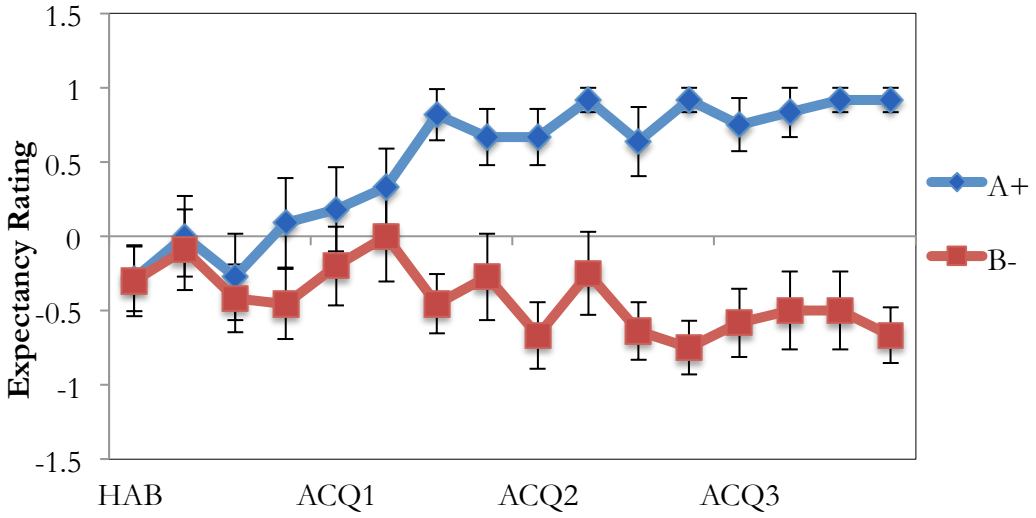

Figure 6. US Expectancy Ratings for Acquisition Phase. Ratings of 1.0 = "danger," 0 = "I don't know," and -1.0 = safety." A+ represents the danger signal and B- represents the safety signal.

EMG Magnitude.

Figure 7 displays the average startle magnitudes in response to the NA trials, the danger signal (A+), and the safety signal (B-). On average, participants had lower magnitudes than what is typically seen in the literature. For example, Norrholm and colleagues (2008) report average magnitudes of ~100μv to NA trials and >150μv to A+ trials in ACQ1 for a non-clinical population. Our participants display startle magnitudes of <100μv to NA and A+ trials in ACQ1.

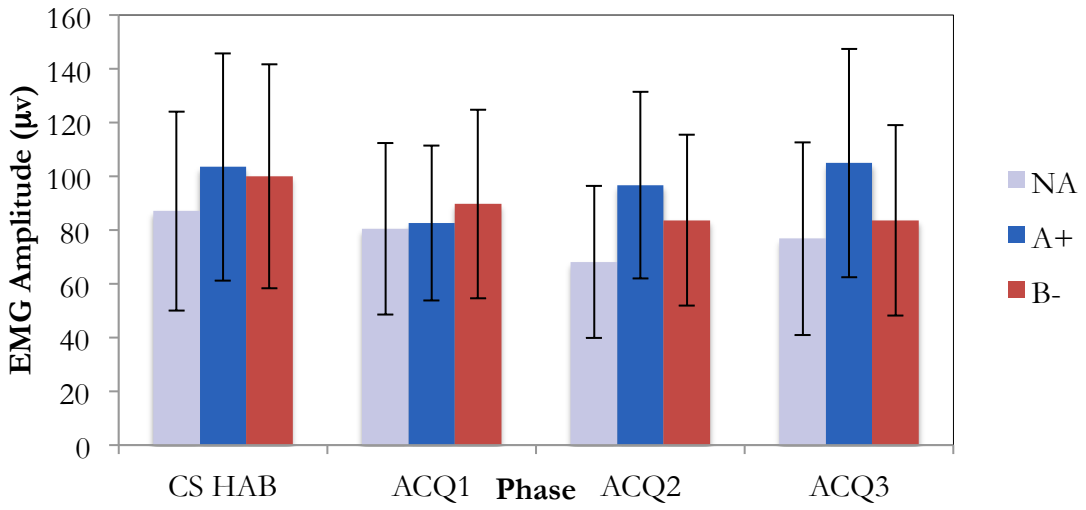

Figure 7. EMG Magnitudes for Acquisition Phase. NA = Noise Alone, A+ = danger signal, B- = safety signal. Error bars are +/- one Standard Error (SE).

EMG Difference Score.

Figure 8 illustrates the DS for each acquisition block plotted, which reflects the average EMG startle amplitude of the NA trials for a specific block subtracted from the average startle amplitudes to the shapes for the same block. The shapes on the screen appeared as either the blue square (A+, or the CS+) or the purple triangle (B-, or CS-). During the habituation phase, "CS HAB" on Figure 8, both

A+ and B- flashed on the screen but no airblast occurred. For the remainder of the acquisition trials, A+ (the danger signal) was always preceded by the air blast and B- (the safety signal) was not.

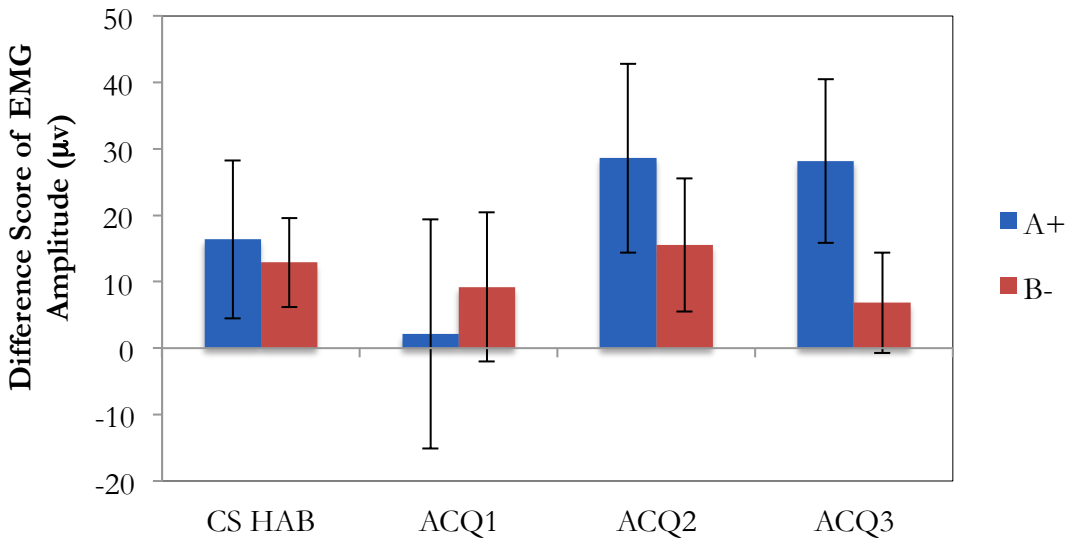

Figure 8. EMG Difference Scores for Acquisition Phase. A+ = danger signal, B- = safety signal. Error bars are +/- one Standard Error (SE).

The severity of partially dissociated intrusions, a symptom that is more specific to dissociative identity disorder, was negatively correlated with the startle to the safety signal, B-, in acquisition block 1, $r(10) = -.42$, $p = .086$. Thus, the higher the Partially Dissociated Intrusions subscale score of the MID, the less the participant startled to the B-. This same pattern was observed in acquisition block 2, where participants reporting a higher score on the scale of partially dissociated intrusions startled less to the safety signal $r(10) = -.44$, $p = .077$. This indicates that more DID symptoms point to a trend towards better discrimination between the safety signal, the B-, and the danger signal, the A+.

Our key hypotheses for the acquisition phase involved a two-way interaction between the effect of block and stimulus type. Based on the literature, we hypothesized that throughout acquisition, there would be a gradual discrimination between A+ and B- reflected by the EMG difference scores

(e.g. Norrholm et al., 2008). This discrimination would reflect that participants learned which shape was the safety signal and which was the danger signal, and that they were startling more in apprehension of the danger signal.

To examine the EMG data and determine whether the participants eventually discriminated between A+ and B-, we performed a Repeated Measures ANOVA to analyze the effect of block (CSH, ACQ1, ACQ2, ACQ3), stimulus type (A+ or B-), and the block x stimulus type interaction.

Contrary to our hypotheses, the interaction between the measures was not significant, $F(3, 33) = 1.78$, $p = .170$. Additionally, we did not find a main effect for the block type, $F(3, 33) = .92$, $p = .443$, or a main effect of stimulus type, $F(1, 11) = .97$, $p = .347$. This reflects that there was no significant difference in EMG startle DS for the participants across the acquisition blocks, or between the safety and the danger signals. Overall means (see Figure 7), however, do suggest this expected pattern of A+/B- discrimination across the acquisition blocks. ACQ3 reflects a higher overall startle than CSH HAB, and it also appears that there is a discrimination between A+ and B- in the final block, with there being a greater startle to the A+. A lack of a statistically significant difference is likely because the observed power values for our acquisition data were extremely low, in the range of $\beta = .14$-$.42$.

Another hypothesis we had for the acquisition phase of the startle paradigm was that higher levels of pathological dissociation might be correlated with lower EMG DS values, due to the fact that dissociative symptoms are frequently associated with diminished ANS output. To test this, we ran bivariate correlational statistics with standardized Z-scores of several questionnaires.

On the contrary, depersonalization and derealization scores on the Multidimensional Inventory of Dissociation (MID) Questionnaire were significantly correlated with startle to the A+ in acquisition block 3, $r(10) = .65$, $p = .017$, reflecting that participants with more severe derealization and depersonalization symptoms had higher FPS in the final block of acquisition. This same MID score of

derealization and depersonalization was also positively correlated with startle to the habituation block, $r(10) = .56$, $p = .030$. This result will be further examined in the discussion. Overall, however, our participants' DS did appear low when compared to some data from the literature. For example, Norrholm et al. (2008) report DS of ~70μv in ACQ1 to both A+ and B-, whereas the DS of our participants are less than 10μv for the same block.

Extinction

US Expectancy.

Participants continued to rate their expectation of the US during each presentation of the CS (see Figure 9). In order to determine whether or not the participants sustained their learning from the acquisition phase, we ran a paired-samples t-test on their response pad data from extinction block 1. The results indicated that, during the first block of extinction, participants more often rated A as "I don't know" ($M = .06$, $SD = .66$), and B as the more safe signal ($M = -.27$, $SD = .66$), $t(11) = 2.40$, $p = .035$.

We ran another paired samples t-test to compare the expectancy ratings of A during early extinction and late extinction to investigate whether our participants behaviorally extinguished their fear. There was not a significant difference between the expectancy ratings for A during EXT1 ($M = 0.06$, $SD = .66$) and for A during EXT3 ($M = .00$, $SD = .79$), $t(11) = .24$, $p = .812$. This reflects that participants continued to rate A as "I don't know" throughout the 20 minutes of extinction.

EMG Magnitude.

Figure 10 displays the startle magnitudes to NA, A, and B trials throughout extinction. As in the case of acquisition, participants startled at a much lower magnitude than what is typically seen in the literature for control participants. For example, Norrholm et al. (2006) reports initial startle magnitudes of ~200μv to the danger signal at the start of extinction, whereas our participants startled at levels of ~70μv.

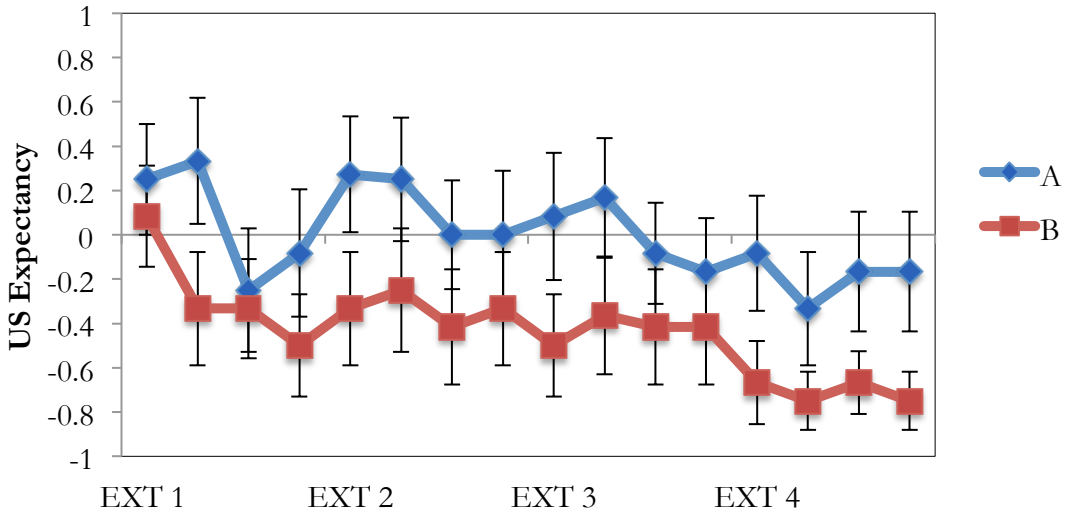

Figure 9. US Expectancy Ratings for Extinction Phase. Ratings of 1.0 = "danger," 0 = "I don't know," and -1.0 = safety." A represents the former danger signal and B represents the former safety signal. Error bars are +/- one Standard Error (SE).

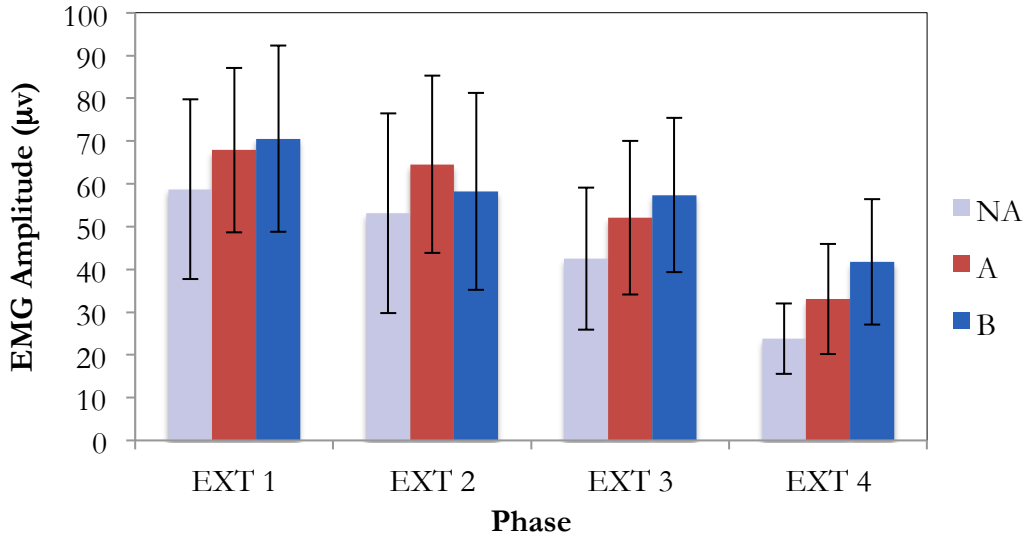

Figure 10. EMG Magnitudes for Extinction Phase. NA = Noise Alone, A = former danger signal, B = former safety signal. Error bars are +/- one Standard Error (SE).

EMG Difference Score.

Figure 11 depicts the EMG startle difference scores for the extinction phase of the behavioral task. The difference score for each extinction block reflects the average EMG startle amplitude of all the NA trials throughout the whole extinction session (19 values in total) subtracted from the average startle amplitudes to the CS for the four specific blocks (EXT1, EXT2, EXT3, EXT4).

For the extinction phase, we hypothesized that the participants would maintain discrimination between the safety signal and the danger signal at the start of extinction due to generally established extinction patterns (e.g. Norrholm et al., 2006). According to the typical pattern, we also expected that repeated presentation of the two shapes without an airblast would lead to the eventual extinction of an increased fear response towards A+ (e.g. Norrholm et al., 2006). As there was no airblast presented in this phase, A+ and B- are referred to as A and B in extinction.

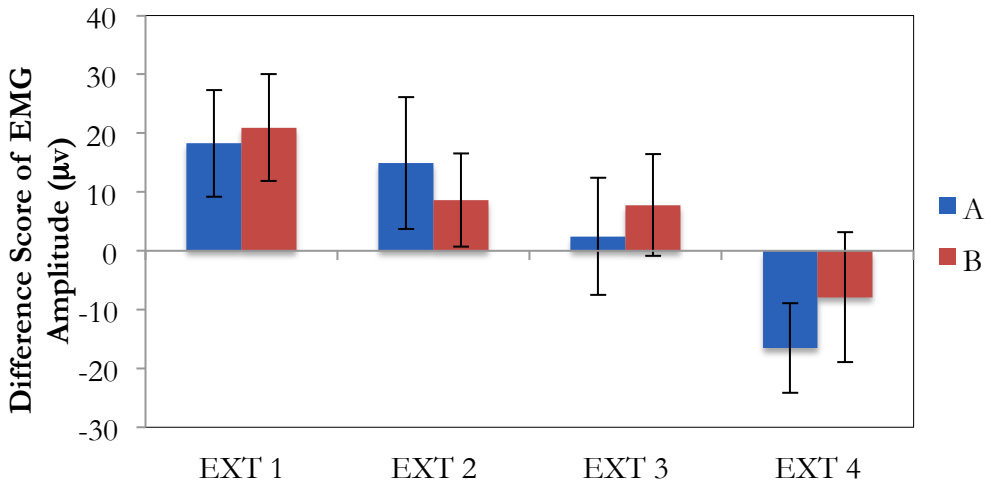

Figure 11. EMG Difference Scores for Extinction Phase. A = former danger signal, B = former safety signal. Error bars are +/- one Standard Error (SE).

We performed a Repeated Measures ANOVA to analyze the effect of block (EXT1, EXT2, EXT3, EXT4), stimulus type (A or B), and the block x stimulus type interaction. There was a main effect of block indicating that participants significantly extinguished fear across the four blocks, $F(3, 33) = 5.75$, $p = .003$. This mirrors the expected pattern for extinction found in the literature, and is consistent with our hypotheses.

Follow-up pairwise comparisons further demonstrated a difference between EMG DS in the earlier extinction blocks compared to the later extinction blocks. Extinction block 1 was significantly different compared to block 4, $p = 0.039$. There was also a trend towards extinction block 1 being different from extinction block 3, $p = .087$, and a trend towards extinction block 2 being different from extinction block 4, $p = 0.094$.

There was no main effect of stimulus type, $F(1, 11) = .50$, $p = .496$, reflecting that participants' startles to the former danger and safety signals were not significantly different even at the start of acquisition. The interaction between block and stimulus type was also not significant, $F(3, 33) = .44$, $p = .725$, showing that there was no change in discriminatory patterns throughout the four extinction blocks. The observed power values for these measures were very low, i.e. $\beta = .093$-$.129$.

In line with our acquisition hypothesis of higher dissociative symptoms potentially correlating with diminished startle scores, we proposed that participants who scored higher on measures of dissociation might reflect lower EMG difference scores throughout extinction as well. As in acquisition, we found that our DS overall were lower than what is seen in the literature for both clinical and non-clinical populations (e.g. Norrholm et al., 2011). For example, Norrholm and colleagues (2011) report DS of ~30μv in control participants for early extinction (blocks 1 and 2 in a 6-block extinction paradigm) and DS of ~50μv for participants with PTSD. In comparison, our participants display DS of ~20μv in EXT1.

We ran a bivariate correlation analysis with standardized Z-scores of several questionnaire means to further examine the relationship between dissociation and extinction. The difference score of the startle to A, the former danger signal, in extinction block 1 was positively correlated with Cluster E scores of the PCL-5, $r(10) = .49$. $p = .054$. Cluster E of the PCl-5 assesses for arousal and hypervigilance symptoms. Thus, the more hypervigilant a participant was according to their score on PCL-5 Cluster E, the higher they startled to the danger signal in block 1 of extinction. The difference score of the startle to B, the former safety signal, in extinction block 3 was negatively correlated with Cluster C scores of the PCL-5, which assesses for avoidance symptoms, $r(10) = -.51$, $p = .044$. Thus, the higher the startle to the safety signal in extinction block 3, the lower participants scored on the PCL-5 cluster assessing for avoidance symptoms. Put another way, more avoidant individuals startled less to the safety signal at the end of extinction, reflecting that they may have enhanced discrimination between A and B, or had lingering fear of the danger signal.

Discussion

Acquisition

We had two major hypotheses for the acquisition phase of the fear startle paradigm. The first was that there would be a two-way interaction between block type and stimulus type, or that as the acquisition blocks continued, the participants would eventually discriminate between the two conditioned stimuli. The danger signal, or A+, was followed by an airblast throughout extinction and the safety signal, B-, was never followed by an airblast. Thus, we expected to see such a pattern based on the expansive literature on this classic fear conditioning and extinction paradigm.

Our hypothesis was partially supported. The pattern in the EMG DS reflected this discrimination (see Figure 8). The startle to A+ not only appears to increase across the acquisition blocks, but there also appears to be a difference in startle in the final block. The ACQ3 difference scores suggest that participants were startling more to A+ than to B-, which would be confirming the

expected pattern. Though our results on the repeated measures ANOVAs measuring main effects of block type, stimulus type, and the interaction between the two were not statistically significant, this is likely due to the small sample size ($N = 12$) and subsequent low power values.

Further evidence that our participants learned the paradigm, at least on a behavioral level, comes from the US expectancy data. Our results showed that participants significantly rated A+ as the danger signal and B- as the safety signal during the final block of acquisition, indicating that participants were aware of which stimulus predicted the airblast and which did not. Interestingly, other studies have reported a characteristic disconnect between US expectancy ratings and startle measures; however, there is no evidence to show that the US expectancy ratings have any impact on the physiological measures (Hermans et al., 2005; Norrholm et al., 2006). Thus, we saw that participants behaviorally discriminated between the danger and safety signals, but the lack of a correlative and significant physiological pattern could be from the underpowered results or from the characteristic disconnect described in the literature.

Our second major hypothesis was that increased symptomology of dissociation might correlate with lower fear-potentiated startle, as the dissociative response to aversive stimuli is associated with diminished physiological output. The data we found did not directly support this hypothesis. We found that the depersonalization and derealization scores on the Multidimensional Inventory of Dissociation (MID) Questionnaire were in fact correlated with increased FPS to the danger signal (A+) in the final acquisition block. This would seem to suggest that more pathological dissociators startle more to the danger signal and exhibit a response that is closer to that of a classic PTSD presentation. The DS values were still very low compared to what is seen in some of the literature for both PTSD populations and control populations (e.g. Norrholm et al., 2008; Norrholm et al., 2011). A possible explanation for the observed effect could be that more severe dissociators also have more severe PTSD symptoms, and thus the startle is mediated by the hypervigilance and hyperarousal

symptomatology. In fact, the relationship between the mean scores of the MID and mean scores of the PCL-5 total symptom measure was positively correlated, supporting this conjecture that higher levels of pathological dissociation are linked with higher levels of PTSD symptomology (see Figure 5).

The fact that the EMG difference scores for our DID population were very low overall compared to what is seen in the literature is an important preliminary finding. The differences scores are calculated in acquisition by subtracting the startle amplitude of the noise-alone (NA) trials from the startle amplitudes to either A+ or B- in a certain block. Thus, a low difference score reflects a small difference between NA trials and fear-potentiated startle trials. It is possible that individuals with dissociation could be startling to NA trials and fear-potentiated startle trials on similar levels, whether these levels are high or low.

Our population's low difference scores could also reflect a DID-specific phenotype and thus be in line with our hypothesis that dissociative symptoms correlate with overall diminished physiological output. Norrholm and colleagues (2011) found DS for a clinical PTSD population at ~50μv in the first block of acquisition (ACQ1) to both CSs. Control participants during ACQ1 displayed DS of ~40μv to both CSs as well. Our data reveals DS for our DID population at <10μv during this same period.

Figure 7, which displays the startle magnitudes for acquisition, confirms that both the FPS to NA and the FPS to the two CS are lower than what is typically seen in this paradigm. We hypothesize that this may be a DID-specific feature. Dissociative individuals often endure aversive experiences by detaching themselves from the situation, feeling removed, or even sleepy. In this case, it is entirely possible that individuals with DID do not show fear potentiated startle as much as someone with PTSD, or even a control, due to their symptomology and disorder that has adapted to cope with aversive situations by dampening the autonomic response.

Thus, it appears that this population of individuals with DID may not fear-potentiate in the same way that someone with PTSD, or a control participant, would. Several studies support the pattern that individuals with PTSD will startle at a greater magnitude and potentiate their fear, whereby they continue to startle more to the A+ and less to B- as the experiment progresses (e.g. Grillon et al., 1999; Jovanovic et al., 2010). A similar pattern is seen for controls in terms of potentiation, but with a much smaller overall magnitude. The nonsignificant scores could also be a result of having a small sample size, and either way more testing within this population is necessary.

In sum, the block type, stimulus type, and block x stimulus interactions were not statistically significant for the acquisition phase of the paradigm. Figure 8, which displays the average EMG difference scores across the acquisition blocks, does reflect the pattern that we would expect in normal acquisition. It appears that the participants were learning the paradigm, i.e. discriminating between A+ and B-, which is further confirmed by the US expectancy data (see Figure 6). It is clear that our sample was underpowered, which may be a potential reason for nonsignificant interactions. In general, it does seem that our population of women with pathological dissociation may show diminished FPS compared to someone with PTSD or a control, as reflected by results from studies with a similar methodology and paradigm (Jovanovic et al., 2010; Norrholm et al., 2011).

Extinction

The first of our two central hypotheses regarding extinction was that participants would maintain an increased fear-potentiated startle to A at the start of extinction, and that these initial levels of startle would be significantly different from those towards late extinction. Control participants in this paradigm gradually exhibit a diminished fear-potentiated response during extinction in the absence of the unconditioned stimulus, thus "extinguishing" the fear (Norrholm et al., 2011). Participants with PTSD typically demonstrate a gradual decline in fear as well, yet usually exhibit a higher level of FPS in

apprehension of the formerly threatening environment. Essentially, they may extinguish the fear response, but not to the complete degree as a control participant might (Norrholm et al., 2011).

Participants with PTSD also sometimes exhibit a "high fear load" at the start of extinction, meaning that their startle levels at the start of extinction are higher than they were at the end of acquisition (e.g. Norrholm et al., 2011), where participants may view both stimuli as renewed and potential threats at the start of the second session. Since there was no reinforcing airblast during extinction, any exaggerated startle a participant may have to A over B is a result of prior conditioning, and not based on a pattern that is fortified during extinction. At the start of extinction, DID participants did not startle to A or B significantly differently. The lack of significance between a block x stimulus type interaction reflects that there was no change in discrimination throughout extinction; that is, participants did not startle more to A over B. Additionally, the power values for both these ANOVAs were extremely low, i.e. $\beta = .093\text{-}.129$.

Though the EMG startle data did not indicate differential FPS to A over B, US expectancy ratings indicate that participants were more wary of the former danger signal. During the first block of extinction, participants tended to rate A as "I don't know" and B as the safety signal. The expectancy ratings for A did not differ across extinction. This indicates that most individuals were reluctant to say that the airblast was definitely not coming after A, though they did so for B.

We did find a significant effect of block within extinction. A further pairwise comparison demonstrated a general trend for participants' levels of fear during early extinction being significantly different from their fear levels at the end of extinction, or late extinction. This is an established pattern in the literature, and serves as a confirmation that participants successfully extinguished their physiological fear responses to the danger signal (e.g. Norrholm et al., 2006).

With respect to our second hypothesis, it appears that the EMG DS and magnitude data are both lower than what is seen in the comparison literature, as in the case of acquisition (see Figure 11).

Norrholm and colleagues (2011) report DS of ~30μv in control participants for early extinction (blocks 1 and 2 in a 6-block extinction paradigm) and DS of ~50μv for participants with PTSD. Our participants display DS of ~20μv in EXT1, which is lower than both the clinical group and the control group.

A similar pattern is also seen in the EMG magnitude data (see Figure 10). While Norrholm and colleagues (2006) report levels of FPS at ~200μv to the former danger signal at the start of extinction, our participants displayed FPS levels of ~70μv to A within that same timeframe. This supports the hypothesis that a diminished FPS output could be a pattern specific to dissociation or DID.

The results from our correlational analyses give further insight into the interaction between symptomology and physiological startle. Cluster E scores of the PCL-5, which assess hypervigilance symptoms, were correlated with greater startle to A at the beginning of extinction. This reflects similarly increased fear to the danger signal that participants with PTSD usually exhibit. These results mirror those from acquisition that showed that higher levels of dissociation, as measured by derealization and depersonalization subscales, were correlated with higher levels of startle. Thus, more severe dissociative pathology might link with more severe PTSD pathology.

Additionally, the less avoidant participants, as assessed by the PCL-5 Cluster C subscale, startled more to the safety signal in extinction block 3. Inversely, more avoidant participants showed decreased FPS. Avoidant behavior is similar to detachment, which is a typical response to an aversive situation in someone with DID. This could reflect a diminished autonomic output, or that more avoidant individuals simply possess enhanced discrimination between the danger and safety stimuli.

Anecdotal Reports

The unconditioned stimulus is an aversive stimulus that is designed to elicit a fear response; as discussed, this could lead individuals with DID to dissociate as a reaction the threatening environment.

Six of our participants reported feeling dissociative to differing degrees at some point during the paradigm. They reported experiences such as feeling spaced out, daydreaming, not being able to feel their bodies, feeling as if nothing was real, and floating "in and out" of the present. According to at least one participant, the discomfort of the acquisition phase caused her to ruminate on "bad things," from which she felt compelled to try and distance herself.

Interestingly, some participants reported the acquisition phase as much more difficult to handle, while others described extinction as more anxiety provoking. While a few participants reported being on "high alert" during extinction - indicative of more PTSD-like reactions - others disclosed that they felt dissociative during extinction, as well. One participant stated that she was falling asleep at the beginning of the phase, which could be evidence of dissociation. She reported these feelings towards the beginning of the extinction phase, which makes it unlikely that she was extremely bored. She also only missed one response pad rating, lending credibility to the fact that she did not actually fall asleep. A noteworthy anecdote came from another participant who stated that, even though she learned during extinction that the airblast was no longer coming, she could not bring herself to press the "minus" button indicating that the blast would not come. She found it helpful to complete the task and found it gave her "insight" into her "trust issues."

Despite feeling either highly dissociative or highly alert during extinction, the EMG results demonstrate that the participants registered the extinction of fear. This disconnect between the cognitive experience and the physiological experience is one that has been reported in other studies (e.g. Hermans et al., 2005; Norrholm et al., 2006), is particularly interesting in the case of DID, and should be explored in future investigations.

Limitations

There are a few substantial limitations to this study. First, regarding the exclusion criteria, participants were only excluded if they had used illegal substances a month prior to their involvement

in the study. We documented all medications and dosages, as well as any medications the person may have taken that day. As noted, a majority of our participants were taking a variety of medications. The study design included such participants because substance use and extensive medications are extremely common within the target population of women with histories of childhood abuse. This is especially the case within our study pool, as the participants are recruited from McLean Hospital. These women are typically excluded from research for their lack of eligibility, and this study strove to give these women a voice and produce results that are generalizable to the true population of those suffering from DID. The larger study hopes to recruit 150 participants and control for potential interactions with the documented data of all substance use.

Another significant limitation is that we did not have a camera set up in the room where the participant completed the task. While members of the research team were on hand and ready to respond if the participant wished to end the study at any time, there was no way to monitor whether or not they were paying attention or had fallen asleep (other than their behavioral expectancy ratings). Since the data collection of these twelve pilot participants, a camera has been installed in the room so that research personnel can monitor participant engagement.

A third limitation is that we did not have a control population with which to compare data. Dr. Tanja Jovanovic and Dr. Seth Norrholm at Emory University in Atlanta, Georgia developed the paradigm we used, and they have conducted several subsequent experiments. Members of the Neurobiology of Fear Laboratory at McLean Hospital received training on their methods, and analyses were performed according to their procedure. Due to the symmetry in methods and task design, I have referred to their work several times throughout the result and discussion as a way to compare findings. The study is ongoing and control group recruitment is planned for the future including a PTSD only control group and nonpsychiatric control group. The outcomes of the larger study will be more

powerful with comparison to these control population. Due to these limitations, these initial findings need to be replicated in further studies.

Significance and Future Directions

Continual research with the population of individuals who have DID, and other dissociative disorders, are crucial for a number of other reasons. The diagnosis of DID has been a controversial one for many years, for explanations ranging from improper or illogical diagnostic criteria (Gharaibeh, 2009), to the belief held by some professionals that people with DID are faking their symptoms (Tartakovsky, 2016), to concern over the provocation of "false memories" during treatment (Boysen, 2011). A majority of the research done on DID occurred during the 1980s and 1990s, and the controversy is still unresolved (Boysen, 2011). This concern is reflected in one of the main goals of this larger study. We aim to establish physiological and neural correlates of DID to lend further legitimacy to the disorder and dispel the various myths within the scientific community that discount the experiences of these individuals.

As discussed, many individuals with DID also have symptoms of PTSD due to the trauma and maltreatment that they have faced (Tartakovsky, 2016). Studies like this one can help establish how DID is fundamentally and physiologically different from PTSD beyond a symptomatic level. Future research should consider comparing populations of individuals with DID and with PTSD and control for their scores on measures of PTSD symptoms and of dissociation, such as the PCL-5 and the MID. This research is crucial for directing treatments and practitioners towards providing care that is tailored towards the specific symptoms in DID, beyond generalizing the diagnosis as a PTSD-like response to trauma. Establishing physiological correlates and biomarkers can also guide research towards medications that may be able to target the specific dysfunctions of individuals with DID, as well.

The results and implications of this study hope to contribute to the growing body of literature surrounding DID. The present study represents a crucial first step towards comparing and contrasting

DID with PTSD, a well-studied trauma-spectrum disorder, through the fear-potentiated startle paradigm. This is done in the hope that studies such as these can help both the professional and patient communities further understand the etiology of the disorder and inform investigation into the neural and physiological correlates of trauma-spectrum disorders.

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