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Psychological and Psychobiological Responses to Immediate Early Intervention in the Emergency Department: Case Report of One-Session Exposure Therapy for the Prevention of PTSD

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Research suggests that exposure therapy provided in the hours immediately following trauma exposure may prevent posttraumatic stress disorder (PTSD). This case report presents data on an at-risk-for-PTSD participant involved in a motor vehicle crash that caused her severe distress. She received one session of exposure therapy in the

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emergency department (ED) as part of an ongoing randomized controlled study examining the optimal dose of exposure therapy in the immediate aftermath of trauma. PTSD and depression measures were collected at pretreatment assessment and at 1- and 3-month follow-up. Potential PTSD biomarkers were also examined. Psychophysiological reactions were measured using skin conductance data during the exposure therapy session and the follow-up assessments. A fear-potentiated startle paradigm and a functional MRI (fMRI) behavioral inhibition task were used at follow-up. The participant demonstrated subjective and psychophysiological extinction from pre- to postimaginal exposure. At follow-up, she did not meet Diagnostic and Statistical Manual of Mental Disorders-IV (American Psychiatric Association, 2000) criteria for PTSD or demonstrate hyperarousal to trauma reminders, and showed robust fear extinction and the ability to inhibit responses in an fMRI behavioral inhibition task. In line with previous early intervention for the prevention of PTSD studies, this case report supports the need for ongoing empirical research investigating the possibility that one session of exposure therapy in the ED may attenuate risk for PTSD. Furthermore, the current findings demonstrate psychophysiological extinction serving as a prognostic indicator of treatment response for PTSD early intervention to be an avenue to explore in future systematic research.

Public Significance Statement

This case study provides further support for ongoing investigation into the effectiveness of one session of exposure therapy in the immediate aftermath of trauma for the prevention of PTSD. If shown to be an effective treatment, the efficiency of this approach and the demonstrated viability of its implementation by mental health providers suggest that it could be feasibly disseminated in order to prevent the development of PTSD, a disorder with a tremendous psychological and societal impact.

Keywords: early intervention, memory consolidation, exposure therapy, PTSD psychobiology

Conditioned fear reactions typically occur shortly after a traumatic stressor and naturally decline over time (Breslau et al., 1998; Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). A failure of natural extinction of conditioned fear may manifest as posttraumatic stress disorder (PTSD). Biological factors have been shown to play an important role in PTSD development and maintenance (Milad et al., 2009; Pitman et al., 2012; Skelton, Ressler, Norrholm, Jovanovic, & Bradley-Davino, 2012). With continued research, biomarkers may further the mechanistic understanding of PTSD that could lead to the development of targeted PTSD prevention interventions for those at risk.

Individuals with PTSD exhibit stronger psychophysiological responses to trauma reminders than non-PTSD trauma survivors (Orr, Metzger, & Pitman, 2002), as well as failure to extinguish hyperarousal in a fear-potentiated startle (FPS) paradigm and to distinguish between safety and danger cues (Jovanovic et al., 2010). Reduced activation of the rostral anterior cingulate cortex (rACC) is coincident with impaired behavioral inhibition in traumatized individuals (Jovanovic et al., 2013). In connection with psychological symptoms, these characteristics may be important potential biomarkers of treatment outcome (Rothbaum, Price, et al., 2014) and risk for developing PTSD following trauma exposure.

Both animal studies (Myers, Ressler, & Davis, 2006) and translational human studies (Norrholm et al., 2008) suggest that immediate extinction training (10 min to 1 hr after fear conditioning) results in more sustained fear reduction than later extinction training (24–72 hr after fear conditioning). The literature surrounding PTSD and fear extinction models suggests exposure-based interventions implemented soon after trauma may interrupt the consolidation of the fear memory and thus result in more

effective prevention of PTSD (Bisson et al., 2010; Rothbaum et al., 2008, 2012). With PTSD directly linked to a causal event, there is the possibility of intervening proximally and potentially preventing PTSD development.

There has been examination of an early intervention—a modified version of the empirically validated Prolonged Exposure (PE) procedure (Foa, Hembree, & Rothbaum, 2007)—implemented in an emergency department (ED) in the hours immediately following trauma exposure. Table 1 provides a breakdown of treatment components. In a pilot study, patients who received one session of the early intervention demonstrated decreased levels of depression and overall symptom severity at 1-week follow-up compared with those who received assessment only (Rothbaum et al., 2008). In a randomized trial, patients who received three sessions of the early intervention reported significantly decreased PTSD and depression at 1 and 3 months posttrauma, and were half as likely to meet criteria for PTSD 3 months posttrauma compared with those in the assessment-only control group (Rothbaum et al., 2012). Further, this early intervention was particularly effective with sexual assault survivors (Price, Kearns, Houry, & Rothbaum, 2014) and seemed to mitigate genetic risk for PTSD (Rothbaum, Kearns, et al., 2014). These results are particularly important in light of the results with critical incident stress debriefing indicating that early intervention could potentially be harmful (Bisson et al., 2010).

A randomized controlled study is underway to examine the optimal dose of this early intervention via the comparison of three weekly exposure therapy sessions commencing in the ED to one exposure therapy session in the ED, and assessment only. Determining whether one session (with homework practice) is as effective in attenuating risk for PTSD as three sessions is critical, as such an intervention would be more easily disseminated. In order to explore whether autonomic nervous system activity is predictive of response to treatment for PTSD prevention, this study is prospectively monitoring fear reactions using skin conductance (SC) data collected during the ED exposure therapy session and the follow-up assessments, and assessing psychophysiological hyperarousal and impaired fear extinction at follow-up using an FPS paradigm (Jovanovic et al., 2010). Additionally, this study is obtaining functional MRI (fMRI) data at follow-up to explore brain activation associated with PTSD development. The current case report documents the treatment and follow-up response of one participant enrolled in this ongoing study. With the randomized controlled study still in progress, this case report is necessary to describe the innovative assessment and one-session exposure therapy techniques being used in the ED within hours of trauma exposure in the context of an enrolled participant, and to

Table 1
Exposure-Based Early Intervention Description

Early intervention	Task
Session 1 – 1 hr	Introduce treatment components and rationale (2 min)
	Conduct imaginal exposure (30–45 min)
	Process the imaginal exposure and identify positive self-statements to reframe unhelpful cognitions resulting from the trauma (10–15 min)
	Identify behavioral exposure(s) for the coming week (5 min)
	Provide psychoeducation on normal reactions to trauma and identify self-care activities for the coming week (3 min)
	Introduce and practice breathing retraining exercise (5 min)
	Review assigned homework (2 min)
Assigned homework[a]	Listen to audio recording of imaginal exposure daily
	Practice the use of positive self-statements
	Practice the behavioral exposure(s)
	Engage in self-care activities
	Practice breathing retraining exercise

Note. Component lengths vary based on the individual needs of each participant. Total session length is recommended to not exceed 1 hr.
[a] The therapist calls the participant to assess homework compliance 1 week after the last treatment session.

support the need for ongoing empirical research to determine treatment efficacy. This participant was selected for the case report because she was the first participant randomized to the one-session condition with all psychobiological and psychological assessments completed.

Method

Participant

The participant is a 23-year-old single Hispanic female who was brought to a Level 1 trauma center ED via ambulance following a motor vehicle crash (MVC) in which she was the driver of a car that was totaled by a semitruck. She reported a brief loss of consciousness immediately after her car was struck, and numbing and disbelief upon consciousness and resulting overall body pain and bruising.

Screening and Enrollment

The participant was recruited from the ED. After a brief study overview, with the patient's assent, the assessor conducted the study preliminary screening, verifying that the participant met inclusion criteria (Age 18–65, endorsement of Diagnostic and Statistical Manual of Mental Disorders-IV [DSM-IV; American Psychiatric Association, 2000] Criterion A trauma experienced within the past 24 hr) and did not endorse any exclusion criteria (history of mania, schizophrenia, or other psychoses; prominent suicidality; substance dependence in the past month; intoxication; altered mental status). Informed consent was obtained after successful completion of the preliminary screening. The participant was randomized to the one-session exposure therapy condition. Compensation included $50.00 for each assessment and the treatment session. Study procedures were approved by the study site's institutional review board and research oversight committee.

Psychological Assessments

The pretreatment assessment included the following measures.

Predicting PTSD Questionnaire (PPQ; Rothbaum, 2014). The PPQ is a five-item screen of the following PTSD risk factors: trauma history (yes–no), current trauma subjective severity (3 or higher on a 0 [*not at all*] to 5 [*near death*] scale), current trauma peritraumatic dissociation (2 or higher on a 0 [*not at all*] to 4 [*completely*] scale), childhood trauma exposure (yes–no), and family history of psychopathology (yes–no). To date, there is preliminary evidence that the PPQ is useful to screen for PTSD risk (Rothbaum, 2014). In this study, participants are required to endorse a minimum of three of the five PPQ PTSD risk factors to be eligible for the intervention.

Posttraumatic Stress Diagnostic Scale (PDS; Foa, Cashman, Jaycox, & Perry, 1997). The PDS screens for prior traumatic events and consists of a list of 17 items assessing *DSM–IV* (American Psychiatric Association, 2000) PTSD diagnosis and severity during the past 2 weeks. Symptoms are rated on a 4-point scale from 0 (*not at all*) to 3 (*very much*). The PDS has high internal consistency (α = .92) and test–retest reliability for total score (r = .83; Foa, Riggs, Dancu, & Rothbaum, 1993). In this study, the PDS was used to assess lifetime PTSD prior to this presenting traumatic event.

Standardized Trauma Interview (STI; Foa & Rothbaum, 1998). The STI is a clinician-administered interview that gathers information regarding demographic variables and characteristics of the index trauma such as injury and life threat.

Mini-International Neuropsychiatric Interview Version 5.0.0 (MINI; Sheehan et al., 1997). The MINI is a structured interview designed to assess Axis I diagnoses based on *DSM–IV* (American Psychiatric Association, 2000) criteria. The MINI has strong interrater reliability, with kappa values ranging from 0.88 to 1.0, and good test–retest reliability for the diagnoses, with kappa coefficients between 0.76 and 0.93 (Lecrubier et al., 1997).

Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). The BDI is a 21-item self-report inventory measuring depression severity. Each item consists of statements scored from 0 to 3, with increasing scores indicating greater severity of depression. The BDI has a split-half reliability of .93 (Beck et al., 1961) and correlates strongly with clinical ratings of depression (r = .55 to .96; Beck, Steer, & Carbin, 1988).

The 1- and 3-month posttreatment assessments incorporated the pretreatment BDI measure as well as the PTSD Symptom Scale (PSS;

Foa et al., 1993). The PSS is a semistructured interview assessing PTSD diagnosis and severity and was used in this study to obtain a diagnosis and severity of PTSD in response to the index trauma. The measure consists of 17 items corresponding to the *DSM–IV* (American Psychiatric Association, 2000) PTSD symptoms. Items are rated on a scale from 0 to 3 for combined frequency and severity in the past 2 weeks (0 = *not at all*, 3 = *5 or more times per week/very much*). The PSS has good convergent validity with other PTSD interview measures (Foa & Tolin, 2000).

Psychobiological Assessments

SC data were acquired using the eSense system connected to an iPad. Two 5-mm Ag/AgCl electrodes filled with isotonic paste were attached to middle phalanges of the second and fourth fingers of the nondominant hand. Baseline data were collected for 2 min prior to the start of the ED exposure session and for the duration of the imaginal exposure portion of the intervention. At the follow-ups, baseline data were collected for 2 min prior to the start of the assessment and throughout the administration of the PSS (Jovanovic, Rauch, Rothbaum, & Rothbaum, 2017).

At the 1-month follow-up, fMRI was used to assess performance on a go/no-go task (Jovanovic et al., 2013), wherein participants were asked to press a button whenever an "X" or "O" appeared on the monitor, but not if a red square was also presented behind the letter. This task was designed to assess behavioral inhibition, the ability to appropriately inhibit a behavioral response to a stimulus. Behavioral inhibition has been shown to be impaired in individuals with chronic PTSD, and relies on shared neurobiology with PTSD-related difficulties in fear inhibition (Jovanovic et al., 2013). This assessment was conducted during the 1-month follow-up visit, the earliest procedurally practical time point, in order to facilitate observation of treatment effects in the greatest proportion of participants before symptoms began to recover in moderately affected participants.

At the 3-month follow-up, a time when chronic PTSD is diagnosable, the participant underwent the FPS paradigm as described in Glover et al. (2011) and Norrholm et al. (2011). Briefly, startle response data were acquired using electromyography measuring the eyeblink component of the acoustic startle response during fear acquisition and extinction protocols. The FPS consisted of an initial habituation phase in which presentation of conditioned stimuli (CS) without any reinforcement occurred (Glover et al., 2011; Norrholm et al., 2011). The fear acquisition phase consisted of three blocks with four trials of each type of CS (reinforced conditioned stimulus, CS+, danger signal; nonreinforced conditioned stimulus, CS–, safety signal; noise probe alone, NA). Both CSs were colored shapes (i.e., blue square, purple triangle) presented on a computer monitor for 6 s each. The unconditioned stimulus (US; aversive stimulus) was a 250-ms air blast of 140-psi intensity to the larynx that produces robust FPS (Glover et al., 2011; Norrholm et al., 2011). The air blast was delivered from a compressed air tank via a polyethylene tub and controlled by a solenoid switch. Ten minutes after fear acquisition, an extinction session was performed wherein neither the CS+ (danger signal) nor the CS– (safety signal) was paired with the US (Norrholm et al., 2011).

Treatment

The exposure intervention (see Table 1) was initiated immediately following assessment procedures and collection of a 2-min baseline SC with the iPad. The therapist's explanation of treatment components and procedures was adapted from the PE treatment therapist manual (Foa et al., 2007). The treatment session began with the therapist presenting the treatment rationale emphasizing that the purpose of the intervention was to help the participant feel like herself again with the goal to help make thinking and talking about the memory of the event easier. The therapist explained that the two factors that help people to move forward from a traumatic event are emotionally processing the event by talking about the event in a therapeutic manner and not avoiding safe reminders of the event.

The therapist then discussed imaginal exposure, or revisiting the trauma memory, explaining that the participant would be asked to visualize and emotionally connect with the traumatic event while recounting the experience, including all thoughts, emotions, and sensations, aloud in present tense for 30 to 45 min. The therapist noted that the participant would be free to recount the mem-

ory in her own words and at her own pace, with the therapist occasionally asking questions for clarification or for more detail. The therapist introduced the Subjective Units of Distress Scale (SUDS) and explained that a rating of current distress from 0 to 100 would be elicited from the participant at different time points surrounding the imaginal exposure. Finally, the participant was told that after the imaginal exposure, she and the therapist would discuss her experience of revisiting the trauma memory and her feelings and thoughts about the trauma and its meaning in her life to facilitate emotional processing. The processing included the participant being encouraged to identify any maladaptive thoughts about the traumatic experience and to replace them with more adaptive thoughts. The recounting of the trauma was audio recorded, and the participant was asked to listen to the recording daily.

After the imaginal exposure and processing, the therapist reviewed the rationale for behavioral exposures asking the participant to consider possible trauma reminders that are realistically safe that she might have a tendency to avoid upon release from the hospital such as driving or driving in the area where the MVC occurred. The therapist emphasized that in order for a behavioral exposure to be therapeutic, the participant must confront the situations repeatedly and for an extended amount of time until her distress decreased.

In conjunction with the discussion of trauma reminders, the therapist discussed common reactions to trauma with the participant. In order to further support the healing process, the participant was asked to identify self-care activities to engage in over the coming hours, days, and weeks following the MVC. The therapist ended the session by teaching the participant a brief breathing retraining exercise to use at times when her anxiety increased but the use of exposure was not appropriate, for example, when trying to go to sleep at night.

Results

Pretreatment Assessment

The participant began the pretreatment assessment approximately 3 hr after the MVC. On the PPQ, the participant reported prior trauma including a nonsexual assault by a stranger and by her boyfriend, a rating of 5 (*near death*) for her current trauma severity, and a rating of 2 (*somewhat*) for her current trauma peritraumatic dissociation. On the STI, she endorsed the highest severity response to feeling helpless, horrified, terrified, and out of control during the MVC. The participant did not meet *DSM–IV* (American Psychiatric Association, 2000) criteria for lifetime PTSD on the PDS or current MDD on the MINI. She reported current minimal depressive symptoms (BDI-II = 7).

Treatment Session

The participant completed three repetitions of her traumatic event narrative for a total of 44 min. The session data demonstrated a decrease in the participant's subjective distress from pre- to postexposure (pre-SUDS = 5, peak-SUDS = 40, post-SUDS = 0). The SC increased during each imaginal exposure compared with baseline levels, but the peak in SC decreased over the course of each imaginal exposure and was lower than baseline at the end of treatment (Table 2A; Figure 1A).

Following the recounting, the participant and therapist discussed the emotional material that emerged during the narrative. The participant identified effective coping strategies that she used during the trauma, as she was able to transition her fears of dying to thoughts of being "hopeful" and "grateful" while she was riding in the ambulance. The participant was able to generate positive self-statements related to the fact that she survived the situation and believed that good things would come out of the accident, such as her being more responsible and caring of others. Additionally, although she felt anger toward the truck driver, she made the decision to focus on the kindness and helpfulness of the bystanders and paramedics, a part of the trauma that became more evident to her as she was going through the imaginal exposure.

The therapist and participant completed a list of behavioral exposures to be completed over the coming weeks. In response to the participant feeling hesitant to resume her normal driving routine, a hierarchy was constructed in which the participant agreed to first complete less anxiety provoking exposures such as sitting in the driver's seat with the car turned off and driving around her neighborhood side streets. The participant identified doing yoga and spending time with her family as self-care activities.

Table 2
Summary of SC During Each IE in the ED (A), at 1-Month Follow-Up During the PSS Assessment (B), and at 3-Month Follow-Up During the PSS Assessment (C)

Time point	Average SC (μS)	Δ from baseline average	Δ from first to last minute
(A) ED			
Baseline	2.84	—	↓ .34
First IE	3.76	↑ .92	↓ .21
Second IE	3.74	↑ .90	↓ 1.92
Third IE	2.48	↓ .36	↓ .64
(B) 1-month follow-up			
Baseline	1.57	—	↑ .01
PSS	1.50	↓ .07	↑ .03
(C) 3-month follow-up			
Baseline	1.61	—	↓ .22
PSS	1.32	↓ .29	↑ .34

Note. Arrows denote increases (↑) and decreases (↓) in SC over time. SC = skin conductance; IE = imaginal exposure; ED = emergency department; PSS = PTSD Symptom Scale; μS = microSiemens.

Follow-Up Assessments

In assessing responses to the MVC, the participant reported mild PTSD symptoms (PSS = 11) at the 1-month follow-up, but did not meet criteria for PTSD. The SC data indicated no increase in psychophysiological arousal in response to the PSS (Table 2B; Figure 1B). The participant reported minimal depressive symptoms (BDI = 5) that were not significantly different from her pretreatment report. The participant showed significant prefrontal inhibition response to the fMRI go/no-go task that was associated with activation of the rACC (Figure 2A).

At the 3-month follow-up, the participant continued to report mild PTSD symptoms (PSS = 8) and not meet criteria for PTSD, minimal depressive symptoms (BDI = 9) that were not significantly different from her pretreatment and 1-month follow-up reports, and did not show an increase in SC in response to trauma reminders during the PSS (Table 2C; Figure 1C). The participant showed significant discrimination between safety and danger cues, and fear extinction during the FPS paradigm (Figure 2B).

Discussion

This is the first report of a single session of an early intervention delivered in the ED within hours of the traumatic event. The case report outcomes are important, as they indicate a potentially important step in the development of continued randomized controlled studies investigating the efficacy of early intervention for the prevention of PTSD. This case report demonstrated that a participant at risk for developing PTSD involved in a severe MVC only hours before the start of treatment experienced subjective and psychophysiological extinction within a single exposure therapy session. Further, after receiving only one session of exposure in the ED followed by homework, the participant did not display an increase from postextinction in psychophysiological arousal in response to trauma reminders, a PTSD diagnosis, or an increase in depressive symptoms at 1- and 3-month follow-up. Lastly, the participant showed safety and danger cue discrimination, as well as fear extinction and inhibition-related brain activation at follow-up on objective neurobiological measures (FPS and fMRI). This study was limited in its ability to assess treatment-related change in such neurobiological measures because it did not assess pretrauma functioning. These findings do, however, indicate an adaptive pattern of function associated with low PTSD symptoms (e.g., Jovanovic et al., 2013).

Thus, the participant who experienced a *DSM–IV* (American Psychiatric Association,

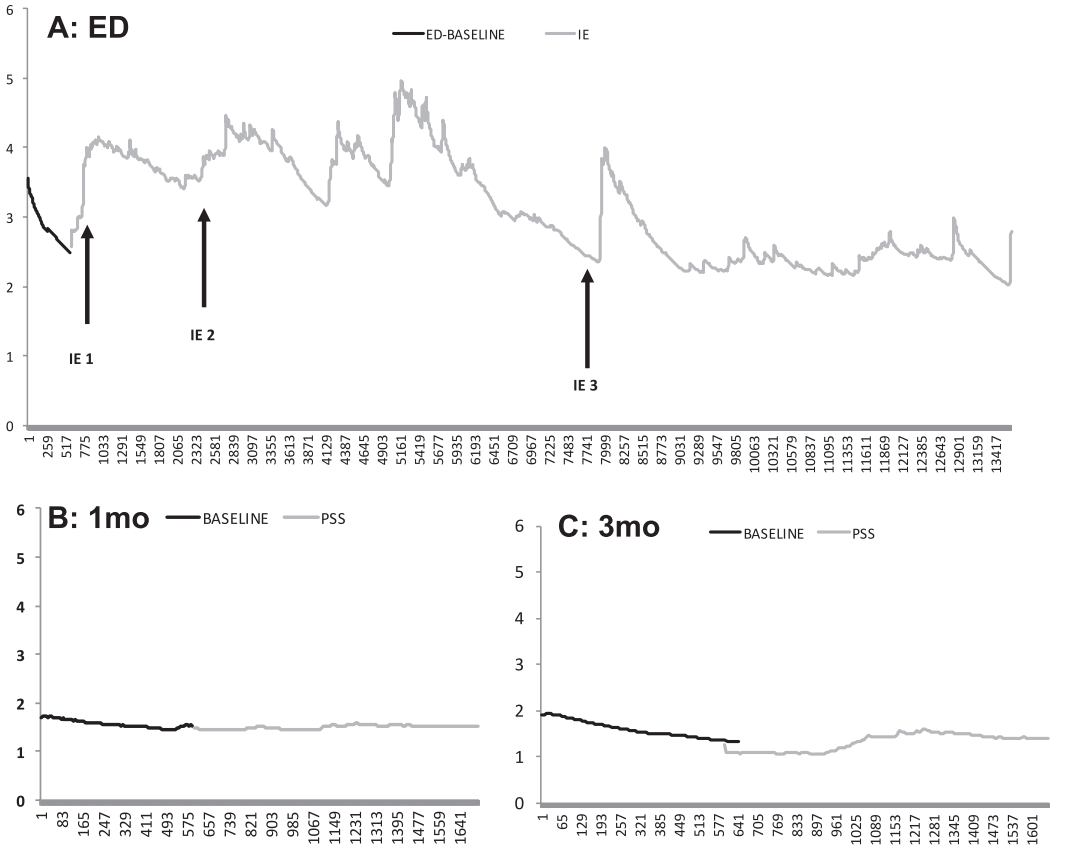

Figure 1. SC response (μS) at baseline and during each IE in the ED (A), and at 1-month (B) and 3-month (C) follow-up during the PSS assessment. Each IE repetition is denoted by arrows. SC = skin conductance; μS = microSiemens; IE = imaginal exposure; ED = emergency department; PSS = PTSD Symptom Scale.

2000) Criterion A trauma and reported several empirically supported PTSD risk factors did not display symptom worsening from pretreatment to 3-month follow-up or develop PTSD. The currently ongoing randomized controlled study from which this case report was drawn will help determine whether one session exposure therapy potentially prevents the development of chronic PTSD. Additionally, the decrease in autonomic nervous system activity throughout the exposure therapy session may be an important biomarker in predicting whether trauma-exposed individuals are more or less likely to respond to preventative treatment of PTSD. Notably, this was gathered on an iPad with free software. Viewed within the context of the provision of early intervention, the absence of both an increase in SC in response to trauma reminders and reduced activation of the rACC, and the presence of discrimination between safety and danger cues and extinction of conditioned fear at the posttreatment follow-ups, support further examination into whether immediate exposure therapy affects possible mechanisms involved in the development of PTSD. With preliminary data supporting the efficacy of three sessions of exposure therapy commencing in the ED in attenuating risk for PTSD, the current case report supports the possibility that the current ongoing randomized controlled study will demonstrate that one session of exposure in the ED followed by homework may also be effective and, at the least, not harmful. If this is the case, there is the potential for mental health providers to have the treatment tools needed to help patients with recent trauma exposure discuss their

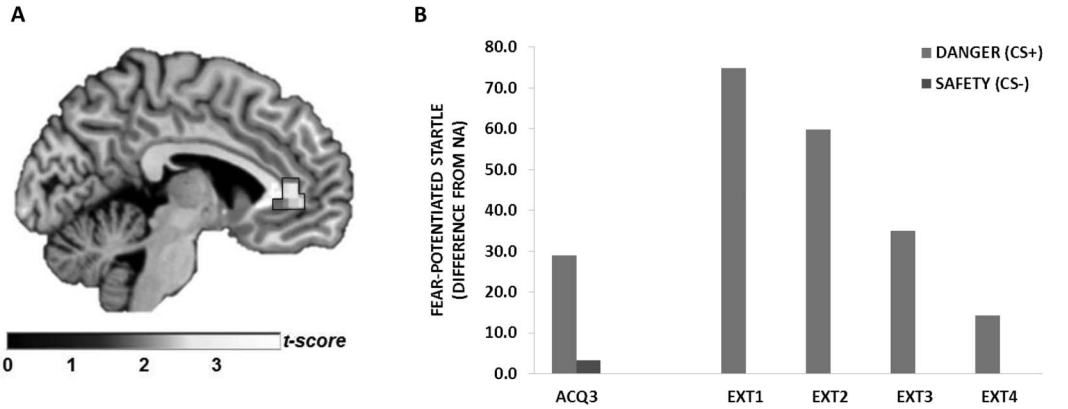

Figure 2. (A) Inhibition responses at 1-month follow-up. Significant activation (outlined in black) within an anterior cingulate cortex (ACC) region of interest is overlaid on a sagittal slice in Montreal Neurological Institute space, for the Stop > Go contrast (x, y, z = −6, 40, 2, $z = 2.57$, k = 29, $p < .05$, corrected). The ACC region of interest (ROI) was created using a bilateral mask based on the cytoarchitectonic map from the Automated Anatomical Labeling toolbox (Tzourio-Mazoyer et al., 2002). Correction for multiple comparisons used a combined height-extent threshold calculated using Alphasim Monte Carlo simulation, with 1,000 iterations and a cluster-forming threshold of $p < .05$. (B) Fear conditioning and extinction at 3-month follow-up. Significant safety/danger cue discrimination and fear extinction at three months posttrauma. NA = noise alone; CS = conditioned stimulus; ACQ = acquisition; EXT = extinction.

experience in an effective and efficient manner such that common responses to trauma (i.e., conditioned fear reactions) are less likely to develop into PTSD.

One limitation of this case report is that there is no comparison group. Thus, the case report alone cannot determine whether the one session intervention offers an advantage over normal recovery. Completion of the current randomized controlled study is needed in order to draw any conclusions about the effectiveness of a one session exposure early intervention. However, our recent data suggest that individuals who were randomly assigned to assessment-only reported significantly more PTSD symptom severity at 1-month posttrauma than those who received the intervention (Rothbaum et al., 2012). Additionally, because it is not valid to measure PTSD to a trauma that occurred only hours prior, this case report does not follow a typical pre–post design in which patients start out symptomatic and then improve. Thus, in a prevention design, the lack of an increase in symptoms is evidence of a good response.

Overall, there could be a tremendous effect on public health through greater reach and easier dissemination if one session proves to be an effective dose for the delivery of exposure therapy in the immediate aftermath of trauma for the prevention of PTSD (Kearns, Ressler, Zatzick, & Rothbaum, 2012).

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