Title
Evidence for acute central sensitization to prolonged experimental pain in posttraumatic stress disorder

Permalink
https://escholarship.org/uc/item/64q4x8qf

Journal
Pain Medicine (United States), 15(5)

ISSN
1526-2375

Authors
Moeller-Bertram, T
Strigo, IA
Simmons, AN
et al.

Publication Date
2014

DOI
10.1111/pme.12424

Peer reviewed
Evidence for Acute Central Sensitization to Prolonged Experimental Pain in Posttraumatic Stress Disorder

Tobias Moeller-Bertram, MD, PhD, MS (Clinical Research),*†‡ Irina A. Strigo, PhD,*†§ Alan N. Simmons, PhD,*†‡ Jan M. Schilling, MD,‡ Piyush Patel, MD,†‡ and Dewleen G. Baker, MD*†§

*Center of Excellence for Stress and Mental Health; †VA San Diego Healthcare System; Departments of §Anesthesiology and Psychiatry, University of California San Diego, San Diego, California, USA

Reprint requests to: Tobias Moeller-Bertram, MD, PhD, MAS (Clinical Research), Desert Clinic Pain and Wellness, 36101 Bob Hope Drive, Rancho Mirage, CA 92270, USA. Tel: (760) 321 1315; Fax: (760) 321 1094; E-mail: moellerbertram@yahoo.com.

Financial Support: Funding for this project was provided by the Foundation for Anesthesia Education (TM-B) and Research and the Center of Excellence for Stress and Mental Health, San Diego (TM-B, IAS, ANS, DGB).

Disclosure: No disclosure or conflicts of interest for either author.

Suggested Reviewers:
1. Alexander C. McFarlane, MD, Centre of Military and Veterans Health, University of Adelaide, 122 Frome Street, Adelaide, South Australia 5000, Australia. Tel: +61 303 5200; fax: +61 303 5368; e-mail: alexander.mcfarlane@adelaide.edu.au
2. Christian Schmahl, MD, Department of Psychosomatic Medicine and Psychotherapy, Central Institute of Mental Health, Mannheim, Germany. E-mail: christian.schmahl@zi-mannheim.de
3. Sean Mackey, MD, PhD, Department of Anesthesia, Division of Pain Medicine, Stanford Neuroscience and Pain Laboratory, Stanford University, Palo Alto, California. E-mail: smackey@pain.stanford.edu

4. James A.D. Otis, MD,: Department of Neurology, Boston University School of Medicine, 75 E. Newton St, Boston, Massachusetts 02118. E-mail: jotis@bu.edu

Summary Statement: The PTSD cohort showed higher pain ratings after 15 minutes postintramuscular capsaicin and increased temporal summation consistent with manifestation of acute central sensitization.

Abstract

Background. Post-traumatic stress disorder (PTSD) and pain have a well-documented high comorbidity; however, the underlying mechanisms of this comorbidity are currently poorly understood. The aim of this psychophysical study was to investigate the behavioral response to a prolonged suprathreshold pain stimulus in subjects with combat-related PTSD and combat controls (CC) for clinical evidence of central sensitization.

Methods. Ten male subjects with current PTSD related to combat and 11 CC male subjects underwent baseline quantitative sensory testing (QST), temporal pain summation, and psychological profiling followed by an intramuscular injection of capsaicin into the quadriceps muscle.

Results. There was no significant between-group difference for the initial maximal pain response or an initial pain reduction for the first 15 minutes postinjection on QST or pain ratings. However, we observed significantly higher scores in the PTSD group for the second 15 minutes postinjection on both pain intensity and pain unpleasantness ratings. Assessment of temporal summation to repetitive pressure stimuli showed significantly higher subjective pain in the PTSD group.

Conclusion. These findings are consistent with a significantly higher degree of acute central sensitization.
sensitization in individuals with PTSD. Increased acute central sensitization may underlie increased vulnerability for developing pain-related conditions following combat trauma.

Key Words. PTSD; Experimental Pain; Central Sensitization; Quantitative Sensory Testing; Capsaicin

Introduction

Clinical Phenomenon of High Comorbidity of Pain and Post-Traumatic Stress Disorder

Post-traumatic stress disorder (PTSD) is a debilitating illness characterized by symptoms of reexperiencing, avoidance, emotional numbing, and hyperarousal resulting from an emotionally traumatic event with actual or perceived threat [1]. It is the fifth most common psychiatric disorder in the United States with a lifetime incidence around 7% [2,3], but the prevalence can range from 20% to 80% in high-risk groups [4–11]. PTSD profoundly impacts the individual’s life and health, and has been associated with a number of adverse somatic outcomes [12] including chronic pain [13–17]. This association does not necessarily depend on the occurrence of pain or injury during the initial trauma event [18]; nevertheless, there is a significantly higher incidence of pain symptoms and pain-related conditions in subjects with PTSD [14,15,19–21]. In Vietnam veterans with PTSD, the prevalence of chronic pain has been reported to be as high as 80% [19]. Another study found a physician diagnosed pain condition in 66% of the veterans with PTSD [22]. Although specific pain conditions such as headaches including migraines [23] and musculoskeletal pain [24] have been described, the overall picture shows a rather generalized effect of PTSD on increased bodily pain suggesting a centralized dysfunction [25].

Based on the frequent association of PTSD and pain [13,26], cognitive–behavioral theories were proposed to explain this phenomenon [27]. Pain catastrophizing, anxiety sensitivity, depression [28], as well as negative childhood events [29] have been proposed as mediating links between PTSD and pain. The two nonexclusive main theories are the mutual maintenance [15] model proposed by Sharp and Harvey, and the extension of this model by Asmundson et al. [14], emphasizing a shared vulnerability. In basic agreement with these theories, a generalized sensitization of systems and circuits referred to as neurosensitization has been proposed [30]. Central sensitization (CS) represents plasticity of the somatosensory system and can be defined as a heightened response of neurons and circuits in nociceptive pathways as a result of increased excitability and synaptic efficacy or reduced inhibitory modulation [31–33]. As hyperarousal, one of the defining findings in PTSD, represents a heightened response to an incoming somatosensory stimulus, one can hypothesize that this observed augmentation of central processing also applies to noxious stimuli in PTSD.

Such an involvement has been theorized by some authors, but rigorous experimental evidence so far has been lacking [12,34,35].

The aim of this study was to test the hypothesis that PTSD subjects have sensitized central pain pathways. We applied a prolonged experimental pain stimulus above detection threshold (i.e., suprathreshold) capable of unmasking CS. We tested pain-free subjects with PTSD and controls and measured clinical correlates of CS, namely hyperalgesia (higher pain ratings) and increased temporal summation of pain (increased response to repetitive identical stimuli).

Methods

The local Institutional Review Board (University of California San Diego, San Diego, CA and Research and Development committee, San Diego VA Healthcare Services) approved all study procedures, and all participants provided written, informed consent. All subjects were tested in the Clinical Research Unit (CRU) at the VA San Diego Healthcare System (VASDHS) in a quiet room at ambient temperature after they were familiarized with the testing environment. The data presented in this manuscript were gathered as part of a larger study. A subset of these subjects also performed baseline ASL scanning and provided biological samples to be reported elsewhere.

The study assessments were divided into two separate experimental days. On experimental day 1, each subject underwent a structured clinical interview conducted by two independent psychologists. They filled out several paper-and-pencil questionnaires and experienced baseline QST testing. Diagnosis of PTSD was determined by Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) and the Clinician Administered PTSD Scale (CAPS), which can be used as a continuous measure reflecting severity of PTSD. The clinical interview, the SCID, and a urine toxicology screen were used to determine history and current substance/alcohol use and dependence. History of substance/alcohol abuse and history of depression preceding the PTSD diagnosis were exclusion criteria. All subjects received a comprehensive medical exam.

On experimental day 2 which occurred within 30 days from experimental day 1, all subjects received an intra-muscular capsaicin injection (see below).

Subjects

Subject Selection

Ten male PTSD and 11 male Combat Control (CC) Operation Enduring Freedom (OEF) and Operation Iraqi Freedom (OIF) veterans were enrolled from the greater San Diego area. Inclusion criteria for PTSD group were as follows: 1) history of deployment to a combat zone; 2) diagnosis of PTSD as determined by SCID and the CAPS; 3) within normal range physical examination; 4) no chronic...
or acute pain complaint; and 5) no medication for at least 14 days prior to CRU admission (including nonsteroidal anti-inflammatory medications and over-the-counter pain medications). Exclusion criteria for the PTSD group with the were as follows: 1) younger than age 18 or older than 65 years of age; 2) inability to give informed consent; 3) history of substance abuse within 6 months of study participation; 4) significant head trauma, as judged by loss of consciousness or a postconcussive syndrome by history; and 5) other current Axis I comorbid disorder, except for dysthymia or major depression, if secondary to (chronologically after) PTSD diagnosis. Inclusion/exclusion criteria for CC group: CCs were closely matched for age, race, and health habits to the PTSD group. Inclusion and exclusion criteria for the control subjects are the same as those applied to combat veterans with PTSD, with the exception of inclusion criteria 2 (diagnosis of PTSD).

**Questionnaires**

**CAPS**

The CAPS is a widely used structured interview providing a categorical diagnosis, as well as a measure of the severity of PTSD symptoms as defined by DSM-IV. The CAPS has good psychometric properties across a wide variety of clinical populations and research settings [36]. Interrater reliability is high [37], and the kappa for a categorical PTSD diagnosis is often 1.0 (i.e., 100% agreement [38]).

**Childhood Trauma Questionnaire**

The Childhood Trauma Questionnaire (CTQ) is a 28-item self-report inventory that provides brief, reliable, and valid screening for histories of abuse and neglect. It evaluates five different types of childhood traumatic experiences, including emotional abuse, physical abuse, sexual abuse, emotional neglect, and physical neglect [39]. Rating is performed using a Likert scale ranging from 1 (never) to 5 (very often) with final scores ranging from 5 to 25.

**Quality of Life Inventory (SF-36)**

The SF-36 is a widely used 36-item self-report measure assessing physical and mental health as well as general well-being [40]. It is divided into eight aggregate scales including physical functioning, role-physical, bodily pain, general health, vitality, social functioning, role-emotional, and mental health.

**Pain Anxiety Symptom Scale**

The Pain Anxiety Symptom Scale is a 40-item self-report measure of pain anxiety symptoms associated with pain [41]. It is designed to assess the following four components of pain-related anxiety: cognitive, fear, escape/avoidance, and physiological. It is scored using a 6-point Likert scale ranging from never (0) to always (5).

**Pain Catastrophizing Scale**

Catastrophizing is an irrational thought or belief that something is far worse than it actually is. The Pain Catastrophizing Scale is a 13-item self-report measure of pain catastrophizing assessing past painful experiences [42]. The experiences are rated on a 5-point Likert scale. Its reliability and validity has been demonstrated in studies with undergraduates [42] and pain outpatient samples [43].

**Beck Depression Inventory-2**

The Beck Depression Inventory-2 (BDI-2) is a 21-question, multiple-choice, self-report measure with components related to symptoms of depression including cognition and physical symptoms. It is widely used among health care providers and researchers.

**QST**

Cool and warm detection thresholds, as well as cold and heat pain thresholds, were determined using a thermal sensory analyzer (Medoc Advanced Medical System, Ramat Yashai, Israel). Four trials of cool/warm detection thresholds and three trials for heat/cold pain thresholds were applied and averaged together to determine corresponding thresholds.

Briefly, a standard thermode (3 × 3 cm surface) was placed on the midportion of either left or right thigh (randomly assigned) to heat or cool the skin (ramping at 1°C/s). The standard program using the method of limits [44] was applied, and the subjects were instructed to push a button when they detected change in temperature (detection thresholds) and when the temperature became painful (pain thresholds). Mechanical detection thresholds were obtained using standardized Von Frey hairs delivering increasing force when pushed onto the skin at the same location on the thigh (Aesthesio, range from 0.008 to 300 g). Pressure was applied to bend the filament and held for 1 second. The up–down method was used with the last stimulus detected after the third change as the recorded threshold value.

Pressure pain thresholds were determined using a manual pressure algometer (Wagner FPK manual pressure algometer, size of contactor 1 cm², rate of increase 450 g/s). The pressure pain threshold was derived by averaging three trials with first report of pain as the end point. It was determined every 30 minutes starting 1 hour after the pain stimulus for the total duration of 3 hours.

Data were compared between the two groups, PTSD and CC, with a two-sample t-test.

**Intramuscular (IM) Pain Stimulus**

Each subject received an injection with capsaicin (100 μg in 10 μL) using a microsyringe and a 1.5-inch, 30-gauge needle into the center portion of the quadriceps muscle. Injection site was identical to that where the QST testing had been carried out. A dose response curve for intradermal capsaicin was previously done in a separate small set of healthy volunteers by a collaborator. The data showed
a dose-dependent pain response and that 10 μg of capsaicin elicited pain and 100 μg of capsaicin were required to elicit a reliable production of moderate spontaneous pain response for up to 30 minutes. By contrast, 0.1 and 1.0 μg did not reliably induce pain. Based on these observations, we used a 100-μg dose of capsaicin in 10-μL volume for IM injection.

Spontaneous pain intensity and unpleasantness were quantified with the visual analog scale (VAS, ranging from “no pain” to “worst pain imaginable”) at 5-minute intervals for 30 minutes. Data were analyzed with the repeated measures ANOVA with group (PTSD, CC) as a between-subject factor and subjective pain rating (averaged over first half of 30 minutes, averaged over second half of 30 minutes) as a within-subject repeated measure. Both main and interaction effects were examined. Intensity and unpleasantness ratings were run in two separate models.

Temporal Summation to Pressure Stimuli

Temporal summation of pain was assessed every 30 minutes starting 1 hour after the capsaicin injection (when the spontaneous pain had subsided) for the total duration of 3 hours. Ten repetitive pressure stimuli at 0.3-Hz frequency using the pressure algometer were applied. The applied force for each stimulus was increased manually over 1 second to reach the respective threshold. The resulted maximal force ranged between 50 and 225 g above the threshold. Pain ratings of C fiber mediated secondary pain occurring 1–1.5 seconds after the stimulus application was rated on a VAS after the first, fifth, and 10th stimulus.

Data were analyzed using a linear mixed effects model in which group (PTSD/CC) and repeats (first/fifth/10th stimulus) were entered as fixed factors, and subject was entered as a random factor.

Results

Subject Demographic

All 21 subjects served during the OEF/OIF conflicts in the following military services: PTSD group: Marine Corps (three), Navy (six), Army (one); CC group: Marine Corps (six), Navy (one), Army (four). All were male subjects, and the groups did not significantly differ on average age, race, and the average time of service (Table 1).

Clinical Measures

As expected, the groups differed significantly on the PTSD (i.e., CAPS), childhood trauma (i.e., CTQ), quality of life (i.e., SF-36 mental and physical health outcomes), as well as the depressive symptoms severity (BDI-2; Table 1). Although the groups were not matched for combat exposure, the ratings on the CES were not significantly different between the two groups (Table 1). The ratings for combat exposure were comparable between the two groups

Table 1  Baseline Subject characteristics and clinical measures for both groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD</th>
<th>Combat Control</th>
<th>Stats</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Demographic Variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td>10 M</td>
<td>11 M</td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>28.9</td>
<td>8.8</td>
<td>28.5</td>
</tr>
<tr>
<td>Race†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>8</td>
<td>9</td>
<td>0.01</td>
</tr>
<tr>
<td>Other</td>
<td>2</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Injured during service</td>
<td>3</td>
<td>0</td>
<td>3.9</td>
</tr>
<tr>
<td>Service duration (years)</td>
<td>6.5</td>
<td>7.1</td>
<td>0.3</td>
</tr>
<tr>
<td>Clinical variables</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CAPS score</td>
<td>68.7</td>
<td>12.7</td>
<td>15.6</td>
</tr>
<tr>
<td>Combat exposure</td>
<td>22.5</td>
<td>9.4</td>
<td>18.3</td>
</tr>
<tr>
<td>BDI-2</td>
<td>18.3</td>
<td>9.8</td>
<td>3.2</td>
</tr>
<tr>
<td>Childhood trauma</td>
<td>81.1</td>
<td>8.1</td>
<td>72.3</td>
</tr>
<tr>
<td>Pain Anxiety Symptom Scale</td>
<td>53.5</td>
<td>26.1</td>
<td>52.0</td>
</tr>
<tr>
<td>Pain Catastrophizing Scale</td>
<td>11.3</td>
<td>8.4</td>
<td>10.4</td>
</tr>
<tr>
<td>SF-36 MHC score</td>
<td>35.1</td>
<td>17.5</td>
<td>51.2</td>
</tr>
<tr>
<td>SF-36 PHC score</td>
<td>50.7</td>
<td>8.3</td>
<td>57.8</td>
</tr>
</tbody>
</table>

† This measure was compared using a χ²-test rather than a t-test.

BDI-2 = Beck Depression Inventory-2; SF-36; CAPS = clinician administered PTSD scale; CC = Combat Control group; MHC = mental health component; PHS = physical health component. PTSD = post-traumatic stress disorder group; SD = standard deviation.

Demographics, clinical and psychological variables.
Likewise, the pain anxiety and pain catastrophizing ratings were not significantly different between groups (Table 1). 

QST

PTSD and CC showed no significant differences in baseline thermal and/or mechanical sensitivity in any of the evaluated measures (Figures 1A and 2A, Table 2). To test data normality, skewness and kurtosis of the variables were calculated (for both the raw variable and the natural log-transformed variable). This transformation did not reduce the skewness of the data. For each variable, an F-Test for equity of variance was conducted, which indicated that pain showed different variance. t-Test with nonshared variance was performed on the pain data. In addition, a Mann–Whitney test was performed to determine if there was a difference between groups using non-parametric statistics. Though the variables did show moderate skewness and unequal variance, the groups did not significantly differ ($P > 0.05$).

**Intramuscular Pain Stimulus (Test for Hyperalgesia)**

The response to the intramuscular capsaicin injection measured for 30 minutes was biphasic (Figures 1 and 2). Average VAS score ratings during the first and second half following the injection are reported in Table 2. Repeated measures ANOVA showed a significant effect of group by subjective pain rating interaction for both pain intensity ($F(1,18) = 6.5, P = 0.02$) and unpleasantness ratings ($F(1,18) = 6.5, P = 0.019$). Examination of this interaction showed that in the CC group, both pain intensity and unpleasantness rating decreased over time, whereas this was not the case in the PTSD group (Figures 1 and 2). As both depression and childhood trauma are known to affect pain sensitivity, we wanted to examine whether the observed between-group differences in the change of subjective pain rating over time were related to these measures. Using BDI-2 and CTQ scores as covariates in the ANOVA model did not affect the observed results, with BDI-2 alone (intensity: $F(1,18) = 10.9, P = 0.004$; unpleasantness: $F(1,18) = 12.3, P = 0.003$), CTQ alone (intensity: $F(1,18) = 6.5, P = 0.02$).
F(1,18) = 10.7, P = 0.004; unpleasantness: F(1,18) = 5, P = 0.039) or both (intensity: F(1,18) = 14.2, P = 0.002; unpleasantness: F(1,18) = 8.4, P = 0.01). This suggests that these effects are robustly associated with PTSD in the current sample.

Temporal Summation to Pressure Stimuli

The assessment of the temporal summation to pressure stimuli showed a significant increase in pain intensity ratings over time epoch in both groups (VAS rating for PTSD: first stimulus: 13.5 ± 18.8; fifth stimulus: 31.9 ± 26.8; VAS rating for CC: first stimulus: 5.5 ± 6.4; fifth stimulus: 9.9 ± 9.4; 10th stimulus: 14.2 ± 12.8; see Figure 3). The increase in pain intensity rating following repeated application of pressure stimuli was significantly greater in the PTSD compared with the CC group (F(1,399) = 16.97, P < 0.001; see Figure 3). This significant increase in the temporal summation of pressure pain was not affected by BDI and/or CTQ covariates as the data were significant at the same level (P < 0.001) even after BDI and/or CTQ were covaried out, again suggesting that these effects are robustly associated with PTSD in the current sample.

Discussion

The novel findings of this study showed evidence for acute CS in a group of PTSD subjects in response to intramuscular capsaicin injection. The following results were observed. First, we observed no significant differences in baseline thermal and/or mechanical sensitivity indicating that sensory detection and/or pain thresholds in our PTSD group were comparable with those in the CC group. Second, we found significant between-group differences in the pain ratings over time for both sensory and emotional pain experience following intramuscular capsaicin injection. Specifically, in the CC group, pain intensity and unpleasantness ratings significantly decreased 30 minutes after capsaicin injection. However, pain intensity and unpleasantness ratings were significantly elevated in the PTSD group. Furthermore, this significant group by time interaction was not influenced by the depressive or childhood trauma symptoms severity. Third, the temporal summation of pain was significantly increased in PTSD compared with CC. Taken together, these data support the CS model in PTSD. Currently, our study is the first to use a long-lasting intramuscular pain stimulus in a PTSD population, thereby adding a novel perspective to findings from earlier experimental pain studies.

To the best of our knowledge, there are seven published studies comparing pain perception of an acute

![Figure 3](image-url) The significant increase in pain intensity ratings over time during temporal summation was bigger in the post-traumatic stress disorder group (PTSD) compared with the combat control (CC) group (P < 0.001). Shown are pain intensity responses (visual analog scale [VAS] scores) over time for the first, fifth, and 10th stimulus just above pressure pain threshold.
sexperimental pain stimulus in PTSD and control subjects [21,45–48] principally discussed in a recent review [25]. The majority of these studies used a brief superficial pain stimulus (lasting only seconds) to compare pain sensitivity in PTSD and non-PTSD groups [25]. Such a transient stimulus may not be effective in inducing CS and evoking the stress response. This may limit their usefulness in assessing these phenomena. Indeed, a prolonged input from primary afferent neurons is needed to induce an increased response of second order neurons in the spinal cord. Particularly, the C-fiber afferent neurons [49,50] appear vital in the process known as CS [51]. The intramuscular capsaicin model is well suited for this because Capsaicin causes a sustained activation of A-delta and primarily C-fibers [52]. Capsaicin activates muscle nociceptors in animal [53] and human [54] experiments. Activation of muscle nociceptors increases the excitability of central neurons to a greater extent than activation of cutaneous nociceptors [55]. Suprathreshold experimental muscle pain resembles the deep pain experience associated with clinical and chronic pain conditions [56]. Deep muscle pain is also associated with activation of the stress response and increased arousal [57]. Taken together, this makes our model an ecologically valid stimulus to test our primary hypothesis of increased sensitivity to prolonged pain in PTSD [58].

In humans, CS can manifest as pain hypersensitivity (hyperalgesia) and enhanced temporal summation [31,59–62]. Both have been described for the experimental pain caused by the application of capsaicin [52,63–66]. Although the concept of CS is widely proposed in chronic pain conditions, it also manifests acutely. Activation of muscle nociceptors by intramuscular injection can result in spinal neuron facilitation observed several minutes after stimulus application [50]. A series of preclinical experiments studying the ability of various intramuscular stimuli to modulate the excitability of spinal cord neurons also shows an effect within minutes to hours after stimulation [67].

Our reported normal QST findings suggest that baseline sensitivity of the somatosensory pathways is not disrupted in pain-free PTSD subjects. However, after several minutes of constant nociceptive input, the PTSD subjects showed significant hyperalgesia and significantly more temporal summation compared with the CC. These findings suggest that PTSD itself is associated with a sensitization of the central nociceptive system. This sensitization seemed to be potentiated by the pain stimulus given that significant baseline differences were not observed using QST testing.

Furthermore, these findings remained significant after controlling for childhood trauma, pain anxiety, and pain catastrophizing. Previous observational studies have implicated these as a link of PTSD and pain [14,15,29,68–70]. However, the current study design (i.e., pain- and medication-free PTSD subjects, acute pain stimulus) may have isolated the unique contribution of PTSD.

A clear limitation of our study is the small sample size in both groups. However, the very stringent inclusion and exclusion criteria potentially allowed for the observations described to be detected. Larger studies in this area will help confirm the robustness of our findings. The fact that we studied the acute effects of an experimental pain stimulus on pain perception in pain-free PTSD subjects with no long-term follow-up limits our ability to link the presented acute findings to the potential development of chronic pain complaints. Future longitudinal studies are needed to properly investigate the role of CS as a vulnerability factor for chronic pain.

In summary, the PTSD cohort showed higher pain ratings after 15 minutes postintramuscular capsaicin as well as significantly increased temporal summation consistent with manifestation of acute CS.

Increased excitability and spread of excitation in the CNS are often the initial steps in chronification of pain symptoms. Considering that chronic pain syndromes are multifactorial in their nature, with many contributing personality and psychological factors, our finding may represent an initial step linking PTSD and increased vulnerability to pain-related problems in these individuals.

References


Moeller-Bertram et al.


