Association of Trauma, Posttraumatic Stress Disorder, and Experimental Pain Response in Healthy Young Women

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Background: Evidence of pain alterations in trauma-exposed individuals has been found. The presence of posttraumatic stress disorder (PTSD) may be explaining these alterations, as some of the psychological characteristics of PTSD are hypothesized to increase pain response.

Objectives: To examine differences in pain response and in certain psychological variables between trauma-exposed women (TEW) with PTSD, TEW without PTSD, and non–trauma-exposed women (NTEW) and to explore the role of these psychological variables in the differences in pain response between the groups.

Methods: A total of 122 female students completed a cold pressor task (42 TEW with PTSD, 40 TEW without PTSD, and 40 NTEW). Anxiety sensitivity, experiential avoidance, trait and state dissociation, depressive symptoms, state anxiety, catastrophizing, and arousal were assessed.

Results: TEW with PTSD reported significantly higher pain unpleasantness than NTEW, but not more than that of TEW without PTSD. They also presented higher trait dissociation, state anxiety, depressive symptoms, and skin conductance than the other 2 groups and higher anxiety sensitivity than TEW without PTSD. TEW without PTSD reported more pain unpleasantness than NTEW, but they recovered faster from pain. However, these differences were not explained by any psychological variable.

Conclusions: The results suggest that although trauma-exposed individuals are not more sensitive to painful stimulation, they evaluate pain in a more negative way. Exposure to trauma itself, but not to PTSD, may explain the differences found in pain unpleasantness.

Key Words: PTSD, trauma, pain response, psychological variables, cold pressor task

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Evidence that trauma exposure increases the risk of chronic pain has been recently reported.[1,2] Furthermore, alterations in pain sensitivity have been observed in trauma-exposed individuals (TEI).[3–5] The presence of posttraumatic stress disorder (PTSD) may explain these alterations.[6–8] In fact, empirical evidence suggests that some of the psychological characteristics of PTSD may increase pain response [ie, anxiety sensitivity (AS), experiential avoidance (EA), dissociation, state anxiety, depression, catastrophizing, perceived, and physiological arousal].[9–13] However, the findings of the few studies examining pain response in individuals with PTSD are contradictory,[14] with results pointing to both increased[15,16] and decreased[16–18] pain sensitivity. For example, some researchers have found lower pain intensity in response to heat stimulation among individuals with PTSD,[17,18] whereas others have reported higher pain intensity among these individuals.[16] Similarly, higher[16,18] and lower[15] pain thresholds have both been found among individuals with PTSD, indicating decreased and increased pain sensitivity, respectively. Results indicating no alterations in pain sensitivity in PTSD individuals have also been found.[19] Moreover, the association between the above-mentioned psychological variables and the pain experience has not yet been examined in individuals with PTSD.

In addition, most previous studies have been conducted with military samples in which the presence of serious injuries, chronic pain, and other health problems, as well as psychological disorders, may be overrepresented. These factors may explain the inconsistencies observed in the results.[14] Furthermore, TEI with and without PTSD have been compared with healthy controls in only 1 study.[18] Studies including TEI with and without PTSD are important, as trauma exposure may lead to pain alterations by mechanisms different to those of PTSD (eg, other anxiety disorders and depression). In addition, studies have usually investigated victims of a single type of trauma exposure (e.g., combat veterans and victims of sexual abuse), rather than considering the wider spectrum of traumatic events a person may have experienced over their lifetime, which limits the generalizability of results and may decrease reliability when assessing trauma exposure.[20] Furthermore, with the exception of the study of Santana et al (written personal communication)—in which participants with PTSD reported lower pain tolerance during a cold pressor task—mechanical or thermal superficial pain stimulation have usually been used, instead of experimental pain models producing prolonged and deep pain, such as the cold pressor. Therefore, studies in which different pain modalities are used are needed.[14] In addition, sex and gender are also potentially confounding factors that need to be taken into account in these types of studies.[14] Evidence supporting sex and gender differences in pain modulation between men and women have been found in previous studies.[21–23] Examining pain alterations in trauma-exposed women (TEW) is especially important, as both PTSD and chronic pain are more prevalent among women.[24]

Therefore, the aims of this study were: first, to examine differences in the pain response between TEW with PTSD,

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TEW without PTSD, and non–trauma-exposed women (NTEW) during a cold pressor task. Exposure to a broad range of traumatic situations was assessed. Second, to explore differences between the 3 groups in AS, EA, dissociation, state anxiety, depression, catastrophizing, and perceived as well as physiological arousal. Third, to explore the role of these psychological variables in the differences in pain response found between the groups. Finally, the relationship between posttraumatic stress symptoms and pain response was examined among the TEW. We hypothesized that TEW with PTSD would report increased pain experience, as well as higher scores for each of the psychological variables assessed, than TEW without PTSD and NTEW. Differences between the TEW without PTSD and NTEW in any of the variables were not expected. Among the TEW, positive relationship between PTSD symptoms and pain experience was expected.

MATERIALS AND METHODS

Participants and Procedures

All study procedures were approved by the University of Málaga Institutional Review Board. Potential participants were identified through a screening session conducted at the Psychology Faculty of the University of Málaga, in which trauma exposure was assessed using the Stressful Life Event Screening Questionnaire Revised (SLESQ-R),[25] and PTSD symptoms were assessed using the Davidson Trauma Scale (DTS).[26] Selected participants were contacted by phone and invited to participate in a single individual session lasting approximately 1 hour. Before participation, informed consent was obtained. During this session, PTSD symptoms and trauma exposure were again assessed, as the experiments were conducted approximately 9 months after the screening session. The final sample comprised 122 female students: 42 TEW with PTSD (mean age = 20.84; SD = 5), 40 TEW without PTSD (mean age = 21.05; SD = 3.57), and 40 NTEW (mean age = 19.66; SD = 3.25).

Measures

Sociodemographic Characteristics and Trauma Exposure

Sociodemographics and information regarding presence of chronic pain, menstrual phase,[27,28] oral contraceptive use, smoking habits, time of day the experiment was conducted, and any participation in similar experiments[29] was collected. Trauma exposure was assessed using the SLESQ-R,[25] designed to identify criterion-A traumatic events in non–treatment-seeking samples and to minimize the reporting of subthreshold events.[30] It has very good test-retest reliability and convergent validity and provides good discrimination between criterion-A and non-criterion-A events.[30] As it does not assess criterion-A2 of the PTSD diagnosis, participants were asked to rate from 0 ("Not at all") to 10 ("extreme intensity") the fear experienced during the event and their feelings of helplessness. Criterion-A2 was considered fulfilled when a participant responded with a score of 8 or more to the fear or helplessness scales.[31] Thus, a participant was considered to have been exposed to traumatic events when she answered *Yes* to one of the SLESQ-R items and when she scored 8 or more on the fear or helplessness scales.

PTSD Symptoms

The DTS[26,32] was developed to assess the severity and frequency of the 17 PTSD symptoms found in DSM-IV and to assess treatment outcome. It has been adapted into Spanish.[33] It is composed of 2 scales ranging from 0 to 4. The DTS can differentiate patients with PTSD and partial PTSD from patients without PTSD in the general population and provides normative population data.[34] It has good reliability and validity properties, and diagnostic assessment using the DTS provides reasonable accuracy compared with the Structured Clinical Interview for DSM-III-R.[35] Given a score of 40, the positive predictive value, negative predictive value, and efficiency are 0.92, 0.79, and 0.83, respectively.[32] TEW were assigned to the groups with and without PTSD according to their DTS scores at the time of the experimental session.

Psychological Pain-related Variables

The Dissociative Experiences Scale[36] was used to assess trait dissociation. Both the original instrument[37] and its Spanish version[38] present good psychometric properties.

The peritraumatic dissociative reactions were assessed with the Peritraumatic Dissociative Experiences Questionnaire Modified.[39] It can be used to quantify the amount of acute dissociation that people experience during a specific event. It has good internal consistency, test-retest reliability, and convergent-divergent validity.[39]

The Anxiety Sensitivity Index (ASI) Taxon Scale[40] contains 8 items, namely those ASI items that best discriminated between taxometrically based dichotomous class membership. It presents significant incremental validity above and beyond the dimensional full-scale ASI total score.[40,41] As the latter score does not account for any unique variance above and beyond the ASI Taxon Scale in relation to PTSD,[40] we used the ASI Taxon Scale and not the full-item total ASI score. The Spanish version of the ASI is fully equivalent to the original.[42,43]

The Acceptance and Action Questionnaire[44] was used to measure the tendency to engage in emotional avoidance. Higher scores on the Acceptance and Action Questionnaire have been associated with greater general psychopathology.[44] This 9-item questionnaire and its Spanish version has good psychometric properties.[45]

Depressive symptoms were assessed with the Center for Epidemiologic Studies Depression Scale (CES-D).[46] CES-D items are endorsed on a 4-point scale ranging from 0 (*Never or few*) to 3 (*Uusually*). Both the original and the Spanish version of this questionnaire[46,47] have good psychometric properties.[48,49]

State anxiety was assessed with the state anxiety scale of the State-Trait Anxiety Inventory.[50] The Spanish version of this scale[51] has adequate validity and reliability. We modified its instructions to ask for the anxiety experienced during the previous week.

To assess any catastrophizing experienced during the cold pressor test, the Spanish version[52] of the Pain Catastrophizing Scale[53] was adapted to the experimental situation. The questionnaire has good internal consistency, construct validity, and temporal stability.[53] Situation-specific catastrophizing has shown to correlate more strongly with experimental pain outcomes than standard assessments of pain catastrophizing.[54,55] This modified version presented an internal consistency of 0.83 (Cronbach-α).

Perceived arousal was assessed using an 11-point self-report rating numerical scale ranging from 0 ("Nothing") to

10 (“A lot”) in which the participants were asked “How stressful was the experimental procedure for you?”

Induced-pain Procedure: Cold Pressor Task

A cold pressor task was conducted to induce pain in the participants. The same experimenter tested all the participants. The cold pressor device consisted of 2 metal containers measuring approximately $50 \times 30 \times 30$ cm. One of the containers was filled with warm water at 37°C and the other with cold water maintained between 1.5 and 3°C, which was fitted with a pump providing circulation to prevent heat building up around the immersed hand. The participants first placed their nondominant hand up to their forearm in the warm water for 2 minutes, to ensure that the arms of all the participants had the same baseline temperature, and then they transferred their hand to the cold water container. Participants were asked to try to keep their arm in the cold water for as long as possible but were told that they could remove it at any time if they could no longer tolerate the pain. They were not allowed to keep their hand in the cold water for > 3 minutes[56,57] but were not informed of this upper time limit.

During the procedure, several outcomes were assessed as indicators of the pain response: pain threshold (amount of time until the participant started to feel pain after introducing their hand into the cold water), pain tolerance (amount of time the participant managed to keep her hand immersed in the water), and time to recover from pain (time taken to report the cessation of pain once the task finished). Pain intensity and pain unpleasantness were also assessed at the end of the task, using two 11-point numerical rating scales ranging from 0 (no pain/unpleasantness) to 10 (the worst possible pain/unpleasantness). To clarify the difference between pain intensity and pain unpleasantness, we provided participants with instructions similar to those given by other authors.[58] Specifically, pain intensity was described as being analogous to the volume of music, and pain unpleasantness was described as being analogous to how much the participant likes the music.

Physiological Arousal

A LabLinc V physiological data recording system (Coulbourn Instruments, USA) was used to assess heart rate (HR) and skin conductance (SC) at baseline and during the first 9 seconds of exposure to the cold water.[15,59] WINDAQ software (DATAQ Instruments) was used. SC was measured using 2 reusable 8-mm surface electrodes (AgCl) placed over the medial phalanx of the ring and middle fingers of the nondominant hand.

Data Analysis

Data analysis was conducted using SPSS, version 16.0. A 1-way multivariate analysis of covariance (MANCOVA) was performed considering pain threshold, pain tolerance, pain intensity, pain unpleasantness, and time to recover from pain as dependent variables. Conducting the experiment during the morning/afternoon, menstrual phase, and smoking were considered as covariates. Univariate analyses were also performed. As pooled within-cell correlations, adjusted for independent variables, were in excess of 0.30,[60] and there were theoretical and practical reasons to prioritize the dependent variables, a Roy-Bargmann stepdown analysis was also conducted. To examine which groups significantly differed, post hoc comparisons were performed. The power ($1-\alpha$) of the global effect of the MANCOVA was calculated using GPower software.[61] It was 0.99 for detecting a small effect size of $f^2 = 0.1$ (ie, 99% of chance of finding a significant effect that actually exists in the population).

To examine differences between groups in the psychological variables assessed, a 1-way multivariate analysis of variance (MANOVA) with sequential adjustment for nonorthogonality was performed. Univariate analyses and a Roy-Bargmann stepdown analysis were conducted. The dependent variables were introduced in the following order: AS taxon, EA, trait dissociation, depression, state anxiety, state dissociation, catastrophizing, and perceived arousal. Therefore, the AS taxon was tested using a univariate analysis of variance, EA was tested using an analysis of covariance (ANCOVA) in which AS was introduced as a covariate, trait dissociation was tested with an ANCOVA using both AS and EA as covariates, and so on. Then, post hoc tests were performed. The statistical power for the global effect of the MANOVA was 0.88 for finding a moderate effect size of $f^2 = 0.25$.

To examine group differences in physiological arousal, 2 ANCOVAS were conducted with HR and SC during the first 9 seconds of cold water immersion, respectively, as the dependent variables. Baseline HR and SC as well as room temperature and humidity were considered as covariates. The statistical power for the global effect was 0.68 for finding a moderate effect size of $f^2 = 0.25$.

Finally, to examine the relationship between PTSD symptoms and pain experience among the TEW, zero-order correlations between PTSD symptoms (total DTS scores as well as DTS scores in hyperarousal, reexperience, and avoidance/numbing) and pain variables (pain thresholds, pain tolerance, pain intensity, pain distress, pain unpleasantness, and time to recover from pain) were conducted.

RESULTS

Screening Session

A total of 692 undergraduate university students (142 men and 550 women) participated in the screening session. Of these, 372 (297 women and 75 men) reported trauma exposure. Those trauma exposure students with DTS scores ≥ 40, suggesting PTSD diagnosis, namely 104 individuals (85 women and 19 men) were invited to participate. Unfortunately, it was only possible to contact 4 of the 19 men. Thus, only the women were included in the study. From the 85 female students with DTS scores suggesting PTSD, 52 accepted to participate. In addition, 80 female students who reported exposure to at least 1 traumatic situation during their life and who presented DTS scores < 40 were contacted, and 68 of them accepted participation. Finally, from a sample of 140 women who had never been exposed to a traumatic situation, 81 were invited to participate. In this case, the women were selected only when they responded negatively to all the items of the SLESQ-R.[25] Of these, 10 participated in a simulation that was conducted to ensure that all devices were functioning correctly and as training for the researcher, and 40 participated in the final experimental session.

At the time of the experiment, the TEW completed the DTS again and were regrouped according to their DTS scores at this time. Therefore, those women scoring > 40 on the DTS[32] at the time of the experiment were included in the groups of TEW with PTSD (n = 42), whereas those scoring between 10 and 33 on the DTS were included in the group of TEW without PTSD (n = 40), and 40 female students were included in the NTEW. TEW with DTS scores below 10 or between 33 and 40 were not considered in the analyses (Fig. 1).

The sample of TEW (with or without PTSD) was characterized by exposure to different kinds of traumatic situations, which—together with their DTS scores—are presented in Table 1. There were no statistically significant differences between groups in the frequency of exposure to the different categories of traumatic situations. CES-D means (SD) were 14.58 (7.91) for the NTEW, 14.1 (6.82) for the TEW without PTSD, and 23.17 (9.20) for the TEW with PTSD.

Preliminary Analysis

No multivariate outliers were found. Transformations were performed to eliminate the potential effects of several univariate outliers and to improve the distributions of the variables,[60] that is, the logarithm of pain thresholds, pain tolerance, time to recover from pain, the reverse of pain distress, and the root square of the reverse of pain intensity. Regarding psychological and physiological variables, the root square of depression, trait dissociation, and SC were calculated, the logarithm of HR was computed, and state dissociation was reversed. Assumptions of normality, homogeneity of variance-covariance matrices, linearity, and multicollinearity were applied to both the MANCOVA and the MANOVA. The overall homogeneity of regression and homogeneity of regression for the stepwise analyses were satisfied for the MANCOVA. In the case of the MANOVA, homogeneity of regression was not satisfied for either depression or perceived arousal variables of the stepdown analyses. They were therefore extracted from the analysis, and differences in these variables were examined with two one way analysis of variances. Correlations between the pain variables are presented in Table 2.

Differences in the Pain Response

The multivariate effect of group membership was statistically significant (Wilk criterion was 0.83; $F_{2,115} = 2.11$; $P = 0.024$; $\eta^2 = 0.17$). There were no significant associations between the pain response and the covariates. There were significant differences between the groups in pain unpleasantness and time to recover from pain (Fig. 2). Namely, group membership explained 7% and 6.3% of the variance in pain unpleasantness and time to recover from pain, respectively, after controlling for several covariates and for pain thresholds and pain tolerance (as well as pain unpleasantness in the case of time to recover from pain). Both, TEW with and without PTSD reported more pain unpleasantness than NTEW (both groups $P < 0.05$). Pain unpleasantness differences between TEW with and without PTSD were not significant. TEW without PTSD recovered significantly faster than NTEW ($P < 0.01$). Differences in time to recover from pain between TEW with and without PTSD were not significant. Differences between TEW with PTSD and NTEW were not significant. Similar results were found when examining the results of the univariate analyses (Table 3).

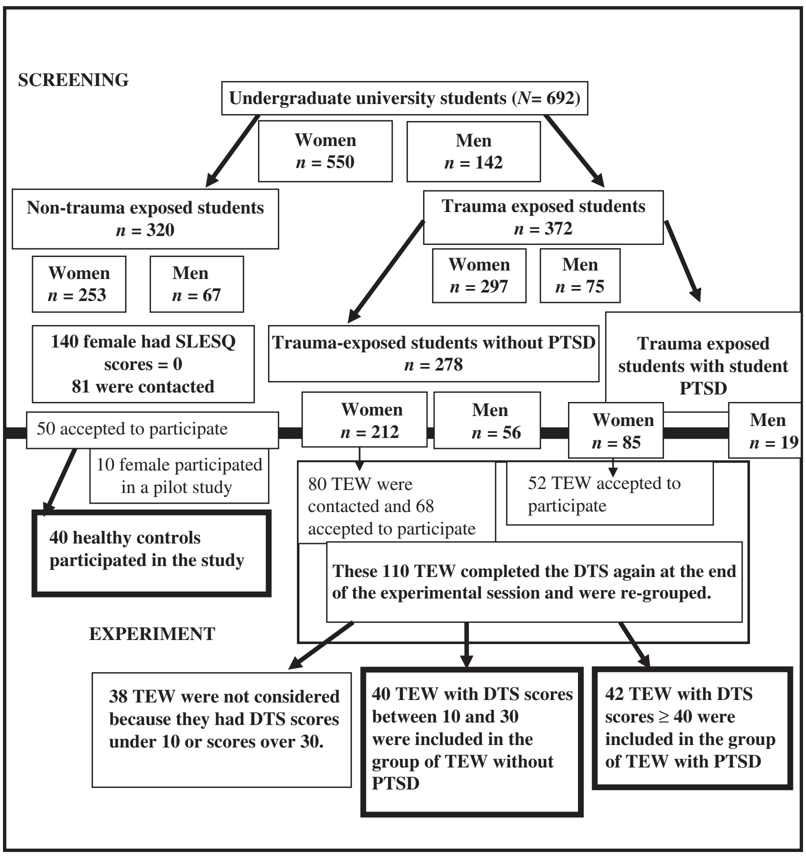

FIGURE 1. Results of the screening session and selection of participants. DTS indicates Davidson Trauma Scale; PTSD, posttraumatic stress disorder; SLESQ, Stressful Life Event Screening Questionnaire; TEW, trauma-exposed women.

TABLE 1. Type of Traumatic Event Experienced and Mean and SD of the DTS Scores of the TEW With and Without PTSD

	n (%)	
Traumatic Events	TEW Without PTSD (n = 40)	TEW With PTSD (n = 42)
Life-threatening illness	1 (2.5)	5 (11.6)
Life-threatening accident	6 (15.0)	8 (18.6)
Robbery with physical force or use of weapon	5 (12.5)	4 (9.5)
Death of a very close person because of accident, homicide, or suicide	6 (15.0)	13 (30.2)
Sexual abuse	8 (9.6)	12 (10.4)
Physical abuse	7 (17.5)	6 (14.3)
Emotional abused	20 (50.0)	21 (50.0)
Being threatened with a weapon	2 (5.0)	2 (4.8)
Witness to violence	9 (22.5)	11 (25.6)
Exposure to other life-threatening situations	12 (30.0)	15 (35.7)
Miscarriage	5 (12.5)	6 (14.3)
	Mean (SD)	Mean (SD)
No. trauma experiences	2.22 (1.44)	2.65 (1.76)
DTS scores	20.75 (6.85)	57.19 (11.82)

DTS indicates Davidson Trauma Scale; PTSD, posttraumatic stress disorder; TEW, trauma-exposed women.

Differences in Psychological Variables

The multivariate effect of group membership was statistically significant (Wilk criterion was 0.66; $F_{12,224} = 4.29$; $P < 0.001$; $\eta^2 = 0.40$). There were significant differences between the groups in AS taxon, trait dissociation, and state anxiety (Fig. 3). TEW with PTSD presented significantly higher AS taxon scores than those without PTSD ($P = 0.001$) but not more than NTEW. There were no differences between TEW without PTSD and NTEW in this variable. TEW with PTSD also presented higher trait dissociation and state anxiety scores than TEW without PTSD (both variables $P = 0.018$) and NTEW ($P < 0.001$ and < 0.01, for dissociation and anxiety, respectively).

Univariate analyses revealed significant mean differences in EA between groups. However, when AS taxon was controlled, mean differences in EA became nonsignificant. Differences between groups in catastrophizing were not statistically significant but came close to reaching significance ($P = 0.057$).

TABLE 2. Correlations Among the Pain Variables Among the Total Sample (n = 122)

	1	2	3	4
1. Pain thresholds	—	—	—	—
2. Pain tolerance	0.40***	—	—	—
3. Pain intensity	0.08	0.35***	—	—
4. Pain unpleasantness	0.13	0.29**	0.61***	—
5. Time to recover from pain	0.03	0.42***	0.35***	0.20*

***$P < 0.001$ (2-tailed); **$P < 0.01$ (2-tailed); *$P < 0.05$ (2-tailed).

TEW with PTSD presented higher depressive symptoms than those without PTSD ($P < 0.001$) and higher than NTEW ($P < 0.001$). Differences between TEW without PTSD and NTEW were not significant. There were no differences in perceived arousal between the groups.

Differences in Physiological Arousal

TEW with PTSD presented significantly higher SC scores than TEW without PTSD during the first 9 seconds of cold water exposure ($P < 0.05$) but not significantly higher than NTEW. Differences between TEW without PTSD and NTEW were not significant. No differences in HR were found between the groups (Fig. 3).

Analyses of the Role of Psychological Variables

To examine whether the differences in pain unpleasantness between TEW with PTSD and NTEW could be explained by the differences between both groups in trait dissociation, state anxiety, and depression, an ANCOVA was performed including these variables as covariates. Menstrual phase was also included as a covariate, as previous analyses showed that it accounted for a significant proportion of the variance in pain unpleasantness. The results of the analysis showed that after controlling for these variables, differences between the groups became nonsignificant, $F_{1,79} = 2.64$; $P = 0.109$. However, regression analyses showed that neither trait dissociation, nor state anxiety, nor depression provided a statistically significant adjustment of pain unpleasantness.

Association Between PTSD Symptoms and Pain Variables Among TEW

The results of the zero-order correlations conducted with the sample of TEW with and without PTSD showed that the total DTS scores were significantly and positively correlated with time to recover from pain (Table 3). DTS avoidance and DTS hyperarousal subscales, but not DTS reexperience, were also significantly related to time to recover from pain. Total DTS scores, DTS avoidance, and DTS hyperarousal scores tend to be positively related with pain intensity, but statistical significance was not reached ($P = 0.054$, 0.060, and 0.068, respectively). DTS scores were not significantly correlated with pain unpleasantness, pain thresholds, or pain tolerance.

DISCUSSION

The purpose of this study was to investigate differences in pain response and in psychological variables between 3 groups of female students (TEW with PTSD, TEW without PTSD, and NTEW) and to explore the role of the psychological variables in the differential pain experience among the groups. After controlling for several covariates, group membership explained 18% of the total variance in the pain response. Both groups of TEW reported higher pain unpleasantness than NTEW. However, TEW (with or without PTSD) did not significantly differ in this variable, which suggests that exposure to trauma itself—and not the development of PTSD symptoms after this event—could explain these differences and that trauma exposure is related to the affective aspects of pain and not to its sensorial aspects. Our results are in line with Creech,[4] who found higher unpleasantness in trauma-exposed than in non–trauma-exposed female undergraduate students, but not with Fillingim and Edwards,[62] who found lower pain

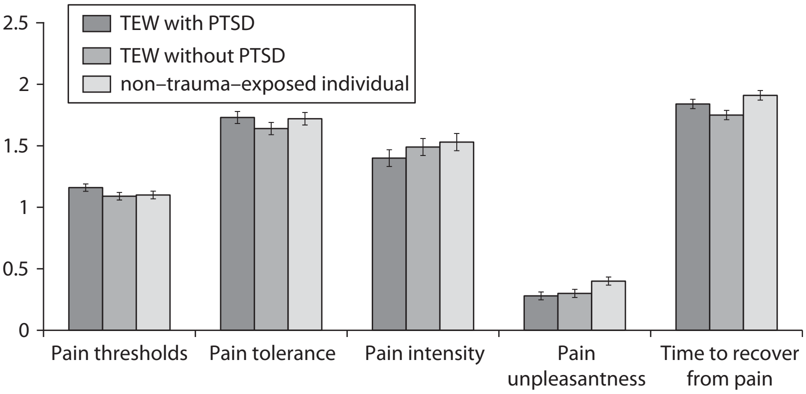

FIGURE 2. Mean differences in pain variables (pain thresholds, pain tolerance, pain intensity, pain distress, time to recover from pain) between TEW with PTSD, TEW without PTSD, and NTEW.
Note: Lower scores in pain intensity must be interpreted as higher scores in pain intensity, as analyses were conducted with the root square of the reverse of pain intensity, and data here presented refer to the scores in the transformed variables. Similarly, lower scores in pain unpleasantness must be interpreted as higher scores in pain unpleasantness, as analyses were conducted with the reverse of pain unpleasantness. NTEW indicates non–trauma-exposed women; PTSD, posttraumatic stress disorder; TEW, trauma-exposed women.

unpleasantness in female victims of childhood abuse. However, these authors did not assess the full range of lifetime traumatic situations experienced by participants, which could have affected their results. In line with Fillingim and Edwards,[62] we did not find group differences between pain thresholds or pain intensity between TEW and NTEW. Differences in these variables were not found between TEW with PTSD and NTEW. This is in line with the results of Schmahl et al,[19] who failed to find significant pain differences in thermal pain thresholds between women with PTSD diagnoses and healthy women. In addition, the absence of differences in pain intensity is in line with the results of Strigo et al,[63] who did not find differences in pain ratings between victims of intimate partner violence with and without PTSD when the authors first exposed them to brief thermal stimulation. Nonetheless, they found a subsequent decrease in pain ratings in the PTSD group with repeated exposure to pain. As we did not take into account the temporal aspect of pain exposure, we do not know whether reductions in pain intensity may have occurred during the prolonged exposure to the cold pressor. Future studies should investigate differences in pain variations during prolonged pain-induction procedures in TEIs with and without PTSD. The absence of differences in the sensory aspects of pain found in the present study was not due to a lack of power of the analysis. They may be due to the fact that laboratory pain has a less threatening meaning for PTSD patients than pain in a clinical setting. Perhaps PTSD patients only amplify those pains with a negative meaning in their lives or those that have been associated with the trauma and could therefore trigger PTSD symptoms.

TABLE 3. Correlations Among Davidson Trauma Scale (DTS) Scores and Pain Experience Variables Among Trauma-exposed Women ($n = 82$)

	1	2	3	4
1. Total DTS	—			
2. DTS reexperience	0.87**	—		
3. DTS avoidance	0.91**	0.67**	—	
4. DTS hyperarousal	0.85**	0.52**	0.66**	—
5. Pain thresholds	0.14	0.04	0.12	0.18
6. Pain tolerance	0.17	0.18	0.11	0.15
7. Pain intensity	−0.21	−0.13	−0.21	−0.20
8. Pain unpleasantness	−0.14	−0.10	−0.18	−0.08
9. Time to recover from pain	0.27*	0.17	0.24*	0.28**

**$P < 0.01$ (2-tailed); *$P < 0.05$ (2-tailed).

Our findings contrast with those of Defrin et al,[16] who reported higher pain thresholds (indicative of decreased pain sensitivity) and higher pain intensity (indicative of increased pain sensitivity) in individuals with PTSD compared with both healthy participants and patients with anxiety disorders. Unfortunately, they did not assess pain unpleasantness. These discrepancies could be explained by the many methodological differences between our study and Defrin et al's study.[16] Similarly, these results contrast with the results of Kraus et al[18] who found lower pain ratings in combat-related PTSD patients compared with a combat group without PTSD and a healthy sample. Nonetheless, in their study, only 10 participants were included in each group, all the participants were men, and their sample also differed from our sample in the type of trauma exposure presented.

Unexpectedly, although the TEW without PTSD had more pain unpleasantness than NTEW, they recovered faster, which could indicate that TEW without PTSD may have some characteristics that protect them from extreme stress. In fact, Liberzon et al,[64] found an increased density of some of the receptors involved in pain processing among TEI without PTSD when compared with non-TEI and with TEI with PTSD, which seemed to reflect an adaptive response. A higher pain unpleasantness in these individuals may serve as an alarm mechanism that produces stronger endogenous or faster pain inhibition.

TEW with PTSD were expected to differ from the other 2 groups in a number of psychological variables previously related to pain. No differences between TEW without PTSD and NTEW were expected. TEW with PTSD showed significantly higher AS than TEW without PTSD and tended to present higher AS than NTEW.[65–67] They also presented higher trait dissociation,[68] state anxiety,

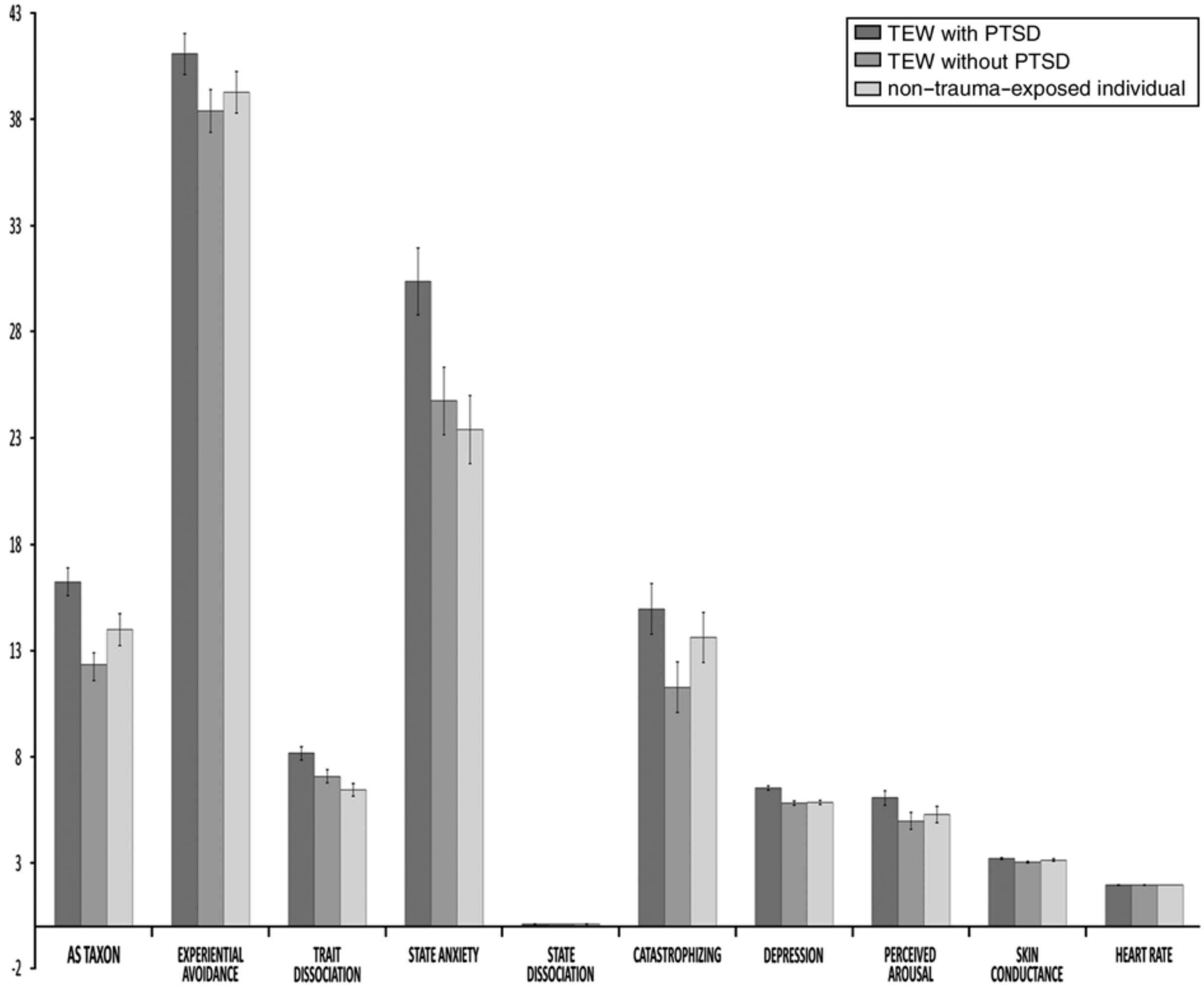

FIGURE 3. Mean differences in psychological and physiological variables between TEW with PTSD, TEW without PTSD, and NTEW. *Note*: Lower scores in state dissociation must be interpreted as higher scores in state dissociation, as analyses were conducted with the reverse of this variable, and data here presented refer to the scores in the transformed variables. AS indicates anxiety sensitivity; NTEW, non–trauma-exposed women; PTSD, posttraumatic stress disorder; TEW, trauma-exposed women.

and depressive symptoms than TEW without PTSD and NTEW. Differences in EA were also found; however, in line with the results of Berman et al,[69] these differences disappeared when AS was controlled. As expected, TEW without PTSD did not differ from NTEW in any psychological variables, which suggests good psychological adjustment within the TEW without PTSD, despite having suffered a traumatic situation. No significant group differences were found in catastrophizing, although TEW with PTSD tended to report higher catastrophizing. TEW with PTSD may present more catastrophizing only in response to a more relevant clinical pain, especially in those cases in which the pain is a consequence of injury during the trauma. Further research on this issue is needed.

Although theorist hypothesize that the heightened arousal of PTSD patients leads to pain amplification,[9,10,70] we did not find differences in perceived arousal between the groups. The fact that perceived arousal was retrospectively assessed at the end of the experiment may have affected these results. Nevertheless, some evidence was found suggesting a higher SC in response to the cold water in TEW with PTSD. PTSD patients are suggested to increase physiological arousal when faced with experimental material related to their trauma but not when exposed to a stressor unrelated to it.[71] This issue also warrants future research.

TEW without PTSD did not differ from the NTEW in any of the psychological variables examined, which suggests that none of these variables accounted for the higher pain unpleasantness found in the former group. Other mechanisms may account for these results (ie, central sensitization).[72] Nevertheless, our results suggest that trait dissociation, depressive symptoms, and state anxiety could account for the differences found between TEW with PTSD and NTEW in pain unpleasantness. Studies examining the interaction between PTSD and these variables are needed.

In line with our hypothesis, among the TEW (with and without PTSD), positive correlations were found between total PTSD symptoms scores—as well as avoidance and hyperarousal—and time to recover from pain. These results are in line with the important role of hyperarousal and avoidance in pain amplification posited by current theories on the co-occurrence of chronic pain and PTSD.[9,10]

However, in contrast, no correlations were found between PTSD symptoms scores and any of the other pain response indicators (pain thresholds, pain tolerance, pain unpleasantness). Nonetheless, total PTSD symptoms scores—as well as hyperarousal and avoidance symptoms—tended to be related to pain intensity. It may be possible that the relationship hypothesized between PTSD symptoms and pain occurs only in samples with a clinically significant level of pain or only in those individuals whose pain is related to an injury suffered in the aftermath of the trauma. More research on the association of PTSD symptoms and experimental pain is needed.

This study has a number of limitations. First, the group of participants assumed to have PTSD was selected according to their scores on a self-report questionnaire. Thus, they could not fulfil all the criteria needed for a full diagnosis of PTSD according to DSM-IV. Second, the group of TEW without PTSD presented DTS scores raging from 10 to 30. According to some authors,[34] these scores may be considered subsyndromal PTSD symptoms. These subsyndromal levels of PTSD symptoms may explain the differences found in pain unpleasantness. Nevertheless, correlation analyses conducted with the whole sample of TEW (with and without PTSD) did not show a significant relationship between DTS scores and pain unpleasantness. Studies examining pain sensitivity in TEW with DTS scores below 10 are needed, as these individuals may present more resilient characteristics that protect them against alterations in pain sensitivity. Third, because adjustment for multiple testing is not recommended in exploratory studies,[73] many comparisons were conducted with the same data; therefore, future confirmatory studies are needed. Fourth, only female students participated in the study. Although studies on women are needed, especially when taking into account that both PTSD and pain are more prevalent among them, future studies should be conducted with samples comprising both sexes, as differences in pain modulation seem to exist between them.[21,23] Fifth, AS and EA were assessed approximately 9 months before the experiments and, although they are considered trait variables, different results may have been obtained if they had been assessed during the experimental session. Moreover, it was not possible to use a double-blind design, which could have affected the results; nonetheless, at the time of the task, the experimenter did not know to which group each participant belonged, because participants were selected from a random list of potential participants by a telephone call. Finally, pain alterations in TEIs may be due, at least in part, to the fact that some traumatic events involve personal physical injuries.[74] Future studies considering the influence of traumatic injury on the pain response of TEIs are needed.

Despite these limitations, this is the first study that has simultaneously compared pain response differences in TEW with PTSD, TEW without PTSD, and NTEW. Making a distinction between trauma exposure and PTSD after trauma is of undoubted relevance. Likewise, because the literature indicates the importance of assessing the entire spectrum of life adversities in which a person has been involved while ensuring that there are no TEIs in the nontrauma group,[20,75] we assessed a broad range of stressful situations experienced during the lifetime of the participants. Furthermore, this is the first experimental study to explore the role of some of the psychological variables that are thought to be involved in pain amplification among PTSD patients. Although many theories point to the role of these variables, empirical studies supporting their role in pain sensitivity among PTSD patients are scarce and more research is needed. Finally, this is the first study in which a prolonged and deep pain-induction procedure, such as the cold pressor task, has been used.

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REFERENCES

1. Lampe A, Doeringa S, Rumpold G, et al. Chronic pain syndromes and their relation to childhood abuse and stressful life events. *J Psychosom Res*. 2003;54:361–367.
2. Jones GT, Power ST, Macfarlane GJ. Adverse events in childhood and chronic widespread pain in adult life: results from the 1958 British Cohort Study. *Pain*. 2009;143:92–96.
3. Scarinci IC, McDonald HJ, Bradley LA, et al. Altered pain perception and psychosocial features among women with gastrointestinal disorders and history of abuse: a preliminary model. *Am J Med*. 1994;97:108–118.
4. Creech SK. *Impact of Written Emotional Disclosure of Trauma on Laboratory Induced Pain*. Texas: Texas A&M University Available at: http://hdl.handle.net/1969.1/85964; 2008. Accessed May 30, 2012.
5. Fillingim RB, Maixner W, Sigurdsson A, et al. Sexual and physical abuse history in participants with temporomandibular disorders: relationship to clinical variables, pain sensitivity, and psychologic factors. *J Orofac Pain*. 1997;11:48–57.
6. Ciccone DS, Elliott DK, Chandler HK, et al. Sexual and physical abuse in women with fibromyalgia syndrome. A test of the trauma hypothesis. *Clin J Pain*. 2005;21:378–386.
7. Campbell J, Greeson MR, Bybee D, et al. The co-occurrence of childhood sexual abuse, adult sexual assault, intimate partner violence, and sexual harassment: a mediational model of posttraumatic stress disorder and physical health outcomes. *J Consult Clin Psychol*. 2008;76:194–207.
8. Shipherd JC, Pineles SL, Gradus JL, et al. Sexual harassment in the Marines, posttraumatic stress symptoms, and perceived health: evidence for sex differences. *J Trauma Stress*. 2009;22:3–10.
9. Asmundson GJG, Coons MJ, Taylor S, et al. PTSD and the experience of pain: research and clinical implications of shared vulnerability and mutual maintenance models. *Can J Psychiatry*. 2002;47:930–937.
10. Liedl A, Knaevelsrud C. Chronic pain and PTSD: the perpetual avoidance model and its treatment implications. *Torture*. 2008;18:69–79.
11. Sharp TJ, Harvey AG. Chronic pain and posttraumatic stress disorder: mutual maintenance? *Clin Psychol Rev*. 2001;21:857–877.
12. Asmundson GJG, Taylor S. PTSD and chronic pain: cognitive-behavioral perspectives and practical implications. In: Young G, Kane AW, Nicholson N, eds. *Psychological Knowledge and Evidence in Court: PTSD, Pain, and TBI*. New York: Springer; 2006:225–241.
13. Otis JD, Pincus DB, Keane TM. Comorbid chronic pain and posttraumatic stress disorder across the lifespan: a review of theoretical models. In: Young G, Kane A, Nicholson K, eds. *Psychological Knowledge in Court: PTSD, Pain, and TBI*. New York: Springer; 2006:242–268.
14. Moeller-Bertram T, Keltner J, Strigo I. Pain and post traumatic stress disorder. Review of clinical and experimental evidence. *Neuropharmacology*. 2011;62:586–597.
15. Orr SP, Roth WT. Psychophysiological assessment: clinical applications for PTSD. *J Affect Disord*. 2000;61:225–240.
16. Defrin R, Ginzburg K, Solomon Z, et al. Quantitative testing of pain perception in subjects with PTSD—implications for the mechanism of the coexistence between PTSD and chronic pain. *Pain*. 2008;138:450–459.

17. Geuze E, Westenberg HGM, Jochims A, et al. Altered pain processing in veterans with posttraumatic stress disorder. *Arch Gen Psychiatr*. 2007;64:76–85.
18. Kraus A, Geuze E, Schmahl C, et al. Differentiation of pain ratings in combat-related posttraumatic stress disorder. *Pain*. 2009;143:179–185.
19. Schmahl C, Meinzer M, Zeuch A, et al. Pain sensitivity is reduced in borderline personality disorder, but not in posttraumatic stress disorder and bulimia nervosa. *World J Biol Psychiatry*. 2010;11:364–371.
20. Finkelhor D, Ormrod RK, Turner HA. Polyvictimization and trauma in a national longitudinal cohort. *Dev Psychopathol*. 2007;19:149–166.
21. Fillingim RB, King CD, Ribeiro-Dasilva MC, et al. Sex, gender, and pain: a review of recent clinical and experimental findings. *J Pain*. 2009;10:447–485.
22. Popescu A, LeResche L, Truelove EL, et al. Gender differences in pain modulation by diffuse noxious inhibitory controls: a systematic review. *Pain*. 2010;150:309–318.
23. Riley JL III, Robinson ME, Wise EA, et al. Sex differences in the perception of noxious experimental stimuli: a meta-analysis. *Pain*. 1998;74:181–187.
24. Tolin DF, Foa DB. Sex differences in trauma and posttraumatic stress disorder: a quantitative review of 25 years of research. *Psychol Bull*. 2006;132:959–992.
25. Green BL, Chung JY, Daroowalla A, et al. Evaluating the cultural validity of the stressful life events screening questionnaire. *Violence Against Woman*. 2006;12:1191–1213.
26. Davidson JRT. *Davidson Trauma Scale*. New York: Multi-Health Systems; 1996.
27. Goolkasian PA. ROC analysis of pain reactions in dysmenorrheic and non-dysmenorrheic women. *Percept Psychophys*. 1983;34:381–386.
28. Hapidou EG, De Catanzaro D. Sensitivity to cold pressor pain in dysmenorrheic and non-dysmenorrheic women as a function of menstrual cycle phase. *Pain*. 1988;34:277–283.
29. Edens JL, Gil KM. Experimental induction of pain: utility in the study of clinical pain. *Behav Ther*. 1995;26:197–216.
30. Goodman L, Corcoran C, Turner K, et al. Assessing traumatic event exposure: general issues and preliminary findings for the Stressful Life Events Screening Questionnaire. *J Trauma Stress*. 1998;11:521–542.
31. Vázquez C, Pérez-Sales P, Matt G. Post-traumatic stress reactions following the March 11, 2004 terrorist attacks in a Madrid Community sample: a cautionary note about the measurement of psychological trauma. *Span J Psychol*. 2006;9:61–74.
32. Davidson JRT, Book SW, Colket JT, et al. Assessment of a new self-rating scale for posttraumatic stress disorder. *Psychol Med*. 1997;27:153–160.
33. Bobes J, Calcedo-Barba A, García M, et al. Evaluación de las propiedades psicométricas de la versión española de cinco cuestionarios para la evaluación del trastorno de estrés postraumático [Psychometric properties of the Spanish versions of five questionnaires assessing posttraumatic stress disorder]. *Actas Esp Psiquiatr*. 2000;28:207–218.
34. Davidson JRT, Tharwani HM, Connor KM. Davidson Trauma Scale (DTS): normative scores in the general population and effect sizes in placebo-controlled SSRI trials. *Depress Anxiety*. 2002;15:75–78.
35. Spitzer RL, Williams JB, Aibbon M, et al. *Structured Clinical Interview for DSM-III-R (SCID)*. New York: Biometrics Research Department; 1990.
36. Bernstein-Carlson E, Putnam FW. An update on the Dissociative Experiences Scale. *Dissociation*. 1993;6:16–27.
37. Van Ijzendoorn MH, Schuengel C. The measurement of dissociation in normal and clinical populations: meta-analytic validation of the Dissociative Experiences Scale (DES). *Clin Psychol Rev*. 1996;16:365–382.
38. Icarán E, Colom R, Orengo-García F. Estudio de validación de la escala de experiencias disociativas con muestra de población española [Validation of the Dissociative Experiences Scale in a Spanish population]. *Actas Luso Esp Neurol Psiquiatr*. 1996;24:7–10.
39. Marshall GN, Orlando M, Jaycox LH, et al. Development and validation of a modified version of the Peritraumatic Dissociative Experiences Questionnaire. *Psychol Assess*. 2002;14:123–134.
40. Bernstein A, Zvolensky MJ, Feldner MT, et al. Anxiety Sensitivity Taxon and trauma: discriminant associations for posttraumatic stress and panic symptomatology among young adults. *Depress Anxiety*. 2005;22:138–149.
41. Zvolrensky MJ, Forsyth JP, Bernstein A, et al. A concurrent test of the Anxiety Sensitivity Taxon: its relation to bodily vigilance and perception of control over anxiety-related events in a sample of young adults. *J Cogn Psychol*. 2007;21:72–90.
42. Peterson RA, Plehn K. Measuring anxiety sensitivity. In: Taylor S, ed. *Anxiety Sensitivity: Theory, Research, and Treatment of the Fear of Anxiety*. Mahwah, NJ: Lawrence Erlbaum Associates; 1999:61–81.
43. Sandín B, Chorot P, McNally RJ. Validation of the Spanish version of the Anxiety Sensitivity Index in a clinical sample. *Behav Res Ther*. 1996;34:283–290.
44. Hayes SC, Strosahl K, Wilson KG, et al. Measuring experiential avoidance: a preliminary test of a working model. *Psychol Rec*. 2004;54:553–578.
45. Barraca J. Spanish adaptation of the Acceptance and Action Questionnaire (AAQ). *Int J Psych Psychol Ther*. 2004;4:505–515.
46. Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas*. 1977;1:385–401.
47. Lewinsohn PM, Roberts RE, Seeley JR, et al. The Center for Epidemiologic Studies Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. *Psychol Aging*. 1997;12:277–287.
48. Soler J, Pérez-Sola V, Puigdemont D, et al. Estudio de validación del Center for Epidemiologic Studies-Depression (CES-D) en una población española de pacientes con trastornos afectivos [Validation of the Center for Epidemiologic Studies Depression Scale (CES-D) in an Spanish population of patients with mood disorders]. *Actas Luso Esp Neurol Psiquiatr*. 1997;25:243–249.
49. Vázquez FL, Blanco V, López M. An adaptation of the Center for Epidemiologic Studies Depression Scale for use in non-psychiatric Spanish populations. *Psychiatry Res*. 2007;149:247–252.
50. Spielberger CD, Gorsuch RL, Lushene RE. *STAI: Manual for the State-Trait Anxiety Inventory (Self-Evaluation Questionnaire)*. Palo Alto, CA: Consulting Psychologist Press; 1970.
51. Spielberger CD, Gorsuch RR, STAI Lushene RE. *Cuestionario de Ansiedad Estado/Rasgo [State/Trait Anxiety Inventory]*. Madrid: TEA; 1982.
52. Tascón MM, Esteve R. La interacción entre la distracción y el catastrofismo en la respuesta al dolor. In: Giménez A, ed. *Comportamiento y Palabra [Behavior and Word]*. Málaga: Universidad de Málaga; 2005:119–121.
53. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale. Development and validation. *Psychol Assess*. 1995;7:524–532.
54. Dixon KE, Thorn BE, Ward LC. An evaluation of sex differences in psychological and physiological responses to experimentally-induced pain: a path analytic description. *Pain*. 2004;112:188–196.
55. Edwards RR, Campbell CM, Fillingim RB. Catastrophizing and experimental pain sensitivity: only in vivo reports of catastrophic cognitions correlate with pain responses. *J Pain*. 2005;6:338–339.
56. Wolff B. Methods of testing pain mechanisms in normal man. In: Wall PD, Melzack R, eds. *Textbook of Pain*. Edinburgh: Churchill Livingstone; 1984:186–194.
57. Keogh E, Witt G. Hypoalgesic effect of caffeine in normotensive men and women. *Psychophysiology*. 2001;38:886–895.

58. Nouwen A, Cloutier C, Kappas A, et al. Effects of focusing and distraction on cold pressor-induced pain in chronic back pain patients and control subjects. *J Pain*. 2006;7:62–71.
59. Croft RJ, Gonsalvez CJ, Gander J, et al. Differential relations between heart rate and skin conductance, and public speaking anxiety. *J Behav Ther Exp Psychiatr*. 2004;35:259–271.
60. Tabachnick BJ, Fidell LS. *Using Multivariate Statistics*. New York: Hapercollins; 1996.
61. Erdflelder E, Faul F, Buchner A. GPOWER: a general power analysis program. *Behav Res Methods*. 1996;28:1–11.
62. Fillingim RB, Edwards RR. Is self-reported childhood abuse history associated with pain perception among healthy young women and men? *Clin J Pain*. 2005;21:387–397.
63. Strigo IA, Simmons AN, Matthews SC, et al. Neural correlates of altered pain response in women with posttraumatic stress disorder from intimate partner violence. *Biol Psychiatry*. 2010;68:442–450.
64. Liberzon I, Taylor SF, Phan KL, et al. Altered central μ-opioid receptor binding after psychological trauma. *Biol Psychiatry*. 2007;61:1030–1038.
65. Lang AJ, Kennedy CM, Stein MB. Anxiety sensitivity and PTSD among female victims of intimate partner violence. *Depress Anxiety*. 2006;16:77–83.
66. Asmundson GJG, Stapleton JA. Associations between dimensions of anxiety sensitivity and PTSD symptom clusters in active-duty police officers. *Cogn Behav Ther*. 2008;37:66–75.
67. Kiliç E, Kiliç C, Yilmaz S. Is anxiety sensitivity a predictor of PTSD in children and adolescents? *J Psychosom Res*. 2008;65:81–86.
68. Halligan SL, Michael T, Clark DM, et al. Posttraumatic stress disorder following assault: the role of cognitive processing, trauma memory, and appraisals. *J Consult Clin Psychol*. 2003;71:419–431.
69. Berman NC, Wheaton MG, McGrath P, et al. Predicting anxiety: the role of experiential avoidance and anxiety sensitivity. *J Anxiety Disord*. 2010;24:109–113.
70. Leeuw M, Goossens MEJB, Linton SJ, et al. The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *J Behav Med*. 2007;30:77–94.
71. Orr SP, Meyerhoff JL, Edwards JV, et al. Heart rate and blood pressure resting levels and responses to generic stressors in Vietnam veterans with posttraumatic stress disorder. *J Trauma Stress*. 1998;11:155–164.
72. Stam R. Review PTSD and stress sensitisation: a tale of brain and body. Part 1: human studies. *Neurosci Biobehav Rev*. 2007;3:530–557.
73. Bender R, Lange S. Adjusting for multiple testing—when and how? *J Clin Epidemiol*. 2001;54:343–349.
74. Asmundson GJG, Katz J. Understanding the co-occurrence of anxiety disorders and chronic pain: state-of-the-art. *Depress Anxiety*. 2009;26:888–890.
75. Sachs-Ericsson N, Cromer K, Hernandez A, et al. Review of childhood abuse, health, and pain-related problems: the role of psychiatric disorders and current life stress. *J Trauma Dissociation*. 2009;10:170–188.

