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Punishing the Self: Post-Traumatic Guilt Mediates the Link Between Trauma and Deficient Pain Modulation

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Abstract: **Trauma survivors may suffer from post-traumatic stress disorder (PTSD), elevated post-traumatic guilt (PG), and alterations in the pain system. However, the association between PG and alterations in pain perception and modulation among trauma survivors has not been established, nor has the possible underlying role of PG. This longitudinal study investigated: 1) the unique contribution of PG in predicting pain perception and modulation, while controlling for PTSD symptoms; and 2) the mediating role of PG in explaining pain perception and modulation among torture survivors, above and beyond PTSD symptoms. Participants were 59 torture survivors and 44 age-matched controls. PG and PTSD symptoms were assessed in 2003 (T1). Heat-pain threshold, heat-pain tolerance, temporal summation of pain (TSP), and conditioned pain modulation (CPM) were measured 5 years later (T2). Torture survivors had elevated PG and PTSD symptoms, enhanced TSP, and reduced CPM, compared to controls. While PTSD predicted reduced pain tolerance and CPM, PG predicted increased pain tolerance. Moreover, PG mediated the associations between torture and (increased) pain threshold, pain tolerance, and TSP. It appears that PTSD and PG induce opposite effects on the pain modulation capacity of torture survivors, a dichotomy that may explain paradoxical pain responses among trauma survivors, as discussed.**

Perspective: ***This longitudinal study sheds light on the possible mechanisms underlying variations in pain perception and modulation among trauma survivors. PTSD and PG each mediated opposing pain modulation profiles, suggesting that individual responses to trauma, rather than the trauma itself, influence pain responses.***

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Torture is a highly devastating traumatic event. Individuals are often subjected to severe physical pain (eg, beating, burning, suspension, electric shock, among other methods) alongside isolation, humiliation, and other forms of psychological torture (eg,[46]) as a way to obtain information, as well as to inflict intimidation and punishment.[9] As a result, torture survivors often suffer from long-term mental[46] and somatic pathologies (eg,[35]).

The ramifications of the torture extend to the pain system, manifesting in high rates of chronic pain compared to the general population[10,29,55] and alterations in pain perception and modulation. For example, hyposensitivity or hypersensitivity to noxious stimuli has been demonstrated among torture survivors[43,55] as well as an impaired ability to modulate pain, manifested in enhanced temporal summation of pain (TSP) and eradicated conditioned pain modulation (CPM).[13] Post-traumatic stress disorder (PTSD), which is the most common psychiatric sequela of trauma, and torture, in particular,[12,18] may well underlie these alterations. Indeed, quantitative sensory testing has revealed an association between PTSD and reduced pain modulation capacity[14,15] and distinguished pain-related brain activations among individuals with PTSD.[20,39,53]

However, an individual's response to trauma is not limited to PTSD, rather it is multifaceted, consisting of other emotional responses that might shape pain perception.

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Post-traumatic guilt (PG), consistently documented among trauma survivors,[44] might serve as an additional mechanism underlying alterations in the pain system. Guilt reflects real or imagined transgressions wherein individuals believe that their action or inaction led to negative consequences.[56] PG denotes an "unpleasant feeling with accompanied belief (or beliefs) that one should have thought, felt, or acted differently" during the trauma.[33, p. 492] Notably, elevated PG has been recorded among survivors of various types of trauma,[44] including torture.[31] Furthermore, although PTSD and PG may both develop in the aftermath of trauma,[6,44] and self-blaming has been associated with negative mood and cognitions that are incorporated into PTSD,[2] PG and PTSD symptoms are viewed as distinguished phenomena; however, the link between them is evasive.[44]

Interestingly, guilt has been associated with pain in various contexts. For example, people engaged in deliberate self-injurious behaviors have been reported to use physical pain as a self-punishment strategy or as a distraction from guilty feelings.[8,24,38] In a recent study, the pain perceived during a cold-pressor task reduced the feeling of guilt among these individuals.[25] Nonself-injurious individuals who were asked to recall immoral behaviors showed an increase in guilt and were able to sustain their hands in ice water longer than participants who recalled "everyday interactions." Moreover, the former group reported a decrease in guilt after exposure to pain.[4] However, in another study, a nonsignificant association between guilt and pain tolerance was reported among individuals with obsessive compulsive disorder.[26]

Given that PG and alterations in pain perception are common among trauma survivors, and that there is a dearth of information on the link between them, further research is necessary. Among torture survivors in particular, PG may be central to their pain experience because deliberate physical pain was used as punishment. Whether PG in the aftermath of this severe trauma serves as a mechanism underlying pain perception and modulation among torture survivors is, thus, an unanswered question that will be studied here for the first time.

The purpose of this secondary analysis of data, collected in a longitudinal study among torture survivors was to investigate whether: 1) PG has a significant effect in predicting pain perception and modulation, above and beyond the effects of PTSD symptoms; and 2) PG mediates the relations between torture and pain perception and modulation, above and beyond the mediating effects of PTSD symptoms.

Methods

Participants

The present study is part of a large-scale longitudinal study of war captivity (for full details, see[50]). All participants were combatants in the 1973 Yom-Kippur War as part of their mandatory or reserve service, were young adults during the war (age range of 18–26 years), and in good physical condition. Fifty-nine participants were torture survivors, former prisoners of war (mean age 57.5 ± 3 years), and were severely tortured during their captivity, which ranged between 6 weeks and 9 months in the years 1973 to 1974 (for further information on their captivity and torture please, see[15]). Forty-four participants (58.8 ± 4 years) were not held captive and, therefore, not tortured. Torture survivors and controls were initially matched on sociodemographic and military variables. No differences were found between the 2 groups at the time of the present study with regard to religion ($\chi^2(2) = 1.82$, $P = .40$), income ($t(92) = 1.58$, $P = .12$), age ($t(94) = -1.28$, $P = .78$), health before the war ($\chi^2(1) = .28$, $P = .78$), marital status ($\chi^2(1) = .01$, $P = .60$), education ($\chi^2(3) = 4.33$, $P = .23$), substance abuse ($\chi^2(1) = .35$, $P = .34$), alcohol consumption ($\chi^2(1) = 1.98$, $P = .23$), and analgesic medications and/or psychiatric medications with potential analgesic effects ($\chi^2(1) = 2.23$, $P = .20$).

The participants of the present study were recruited out of the lists of veterans who took part in a prior study that was conducted in the year 1991 and included a total of 349 veterans. The present study collected data at 2 time points: in the year 2003 (T1) and 2008 (T2). The recruitment for the present study, for both T1 and T2 assessment periods, started by sending letters to the participants of the prior study, in which the present study's aims were detailed. This was followed by a phone call 2 to 3 weeks later, in which the participants received an oral explanation of the study's aims and procedures. Two hundred and twenty-seven veterans (121 torture survivors and 106 controls) agreed to participate in T1 and 103 veterans (59 torture survivors and 44 controls) agreed to participate in T2. Therefore, the present study included data from the 103 veterans who participated in both T1 and T2. All the participants signed an informed consent form after receiving a detailed explanation of the study's aims and procedures. The study was approved by the Tel-Aviv University Ethics Committee (Tel-Aviv, Israel).

General Procedure

In order to evaluate the temporal relations between PTSD/PG and pain perception and modulation, namely to conclude on a potential causal link between these variables, a longitudinal study was designed in which PTSD and PG were the predicting variables (evaluated in T1) and pain modulation indices were the dependent variables (evaluated in T2). This design took into consideration the relative stability of PTSD levels among the majority of the sample[50] and the lack of significant differences between T1 and T2 in possible confounding factors including the participants' religiosity ($Z = -5.8$, $P = .56$), income ($Z = -5.8$, $P = .56$), as well as psychiatric symptomatology ($t(67) = -.03$, $P = .98$). As mentioned above, the data were collected in 2 waves of the evaluations as follows: in T1, participants completed a battery of questionnaires assessing PG and PTSD symptoms and in T2, participants underwent quantitative sensory

testing in which pain perception and pain modulation were measured.

Sensory testing took place in a quiet room in which the temperature was maintained at 22 ± 2°C. Each subject participated in a single testing session that lasted about 2.5 hours. Testing started after the subjects underwent a training session, of which the results were discarded. Testing included the evaluation and a stimulus-response function for heat-pain, from which stimulation temperatures were extracted for subsequent testing as well as the measurement of the heat-pain threshold, heat-pain intolerance threshold, TSP, and CPM. The order of tests was semirandom (the stimulus-response function always preceded the testing of TSP and CPM) with 2- to 5-minute breaks between each test. The stimulator probe was moved within and between tests.

Equipment

Thermal Stimulator

Contact heat stimuli were delivered using a Peltier-based computerized thermal stimulator (TSA 2001, Medoc Ltd., Ramat-Ishay, Israel), with a 3 × 3 cm contact probe. The passage of a current through the Peltier element produces temperature changes at rates determined by an active feedback system. As soon as the target temperature is attained, the probe temperature actively reverts to a preset adaptation temperature by the passage of an inverse current.

Hot-Water Bath

Hot stimuli were administered with a water bath apparatus (Chillsafe 8/30 Scanvac SHC 2000, Labotal, Israel). This temperature-controlled water tank has a maximum temperature variance of ±.5°C. Water flow is maintained at a constant temperature throughout the water bath by way of a whirlpool and is constantly reticulated to prevent local warming or cooling. The temperature of the water was kept constant at 45°C. Participants were instructed to insert their hand into the bath up to their wrist.

Semmes-Weinstein Monofilaments

Mechanical stimuli were administered with Semmes-Weinstein monofilaments. The kit is comprised of 20 calibrated monofilaments, with sizes ranging between 1.65 and 6.65 units. Each filament is attached to a plastic handle. Vertical pressure applied with the handle induces a calibrated force ranging between .008 and 300 g, respectively.

Visual Analog Scale

Perceived pain intensity was evaluated with a visual analog scale (VAS). The VAS consisted of a 15-cm plastic ruler with a slider in the middle. Moving the slider exposes a horizontal red bar (the visual side) to the subject, while the side facing the experimenter displays an analog scale with values between 0 and 10. The end points are set as "no pain sensation" (0) and "the most intense pain sensation imaginable" (10).

Measures

PTSD-Inventory (PTSD-I)

PTSD symptoms were assessed via the PTSD-I, a well-validated, 17-item, self-report questionnaire. The items on the PTSD-I correspond to the DSM-IV-TR diagnosis for PTSD.[49] Respondents rated symptoms suffered in the previous month relating to their experiences of war torture or combat on a scale ranging from 0 (not at all) to 4 (almost always). The number of positively endorsed symptoms was calculated by the items answered as 3 (often) or 4 (almost). The DSM-IV-TR was the most recent edition to which the PTSD-I could be adequately adapted in order to be used comparatively with criteria from the time that the study was initiated.[51] The scores of PTSD-I could be dichotomized (PTSD, no PTSD) using the *DSM–IV–TR* PTSD criteria. A respondent was considered to have PTSD if he endorsed at least 1 intrusive, 3 avoidant, and 2 arousal symptoms. The PTSD-I has proven psychometric properties with good convergent validity.[49,51] In the present study, Cronbach's alpha was .95.

Trauma-Related Guilt Inventory (TRGI)

PG was assessed via the TRGI,[33] a well-validated, 32-item, self-report questionnaire that measures the affective and cognitive components of trauma-related guilt. The TRGI is made up of 3 scales: distress (eg, "What happened causes a lot of pain and suffering"), global guilt (eg, "I experience intense guilt related to what happened"), and guilt cognitions (eg, "I was responsible for causing what happened").

Respondents rate their feelings about the statements on a 5-point Likert scale, ranging from 0 (not at all true) to 4 (extremely true). Psychometric studies in samples of domestic violence survivors and combat veterans yielded adequate construct- and criterion-related validity.[33] Additionally, the TRGI was found to have high convergent and discriminant validity as well as good internal consistency.[33] In this study, for reasons of parsimony, we computed a total guilt score, which was the mean score of all items. In the present study, Cronbach's alpha was .91.

Pain and Intolerance Thresholds

Heat-pain and heat-pain intolerance thresholds were measured to evaluate the sensitivity range of the pain system. The thresholds were measured with the method of limits using the thermal stimulator, as described in our previous study.[15] In short, the contact probe of the stimulator was attached to the skin of the participants' volar surface of the forearm. For heat-pain threshold measurement, participants received 4 successive gradually increasing heat stimuli at a rate of 2°C/s, starting from a baseline temperature of 35°C, and an interstimulus interval of 30 seconds. During

each stimulus, the participant was asked to press a switch when the first pain sensation was perceived, thus defining the heat-pain threshold. Heat-pain threshold was computed by averaging the readings of 4 successive trials. For the heat-pain intolerance threshold, we repeated the same stimulation protocol; however, the participant was asked to press the switch when pain could no longer be endured. Heat-pain intolerance threshold was computed by averaging the readings of 4 successive trials.

TSP

TSP, the gradual increase in pain during repeated noxious stimuli of identical intensity, was measured as an indication of the magnitude of central pain excitability.[42] Mechanical TSP was measured with a Semmes-Weinstein monofilament no. 6.65 (300 g), as described in our previous study.[15] In short, the examiner applied the filament 4 consecutive times, at 3-second intervals, to the palmar surface of the middle finger. The participant was asked to rate the pain intensity following the first and fourth stimulus on a 0 to 10 VAS. TSP was present if the fourth stimulus produced more pain than the first. The amount of TSP was calculated by subtracting the VAS rating of the first stimulus from that of the fourth. A greater increase in the VAS rating represented a greater magnitude of TSP and, hence, the enhanced level of central excitability.

Stimulus Response Function of Heat-Pain

A stimulus-response function was created for each subject in order to extract the stimulation temperatures for CPM measurement. Subjects received a series of thermal stimuli delivered with the thermal stimulator at an ascending order and were asked to rate their pain using the VAS. The stimuli rose from a baseline temperature of 32°C (increased at a rate of 2°C/s) to a destination temperature ranging between 38°C to the intensity eliciting 6 on the VAS, at which it remained for 1 second and then returned to baseline. The stimulator probe was moved after each stimulus. From the individual stimulus-response functions, the temperatures eliciting a value of 5 on the VAS were extracted.[15]

CPM

CPM was measured as an indicator of pain inhibition capacity.[58,61] CPM was measured in a parallel design, as described previously,[15] by administering a noxious contact-heat stimulus on the volar aspect of one forearm ("test stimulus") and evaluating its perceived pain intensity when administered alone and in the presence of another noxious heat stimulus applied to the contralateral hand ("conditioning stimulus"). The test stimulus was noxious heat equivalent to an intensity of 5 on the VAS (individually adjusted according to a stimulus-response function) applied with the thermal stimulator at its destination temperature for the duration of 10 seconds. The conditioning stimulus was the immersion of the hand into a bath of hot water, at 46°C, for the duration of 30 seconds. The second application and rating of the test stimulus, in the presence of the conditioning stimulus, was performed after 20 seconds of immersion time. CPM was present if the test stimulus produced less pain in the presence of the conditioning stimulus than in its absence. The difference between these 2 ratings was the amount of CPM.[15]

Data Analysis

Overall, 0% to 31.1% of data were missing across waves and variables, as follows: 0% to 12% of the data regarding pain measurement were missing due to technical issues during testing and 20% to 31.1% of the data regarding PTSD symptoms and PG were missing due to the improper completion of the questionnaire. In order to decide whether the data had missing values in a random pattern, we analyzed the differences between participants with missing values and those without missing values in all of the variables, using Little's Missing Completely at Random (MCAR) test.[11] The analysis revealed that the data were missing completely at random and lacked pattern bias ($X^2(61) = 66.836$, $P = .284$). Nevertheless, we decided to use the more advanced method of maximum likelihood (ML) via SPSS 25. Compared to the conventional methods, such as the arithmetic mean, listwise or pairwise deletion, the ML method was recommended as an optimal method for attrition.[11]

First, Pearson analyses were conducted to explore the correlations between the variables among each of the study groups. Next, we conducted a series of one-way Analyses of Variance (ANOVAs), in order to compare torture survivors and controls in PG, PTSD symptoms, and pain measurements. The study groups were the independent variable, and PG, PTSD symptoms, and pain measurements scores were the dependent variables.

We conducted a series of linear hierarchical regressions in order to examine the unique contribution of PG in predicting pain perception and modulation above and beyond PTSD symptoms. The models included 2 steps: the first step consisted of PTSD symptoms and the second step consisted of PG. To assess the mediating role of PG within the association between torture and pain perception and modulation, above and beyond PTSD symptoms as mediator, we used a bootstrapping method with 1,000 bootstrap resamples in PROCESS (model 4).[41] Bootstrapping is a nonparametric method that generates an estimate of the indirect effect, including a 95% confidence interval. When zero is not in the 95% confidence interval, one can conclude that the indirect effect is significantly different from zero at $P < .05$ (2-tailed). Thus, the effect of the independent variable on the dependent variable is mediated by the proposed mediating variable.[41] We referred to PG and PTSD symptoms as mediators to assess the strength of the mediating role of PG, beyond the effects of PTSD symptoms as a mechanism. All the variables' scores were standardized.

Table 1. Means, Standard Deviations, and Univariate F Results of PG, PTSD Symptoms, and Pain Measurements Among Torture Survivors and Controls

	Torture Survivors (n = 59)		Controls (n = 44)		
	M	*SD*	*M*	*SD*	*F(1, 101)*
Post-traumatic guilt	1.30	.48	.92	.40	18.18†
PTSD symptoms	9.71	3.84	4.14	4.36	47.15†
Heat-pain threshold	45.30	3.19	44.83	4.11	.42
Heat-pain tolerance	49.82	1.96	49.99	1.97	.21
TSP	1.24	1.58	.35	.98	10.86*
CPM	−57	1.80	1.01	2.09	17.11†

*$P < .01$.
†$P < .001$.

Results

Table 1 presents means, standard deviations, and univariate F results of PG and PTSD symptoms, and pain measurements compared between the study groups. A significant group effect was found for PG and PTSD symptoms; torture survivors had elevated levels compared to controls. A significant group effect was also found on TSP and CPM; torture survivors had greater TSP and reduced CPM than controls indicating that torture survivors had poorer ability than controls to modulate pain. The differences between the study groups in pain threshold and pain intolerance were nonsignificant.

Table 2 presents the correlations between the study's variables for each group separately. As can be seen, PG levels significantly correlated with PTSD symptoms among both groups, the higher the PG level the higher the PTSD symptoms level. PG levels did not correlate with the pain measurements except for a correlation between PG and heat-pain threshold among torture survivors only, with higher levels of PG associated with higher heat-pain threshold.

Table 3 presents the results of the linear hierarchical regressions predicting pain perception and modulation indices by PG, controlling for PTSD symptoms. In step 1, which consisted of PTSD symptoms alone, PTSD symptoms were found to significantly predict CPM; the higher the PTSD symptoms, the lower the CPM magnitude. Step 2, which consisted of both PTSD symptoms and PG, revealed the unique contribution of PTSD symptoms and PG in predicting pain perception and modulation. Results of this step indicated that both PG and PTSD symptoms had a significant effect in predicting heat-pain tolerance, albeit in an opposite manner; whereas elevated PG predicted higher heat-pain intolerance, elevated PTSD symptoms predicted lower heat-pain intolerance. With regard to the pain modulation indices, only PTSD symptoms alone had significant effects in predicting CPM and TSP; elevated PTSD symptoms predicted higher TSP magnitude and lower CPM magnitude. The effect of PG on CPM and TSP were nonsignificant.

Bootstrapping solutions and unstandardized coefficients are presented in Table 4 and Figs1 to 3. The analyses indicated that PG alone mediated the associations between torture and heat-pain threshold and TSP. Being a torture survivor predicted elevated PG, which in turn, predicted higher heat-pain threshold (Fig 1),

Table 2. Inter-Correlations Among the Study Variables Separated by Group

Measure	1	2	3	4	5	6
1. Post-traumatic guilt						
Torture survivors	-					
Controls	-					
2. PTSD symptoms						
Torture survivors	.58†	-				
Controls	.65†	-				
3. Heat-pain threshold						
Torture survivors	.28*	.12	-			
Controls	.16	.18	-			
4. Heat-pain tolerance						
Torture survivors	.25	.02	.52†	-		
Controls	.15	−.10	.59†	-		
5. TSP						
Torture survivors	−.21	−.14	−.14	−.37†	-	
Controls	−.04	.11	.14	−.23	-	
6. CPM						
Torture survivors	.15	.16	−.10	−.02	−.07	-
Controls	−.02	−.20	−.27	−.06	−.23	-

*$P < .05$.
†$P < .001$.

Table 3. Regression Beta Standardized Coefficients Predicting Pain Perception and Modulation by PG Controlling for PTSD Symptoms (n = 103)

	Heat-Pain Threshold		Heat-Pain Tolerance		TSP		CPM	
	B	R^2 change	β	R^2 change	β	R^2 change	β	R^2 change
Step 1								
PTSD symptoms	.16	.03	−.05	.00	.14	.02	−.23*	.05*
Step 2								
PTSD symptoms	.01	.03	−.31*	.08†	.27*	.02	−.31*	.01
Post-traumatic guilt	.23		.39†		−.21		.13	

*$P < .05$.
†$P < .01$.

Table 4. Unstandardized Regression Coefficients and Bootstrap 95% Confidence Intervals for Predicting Pain Indices (Evaluated at T2) by Study Group Through PG and PTSD Symptoms (Evaluated at T1) (n = 103)

	Heat-Pain Threshold		*Heat-Pain Tolerance*		*TSP*		*CPM*	
	Bootstrap 95% Confidence Intervals	β	*Bootstrap 95% Confidence Intervals*	β	*Bootstrap 95% Confidence Intervals*	β	*Bootstrap 95% Confidence Intervals*	β
Direct	{−2.0047, 1.3971}	−.30	{−1.0066, .7718}	.11	{.3483, 1.6388}	.99†	{−2.4778, −.6256}†	1.55†
Indirect through PTSD symptoms (T1)	{−1.0354, −1.4737}*	.12	{−1.2373, −.1571}	−.65*	{−.4589, .6099}	.13	{−.9597, .2719}	−.25
Indirect through Post-traumatic guilt (T1)	{.0184, 1.6219}*	.64*	{.2426, 1.1192}	.59*	{−.5343, −.0173}*	−.24*	{−.0976, .6975}	.22

Note. 95% confidence intervals are presented in brackets. Confidence intervals that do not include 0 (null association) are significant.
*$P < .05$.
†$P < .01$.

and lower TSP magnitude (Fig 3). The analysis also indicated that both PG and PTSD symptoms mediated the associations between torture and heat-pain tolerance (Fig 2) yet with opposite effects. Specifically, being a torture survivor predicted both elevated PTSD symptoms and PG; however, whereas elevated PTSD symptoms predicted lower heat-pain tolerance, elevated PG predicted higher heat-pain tolerance. The mediating roles of PG and PTSD symptoms within the associations between torture and CPM were nonsignificant (not shown).

Discussion

This study aimed to test whether PG predicts pain perception and modulation as well as mediate the relations between torture and alterations in pain perception and modulation, above and beyond the effects of PTSD symptoms. The results revealed that PG predicted higher heat-pain tolerance. Although both PG and PTSD mediated the relations between torture and heat-pain tolerance and, therefore, served as the underlying mechanisms, they presented a contradictory effect. Whereas PTSD symptoms predicted *decreased* heat-pain tolerance, PG predicted *elevated* heat-pain tolerance. PG also served as a mechanism underlying torture survivors' elevated heat-pain threshold and lower TSP magnitude. It thus seems that PG influences on pain perception among torture survivors counteract the effects of PTSD symptoms.

Different aspects of war-related torture may induce guilt among survivors, even years after repatriation. Torture survivors might blame themselves for having been captured, disclosing confidential information during interrogations, or forming bonds with their captors in order to survive.[48] The significantly higher PG levels among the torture survivors than controls herein corroborate previous findings among survivors of other traumas, for example, combat, childhood sexual and physical abuse, and domestic abuse.[1,40] As expected PG correlated with PTSD, as frequently found among trauma survivors,[44] yet each had a unique effect on pain perception herein.

That PG mediated an increased heat-pain threshold, increased heat-pain tolerance, and reduced TSP magnitude suggests that PG may, overall, reduce responsiveness to the threshold and suprathreshold noxious stimuli (ie, hypoalgesia or "pain numbness"). We could not find studies assessing these relations among trauma survivors. However, there are studies on the link between guilt and pain perception among nontraumatized individuals. Our results coincide with Bastian et al,[4] showing that the induction of guilt by recalling immoral behaviors led students to leave their hands in ice water longer than the control condition. Interestingly, the students in this study and individuals who engaged in deliberate nonsuicidal self-harm behaviors reported that the sensation of pain reduced their guilty feelings.[8,24,28,38] The latter population, in particular, reported that physical pain during self-harm served to distract from or reduce guilt.[44,53,56]Although it is

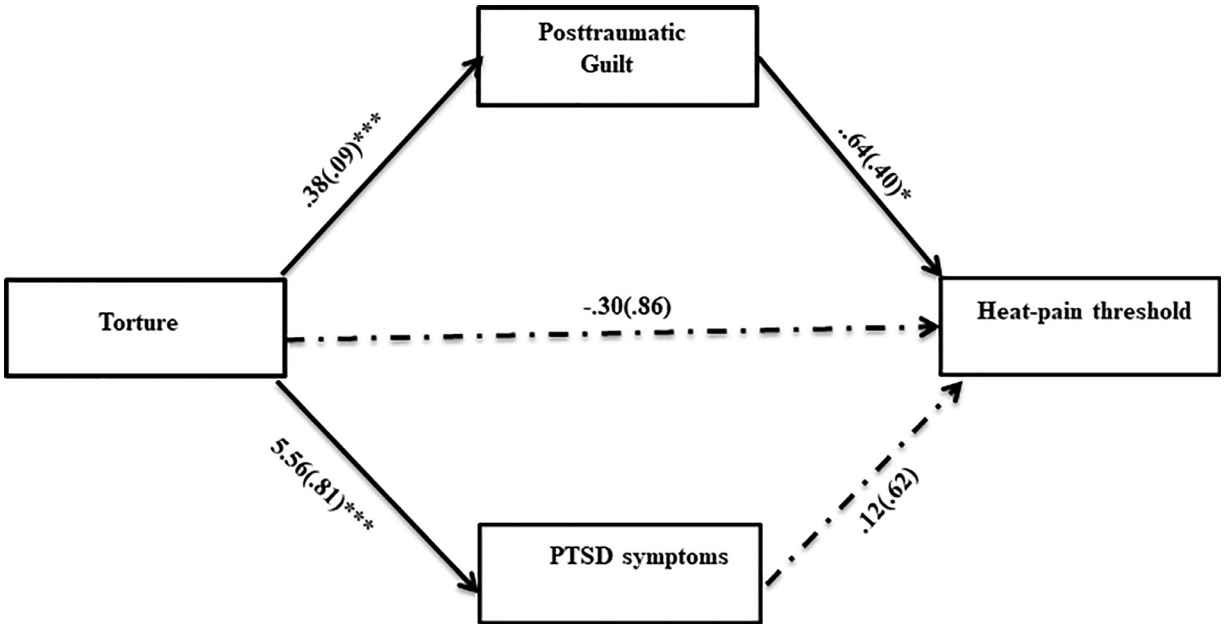

Figure 1. PG and PTSD symptoms as mediators in the relation between torture and heat-pain threshold. All coefficients represent unstandardized regression coefficients. Solid lines represent significant predictions. Dashed lines represent nonsignificant predictions. Value in the brackets represents standard errors (SE). Values of torture: 0 = controls, 1 = torture survivors.*$P < .05$, ***$P < .001$.

difficult to determine whether increased guilt underlies the hypoalgesia or pain numbness in the aforementioned studies, our longitudinal study suggests such a mechanism.

A recent systematic review[5] and meta-analysis[22] of imaging studies that investigated the neural correlates of guilt revealed significant brain activation in the prefrontal, temporal, and parietal regions, mainly in the left hemisphere. Further analysis revealed an association between guilt and activation in the anterior and dorsal cingulate, the left medial frontal gyrus, precuneus, and superior temporal gyrus and the right superior frontal gyrus. Interestingly, some of these brain structures have also been implicated in pain modulation. For example, the level of pain habituation and CPM have been linked to the activation in the anterior cingulate, dorsolateral prefrontal, and orbitofrontal cortices.[7,59,62] Furthermore, the functional connectivity between the frontoparietal network and the rostral anterior cingulate/medial prefrontal cortex has been positively associated with the cognitive modulation of pain.[30] Thus, the increased level of PG among torture survivors may

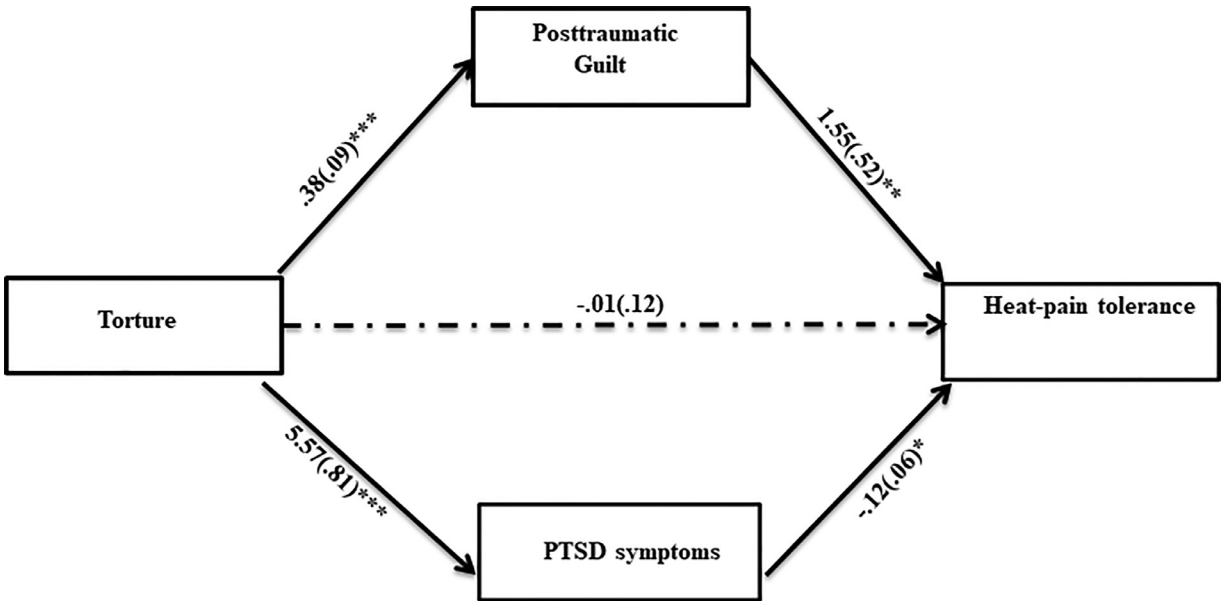

Figure 2. PG and PTSD symptoms as mediators in the relation between torture and heat-pain tolerance. All coefficients represent unstandardized regression coefficients. Solid lines represent significant predictions. Dashed lines represent nonsignificant predictions. Value in the brackets represents standard errors (SE). Values of torture: 0 = controls, 1 = torture survivors. *$P < .05$, **$P < .01$, ***$P < .001$.

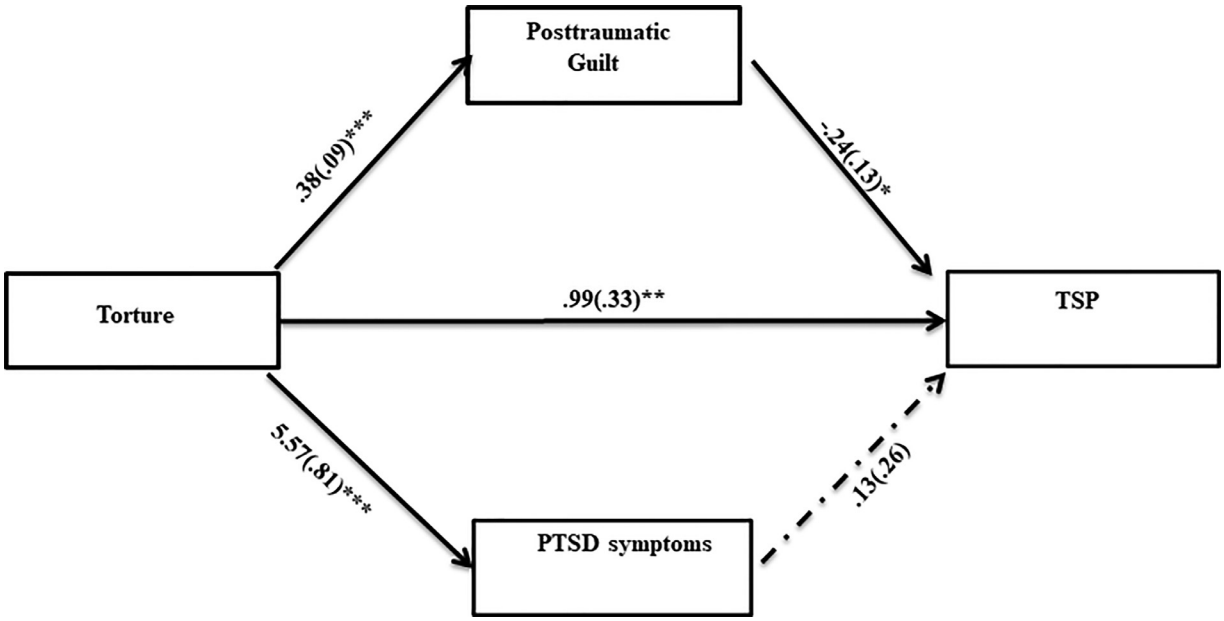

Figure 3. PG and PTSD symptoms as mediators in the relation between torture and TSP. All coefficients represent unstandardized regression coefficients. Solid lines represent significant predictions. Dashed lines represent nonsignificant predictions. Value in the brackets represents standard errors (SE). Explained variance is located above all dependent variables. *$P < .05$, ***$P < .001$ ***$P < .001$.

trigger cognitive and affective processing, which induces greater activation of the descending pain inhibition pathways, thereby promoting hypoalgesia.

Interestingly, despite the co-occurrence of PG and PTSD, PTSD symptoms mediated the opposite phenomenology, that is, lower pain tolerability and poorer CPM. PTSD following various traumas has been previously associated with increased or decreased responses to noxious stimuli[20,32,57] and reduced CPM.[16] In our previous study, PTSD related to shell shock and terror attacks was associated with a unique, paradoxical pain profile of hyper-responsiveness to noxious stimuli along with an increased pain threshold. However, while the former correlated with anxiety sensitivity, the latter correlated with the dissociation levels.[17] Thus, 2 seemingly opposing yet co-occurring facets of PTSD, enhanced and blunted arousal, respectively, were associated with contrary pain responses. Among nontraumatized individuals, acute stress manipulation also induced opposite effects, depending on the individual responses to the manipulation, while "high-stress responders" exhibited reduction in pain modulation, "low-stress responders" exhibited an improvement in pain modulation.[21] Therefore, it is possible that a strong negative affect, as in the case of enhanced PTSD symptomatology, impairs pain modulation and tolerability, while feelings of guilt counteract this effect. Evaluating the contributing role of arousal level within these effects is thus called for.

Painful stimuli might act as trauma reminders, particularly for survivors of traumatic events involving extreme pain, such as torture. Consequently, PTSD symptoms might fuel pain catastrophizing and negative emotional reactions to experimental pain,[3] which may reduce its tolerability.[23,45] PG, on the other hand, may be linked to more positive emotions and attitudes toward pain. Torture survivors who suffer from elevated PG might experience emotional relief, consequential to physical pain. Similar to individuals who engage in self-harm,[38] torture survivors might use physical pain as a distraction from their devastating feelings of guilt or as a means to regulate their guilt-driven distress and, thus, endure it more willingly. Previous findings showing that nontraumatized individuals perceived relative relief of experimental pain as generating positive feelings and a pleasant sensation,[36] and the associations between trauma exposure and deliberate self-harm[37] and between guilt, self-harm, and increased pain endurance (eg,[28]) lend further support to this line of thought.

Not mutually exclusive is the possibility that torture survivors with elevated guilt view physical pain as a way to receive expiation and, as a result, they do not fear the pain. It has been argued that people are socialized to link pain with punishment and to associate the infliction of pain with one's "bad" behavior (eg,[4]). Hence, pain not only signals guilt but also becomes an important medium to amend guilt. This could lead to decreased fear while facing noxious stimuli, thereby decreasing the sensitivity and responsiveness to noxious stimuli. Previous studies have indeed indicated an inverse correlation between fear of pain and pain threshold and tolerance as well as ratings of suprathreshold stimuli.[19,27,47]

PTSD symptoms in this study were associated with reduced pain tolerance, in contrast to previous studies (including our own).[17,32] This inconsistency may result from physical pain being inherent to torture and from the distinct implications PTSD symptoms may have on pain among torture survivors, compared to survivors of other traumatic events. Alternatively, this inconsistency may result from evaluating the effect of PTSD symptoms herein, while controlling for PG. This analysis was important given that PTSD and PG often co-occur and enabled

us to show the unique contributions of PTSD symptoms and PG on pain perception separately.

The literature reveals a controversy regarding the implications of guilt. Some argue that guilt is maladaptive, preventing the integration between the traumatic event and prior beliefs and enhancing avoidant coping strategies, which sustain PTSD symptomology.[34,52] Others claim it to be adaptive, as it atones for past transgressions and leads the individual to avoid this in the future.[54,60] In this study, PG was found to underlie reduced responsiveness to noxious stimuli—a beneficial effect in the lab setting. Nevertheless, the present results are not necessarily applicable to torture survivors' experience of everyday pain nor do they reveal the complete effects of these singular relations between PG and pain. It is unclear whether PG is related to "pain numbness" in other contexts. If it is, it is unclear whether this is adaptive in the long term or, alternatively, mirrors re-enactment of the trauma, wherein victims have to detach from their body, thereby preventing recovery. These questions remain unanswered and denote further study.

The results should be considered in light of several reservations. First, PTSD and PG were not recorded in close proximity to the release from captivity, which limits our ability to control their possible effect on pain prior to the study. Second, although PTSD levels and background variables were relatively stable among the majority of the sample,[50] the 5 years gap between T1 and T2 may increase the risk for additional potential confounders that could influence the results. Third, the sensory profile of torture survivors may have been influenced by medication use; however, this seems unlikely considering the similar rate of medications with the potential analgesic effect among the 2 groups. Forth, given that these findings were based on an entirely male sample, they may not be applicable for women, who are more often the victims of many types of trauma. Moreover, although trauma survivors share many similarities, torture may present with unique features that could limit the generalizability of the results to all trauma survivors. Given that the type of traumatic event might shape the way individuals experience pain and that PTSD has other co-morbidities, which could be associated with pain, future studies should explore the implications of PG and PTSD symptoms alongside other co-morbidities (eg, dissociation).

In summary, to the best of our knowledge, this is the first study in which the unique effects of PG versus PTSD on pain perception and modulation was investigated among trauma survivors. PG and PTSD co-occurred and both shaped pain perception albeit in opposite manners; PG appeared to underlie increased pain tolerability and modulation, while PTSD symptoms underlie reduced pain tolerability and modulation. These results emphasize the need to consider both PG and PTSD symptoms while treating survivors of traumatic events, especially those which involve physical pain (eg, survivors of physical abuse).

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