

Individual Differences in the Emotional Reaction to Shock Determine Whether Hypoalgesia Is Observed

Jamie L. Rhudy, PhD* and Mary W. Meagher, PhD†

*Department of Psychology, University of Tulsa, Tulsa, Oklahoma; †Department of Psychology, Texas A&M University, College Station, Texas

ABSTRACT

Objectives. Our laboratory has shown that electrical shocks induce fear in human participants and subsequently inhibit pain on the hand receiving shocks. The present study examined whether pain modulation is bilateral, by testing pain on the hand contralateral to the one receiving shocks. We also evaluated whether individual differences in emotional response to the shocks influenced pain modulation.

Design. Following baseline tests, 61 participants were randomly assigned to one of two groups. In the shock group, participants received three surprising electrical shocks. In the control group, stimulating electrodes were removed from participants' fingers, and no shocks were presented. Both groups received two more pain threshold tests 2 and 8 minutes later.

Outcome Measures. Similarly to the tail-flick test used in rodents, pain threshold was tested by measuring the latency of finger withdrawal to radiant heat. The following manipulation checks assessed the emotional state induced by shocks and the control procedure: Self-reported affect, skin conductance level (SCL), heart rate (HR), and blood pressure (BP).

Results. Surprisingly, self-reported affect data indicate that some participants reacted to the shocks with humor and fear, while others reacted primarily with fear. Therefore, these groups were analyzed separately. Participants reacting with fear only exhibited hypoalgesia; however, participants reacting with mixed fear and humor showed no change in pain. This divergent effect was not mediated by arousal, because SCL, HR, and BP were similar in both shocked groups (fear only, fear + humor).

Conclusions. These data suggest that fear-induced hypoalgesia occurs bilaterally. However, humor experienced concurrent with fear appears to inhibit hypoalgesia. These findings may help explain individual differences in pain following traumatic events.

Key Words. Fear; Humor; Emotion; Stress-Induced Analgesia; Heat; Shock

Introduction

Fear is a highly arousing negative affective state resulting from a present environmental threat (or cues associated with a threat). It produces an immediate alarm reaction that mobilizes an organism to take action (fight/flight) [1]. Because fear is

produced by situations that threaten the physical integrity of the organism, it would be to the organism's advantage to inhibit responses that might interfere with fight/flight, such as pain and its associated behaviors (e.g., withdrawal). Indeed, in their perceptual-defensive-recuperative (PDR) theory, Bolles and Fanselow argue that fear inhibits pain [2]. Supporting this, innumerable animal studies have demonstrated that stimuli (shock, predators, cues predicting threat) that produce fear-like behaviors also inhibit pain [2–12].

Reprint requests to: Jamie L. Rhudy, PhD, University of Tulsa, Department of Psychology, Lorton Nall 310, 600 South College, Tulsa, OK 74104. Tel: (918) 631-2839; Fax: (918) 631-2833; E-mail: jamie_rhudy@utulsa.edu.

Surprisingly, the phenomenon of fear-induced hypoalgesia has received little attention in humans. One potential reason could be the limited means of eliciting fear in a safe, ethical manner. However, recent human work in our laboratory [13–17] has sought to bridge this gap in the literature by using methods and parameters employed in animal studies demonstrating fear-induced hypoalgesia [7,11–12]. We have shown that three brief, surprising shocks elicit subjective and physiological reactions indicative of fear, and subsequently inhibit pain. These findings are consistent with data from other laboratories examining the impacts of a range of fear stimuli [18–22].

Despite recent progress in characterizing the pain modulatory effects of fear in humans, relatively little is known about the generality of this phenomenon and its underlying mechanisms. For example, it is unclear whether fear causes a diffuse, whole-body reduction in pain, or whether pain modulation is circumscribed to the area most likely to incur damage during the threatening situation. The central nervous system circuit most likely responsible for fear-induced changes in pain (amygdala-PAG-RVM-spinal cord [23–24]) is not somatotopically organized; thus, it would be expected that fear would cause diffuse hypoalgesia. However, some evidence suggests that psychological mechanisms can result in localized pain modulation in humans [25]. And, others have found that electrical shock can cause localized hypoalgesia in animals [26]. Our laboratory has demonstrated that hypoalgesia generalizes to other nonshocked fingers of the hand ipsilateral to fear-inducing shocks [14,16]; however, it is unknown whether fear-induced hypoalgesia is bilateral.

The present study examined whether fear-induced hypoalgesia occurs bilaterally. Like prior experiments [13–14,16], pain was tested before and after a fear-induction procedure (delivery of surprising electrical shocks). However, in the present study, pain was tested on the hand contralateral to the one receiving shocks. An unshocked (neutral) group served as the control. Further, pain was tested using a method analogous to the tail-flick test, a pain measure commonly used in rodents. Participants were told to remove their finger from a radiant heat source as soon as it became painful. The latency from heat onset to finger withdrawal was used as an indicator of pain threshold, the way tail-flick latency is used in rodents. If finger withdrawal latency is

greater following contralateral shocks, this would suggest that fear-induced hypoalgesia occurs bilaterally.

In addition, we examined whether individual differences in emotional reactivity to our putative fear stimulus altered its pain modulatory effects. Although threatening stimuli tend to elicit negatively valenced emotions such as fear, mixed emotional responses can occur. For example, a roller coaster ride can elicit concurrent positive excitement *and* negative fear states in some individuals, whereas others experience fear only. Little is known about the impact of mixed emotional states on pain. However, some evidence suggests that concurrent activation of positive and negative affect may result in a zero net effect on pain [27] and startle modulation [28]. In the present study, some participants unexpectedly reacted to the fear-inducing shocks with a mixed reaction (fear and humor) while others reacted with fear only. Therefore, analyses were conducted with three groups: Neutral (no shocks), shock (fear only), and shock (fear + humor). If positive affect inhibits the effects of negative affect (fear), as others have suggested [27–30], then it is possible that humor could inhibit fear-induced hypoalgesia.

It was hypothesized that contralateral shocks would elicit fear (i.e., negative affect with high arousal), resulting in higher self-reported negative affect (higher ratings on fear, anger, and negative valence) and lower self-reported positive affect (lower ratings of happiness and humor) than unshocked controls. Furthermore, it was expected that shocked participants would report greater arousal and show greater physiological arousal—higher skin conductance level (SCL), heart rate (HR), and blood pressure (BP). Fear-inducing shocks were expected to induce bilateral hypoalgesia, as indicated by greater finger withdrawal latencies in the shocked group than in the neutral, unshocked controls. However, mixed emotional reactions to the shock (fear and humor) would be expected to attenuate or block this effect [27–28].

Methods

Participants

Participants were 67 undergraduate psychology students who received course credit for participation. Two participants were excluded due to equipment problems and four were excluded because their baseline finger withdrawal latencies were at

the ceiling level (8 seconds)¹. Thus, 29 women and 32 men were included in the analyses. Of those, 75.4% were Caucasian, 3.1% were Hispanic, 6.6% were African American, and 4.9% were of other races. The mean age was 19.48 (± 2.49 SD) years. Potential participants were excluded if they had: Circulatory, cardiovascular, or neurological problems; chronic pain; recently used tobacco, analgesics, antidepressants, or alcohol; or experienced a recent trauma.

Apparatus and Physiological Recording

All data acquisition and stimulus presentation were computer controlled. SCL and HR sensors were attached to fingers of the nondominant hand and sampled at 50 Hz. BP was measured before and after the experiment using a digital meter (Model-E7622, Eckerd Drug Company, Largo, Fla.).

Electrocutaneous Stimulation

Fear stimuli were presented using a Grass S88 stimulator with a transformer isolation unit and a constant current unit. Two electrodes were spaced approximately 1.5 cm apart and attached to the proximal, dorsal surface of the participant's nondominant index finger. Three brief shocks were delivered at 200 Hz, with a 1-ms pulse duration and a 0.75-s train duration, at 12.4 mA.

Radiant Heat Pain

Radiant heat pain was tested similarly to experiments in rodents. To do so, a radiant heat device was constructed that focused light from a 300-W projector bulb onto the blackened distal digit of the participant's dominant hand's index finger on the palmar surface. Participants were instructed to remove their finger as soon as the heat became painful. Latency from heat onset to finger withdrawal was calculated by the computer controlling the experiment. This latency was used as the indicator of pain threshold. To avoid tissue damage an automatic 8-second cutoff was used. Visual and auditory cues predicting the light's onset were minimized by having participants focus their eyes

on a target placed on the wall and by the use of sound attenuating headphones.

Manipulation Checks

The self-efficacy to reduce pain (SE-PR) scale assesses an individual's belief that they can influence their pain [13]. This measure consists of 15 items ranging from 0–10. Items are summed, with higher scores indicating greater self-efficacy. The Center for Epidemiological Studies depression scale (CES-D) [31] was used to measure preexisting emotional distress. Scores ranged from 0–60, with higher scores indicating greater distress. Scores greater than or equal to 16 are used to indicate clinically significant distress [31].

After every heat test, participants rated their pain intensity and unpleasantness on two 100-mm visual analog scales (VASs), ranging from 0 (no pain sensation) to 100 (the most intense pain sensation imaginable) and 0 (not at all unpleasant) to 100 (the most unpleasant imaginable). These scores were used as manipulation checks to ensure that participants were removing their finger at the same pain criterion point (i.e., pain threshold) across all radiant heat tests. If participants' subjective criteria did not change, then VAS scores should not vary significantly across trials or between groups.

HR, SCL, and BP were collected to confirm that shocks elicited increased autonomic arousal. Change scores were calculated by subtracting individual level baseline scores from data collected during and after emotion induction. At the end of the experiment, participants rated their affective reaction to the emotion-induction procedure using the Self-Assessment Manikin (SAM) [32] and affective descriptors. Using the SAM, participants rated subjective valence (0 "unhappy" to 9 "happy") and arousal (0 "calm" to 9 "excited"). Affective descriptors were 45 emotion words with Likert-type scales ranging from 0 (not at all) to 4 (strongly). To reduce the number of analyses, five subscales were created by taking the mean from groups of affective descriptors generated by previous research [16]. The five subscales, the affective descriptors (in parentheses) that were used to derive them, and the internal consistencies (using Cronbach's α) for the current sample were: 1) Fear (worried, scared, afraid, fear, nervous, frightened, dread, tense, panicky, uneasy, on edge, restless, helpless, jumpy, anxious, apprehensive, relaxed, nauseated), $\alpha = 0.93$; 2) Anger (spiteful, furious, desperate, hostile, annoyed, ready to fight, resentful, revolting, irritated, angry, disgusted), $\alpha = 0.93$; 3) Humor (funny, humorous, hilarious, lively, energetic, invigorated), $\alpha = 0.95$; 4) Happiness (pleased, cheerful, happy,

¹ All six participants were excluded from the shock condition. However, this is likely due to random factors and not an effect of the shock condition itself. The bulb on the radiant heat device failed and had to be replaced causing the exclusion of two participants. The other four participants were excluded because their baseline finger withdrawal latencies were at ceiling. Because participants were unaware of their experimental condition during baseline testing, it is unlikely that their condition influenced these reactions.

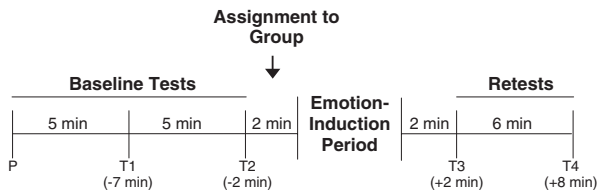


Figure 1 Experimental procedure. After a practice test (P), participants underwent two baseline finger withdrawal tests (T1 and T2). Then, participants were randomly assigned to either shock or control. Two and 8 minutes following shocks (or an equivalent period for the control group) participants received further pain tests (T3 and T4, respectively). Prior to every heat test and during emotion induction, 1 minute of physiological data was recorded.

joyous, elated, jovial, comforting), $\alpha = 0.91$; 5) Fatigue (bored, tired, fatigued), $\alpha = 0.85$. Thus, scores on each subscale (fear, anger, humor, happiness, and fatigue) ranged from 0 to 4.

Procedure

Figure 1 illustrates the experimental procedure. After obtaining informed consent, participants were acclimated for 15 minutes while they completed demographics, SE-PR, CES-D, and BP measures, and the physiological sensors (HR, SCL) and stimulating electrodes were attached. After baseline pain testing, participants were randomly assigned to one of two emotion-induction conditions². In the fear condition ($N = 35$), stimulating electrodes were plugged into the stimulator and participants were told, “You may or may not receive brief, surprising, and painful shocks.” This group received shocks. Participants in the neutral condition ($N = 26$) were told, “You will not receive any electrical stimulations,” and electrodes were removed. After emotion induction, two follow-up constant heat tests were conducted at 2 and 8 minutes following shocks (or an equivalent time for controls). After all pain testing was completed, participants rated their reaction to the emotion-induction condition, and BP was reassessed. All procedures were approved by Texas A&M University’s Internal Review Board.

Analyses

Based on the recommendations of Hack and McLean, change scores were used in all analyses where baseline scores were collected, rather than use the baseline scores in the repeated measures

² It was assumed that the effect of fear might be smaller on the contralateral hand; therefore, it was decided to randomly assign a greater number of participants to the shock condition.

analyses [33]. These scores were generated by subtracting individual level baseline values from those collected during emotion induction (SCL and HR) and postinduction (pain, SCL, HR, BP). Repeated measures mixed analyses of variance (ANOVA) were conducted on VAS, pain threshold, SCL, and HR data, with gender and condition as between-individual factors and trial as a within-individual variable. For high sphericity, the Greenhouse-Geisser correction was used where ϵ is noted. Analyses for self-efficacy, distress, SAM–valence, SAM–arousal, affect descriptor subscales, and BP were conducted using 2 (gender) \times 3 (condition) ANOVA. To examine main effects, Tukey least significant difference tests were used. Interactions were decomposed using two-tailed t -tests, unless effects were in the predicted direction, then one-tailed t -tests were used. Significance was set at $P < 0.05$. No three-way interactions were significant in any analyses.

Results

Initial examination of self-reported affect indicated that some shocked participants reacted with humor (score on humor subscale >0), causing reports of humor to be higher in the fear group than the neutral group ($Mean = 0.90$ vs $Mean = 0.20$; $P < 0.05$). Because prior research suggests that concurrent activation of positive and negative affect may result in different outcomes than positive or negative affect alone [27–28], the shocked group was split into fear only ($N = 14$) and fear + humor ($N = 21$) groups. To do so, participants in the shock group that had a score on the humor subscale greater than zero were placed in the fear + humor group, others were left in the fear only group³.

³ The strategy to examine the effect of humor as a dichotomous, rather than continuous, variable was chosen for theoretical and statistical reasons. First, Lang’s motivational priming hypothesis [28] suggests that activation of the appetitive system (associated with positive affect) should inhibit or cancel out effects of defensive activation (associated with negative affect). Therefore, it is predicted that *any* experience of positive affect (humor) should negate the effects of fear (activation of the defensive system). So, it does not necessarily follow that high levels of humor would necessarily lead to greater inhibition of fear-induced hypoalgesia than low levels of humor. Thus, taking a continuous, correlational approach would not accurately capture the effect of humor. Moreover, many of the shocked participants reacted with no humor (humor subscale = 0) or little humor (humor subscale <1). This resulted in a highly skewed distribution of humor scores that could not have been corrected using a transformation. Thus, it would have been inappropriate to examine the effect of humor in a continuous manner.

Table 1 Means (standard errors) of pain data

Condition	VAS-intensity (0-100)				VAS-unpleasantness (0-100)				Change in finger withdrawal latency (seconds)
	Baseline		Retest		Baseline		Retest		
	T1	T2	T3	T4	T1	T2	T3	T4	
Neutral (N = 26)	26.32 (2.87)	27.70 (3.08)	29.34 (3.10)	29.30 (3.29)	25.56 (3.16)	24.44 (3.26)	27.20 (3.45)	26.20 (3.57)	-0.04 (0.15)
Shock: Fear + humor (N = 21)	34.97 (3.19)	34.05 (3.42)	33.42 (3.44)	32.90 (3.65)	33.88 (3.45)	31.14 (3.56)	30.89 (3.77)	31.60 (3.89)	-0.05 (0.17)
Shock: Fear only (N = 14)	26.50 (4.06)	29.79 (4.36)	28.59 (4.38)	30.54 (4.65)	27.51 (4.39)	30.81 (4.53)	29.66 (4.80)	29.98 (4.96)	0.65 (0.21)

Pain Reactivity

Table 1 presents pain reactivity means and standard errors. First, VAS intensity and unpleasantness scores were examined to determine whether participants were reliably withdrawing their fingers at the same criterion point (Figure 2). No significant effects were found for trial ($F_s < 1.0$, $P_s > 0.50$), condition ($F_s < 1.0$, $P_s > 0.30$), or the Condition \times Trial interaction ($F_s < 1.10$, $P_s > 0.30$),

suggesting that participants were using similar criteria during the finger withdrawal procedure.

Baselines withdrawal latencies (T1 and T2) were equivalent ($t(57) = 1.74$, $P = 0.09$); therefore, individual level baselines were averaged and used to create change scores. A significant main effect for condition was found ($F(2,55) = 4.30$, $P = 0.02$). The fear only group had greater latencies than the other groups ($P_s < 0.05$) (Figure 3). To examine

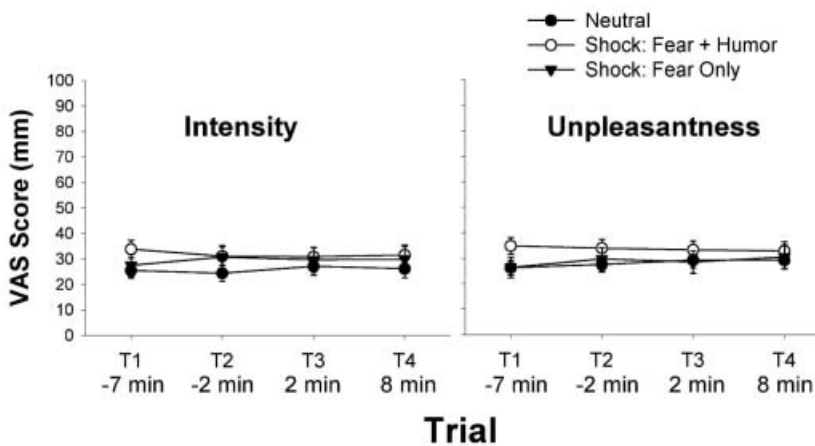


Figure 2 VAS ratings of pain intensity and unpleasantness grouped by condition and trial.

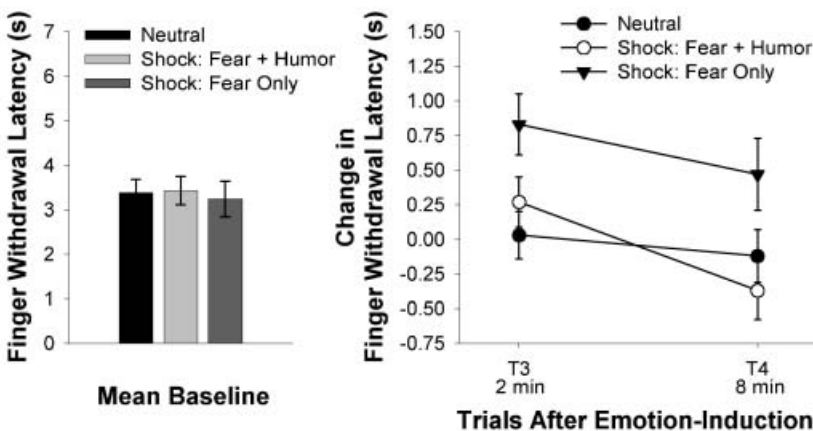


Figure 3 Finger withdrawal latency. Mean baseline latencies grouped by condition are presented in the panel on the left. Mean changes from baseline grouped by condition are presented in the panel on the right. Shocks resulted in hypoalgesia when participants reacted with fear only. Mixed emotion (fear + humor) did not alter pain threshold.

Table 2 Means (standard errors) of self-report psychological status and manipulation check data

Condition	Preexisting psychological status		Affective descriptor subscales					SAM (0–9)	
	SE-PR (0–150)	CES-D (0–60)	Fear	Anger	Happy	Humor	Fatigue	Valence	Arousal
Neutral (N = 26)	45.23 ^a (6.09)	8.29 ^a (1.16)	0.29 ^a (0.14)	0.08 ^a (0.12)	1.46 ^a (0.15)	0.22 ^a (0.14)	0.95 ^a (0.20)	6.57 ^a (0.35)	3.48 ^a (0.37)
Shock: Fear + humor (N = 21)	39.01 ^a (6.64)	13.01 ^b (1.29)	1.23 ^b (0.16)	0.35 ^a (0.13)	0.62 ^b (0.16)	1.42 ^b (0.16)	0.67 ^a (0.21)	4.58 ^b (0.39)	7.26 ^b (0.41)
Shock: Fear only (N = 14)	40.57 ^a (8.13)	6.79 ^a (1.58)	1.42 ^b (0.19)	1.02 ^b (0.16)	0.03 ^c (0.20)	0.00 ^a (0.00)	0.55 ^a (0.26)	3.57 ^b (0.47)	6.93 ^b (0.51)

Lower valence indicates more unpleasant. Means in each column that do not share the same superscript letter are significantly different at $P < 0.05$.

the size of this effect, the two shock groups were recoded (fear + humor = 0, fear only = 1) and correlated with each pain retest change score. Correlations were $r = 0.43$ ($P = 0.009$) and $r = 0.35$ ($P = 0.037$) for Retest 1 and Retest 2, respectively. In sum, fear-inducing shocks resulted in hypoalgesia on the hand contralateral to shock. However, hypoalgesia was only found when *only* fear was produced by the shocks. When shocks elicited humor *and* fear, pain threshold was unchanged.

Confound and Manipulation Checks

Table 2 lists all means and standard errors for self-efficacy (SE-PR), distress (CES-D), and emotion ratings.

Self-Efficacy and Distress

Self-efficacy did not differ by condition ($F(2,54) = 0.26$, $P = 0.77$); therefore, group differences in pain outcomes cannot be attributed to this variable. However, distress ($F(2,55) = 5.70$, $P = 0.006$) was higher in the fear + humor group ($M = 13.01$) than in the fear only ($M = 6.79$) and control ($M = 8.29$) groups; but all were below the cutoff (scores below 16) indicating mild distress [31]. Nonetheless, pain data were reanalyzed covarying CES-D scores to determine if distress influenced pain modulation—results were unchanged.

Emotion Ratings

Table 2 presents data and follow-up comparisons for emotion ratings. A significant main effect for condition was found for SAM valence ($F(2,55) = 14.99$, $P < 0.001$), SAM arousal ($F(2,55) = 27.89$, $P < 0.001$), fear-anxiety ($F(2,55) = 20.55$, $P < 0.001$), anger ($F(2,55) = 11.04$, $P < 0.001$), happiness ($F(2,55) = 17.92$, $P < 0.001$), and humor⁴

⁴ Given that the shocked participants were split into two groups based on this variable, the effect of condition is obviously inflated.

($F(2,55) = 21.70$, $P < 0.001$); but not for fatigue ($F(2,55) = 0.90$, $P = 0.41$). Also, a significant effect of gender was found for happiness ($F(1,55) = 4.05$, $P < 0.05$), with women ($M = 0.90$) being slightly higher than men ($M = 0.50$). No other effects were significant. Compared with controls, both shocked groups (fear only, fear + humor) reported more unpleasantness (lower valence), subjective arousal, and fear-anxiety. However, they did not differ from one another on these variables. Compared with the control and fear + humor groups, the fear only group reported more anger and less happiness. The fear + humor group reported more humor and happiness. To determine whether the statistically higher distress level found in the fear + humor group contributed to the unusual mixed affect reaction, self-reported humor was reanalyzed covarying distress scores. This did not change the results. The fear + humor groups still had higher humor scores. In sum, shocks resulted in increased negative affect and subjective arousal. However, the fear + humor group reacted with some positive affect, whereas the fear only group also experienced mild anger.

Blood Pressure

Table 3 presents the BP data. No significant effects were noted for systolic or diastolic blood pressure ($P_s > 0.44$).

Skin Conductance Level

Table 3 presents means and standard errors for the main effect of SCL. Change scores were created by subtracting individual-level baseline SCL from SCL during emotion induction and prior to retests (T3: +2 minutes and T4: +8 minutes). Significant effects were found for condition ($F(2,54) = 11.08$, $P < 0.001$), gender ($F(1,54) = 9.84$, $P = 0.003$), and trial ($F(1.21,65.41) = 17.42$, $P < 0.001$, $\epsilon = 0.61$). However, the effects of trial and gender were qualified by a Trial \times Gender interaction ($F(1.21,65.41)$

Table 3 Means (standard errors) of psychophysiological manipulation check data

Condition	Change in blood pressure (Δ mmHG)		Change in skin conductance level (μ S)	Change in heart rate (bpm)		
	Systolic	Diastolic		During emotion induction	T1	T2
Neutral (N = 26)	-6.16 ^a (2.63)	-2.60 ^a (2.10)	1.45 ^a (0.50)	-1.84 ^a (1.62)	-1.51 ^a (0.82)	0.03 ^a (0.71)
Shock: Fear + humor (N = 21)	-5.55 ^a (2.96)	-4.42 ^a (2.35)	4.70 ^b (0.56)	1.93 ^b (1.84)	-4.37 ^a (0.93)	0.15 ^a (0.81)
Shock: Fear only (N = 14)	-5.86 ^a (3.54)	-6.23 ^a (-6.24)	4.36 ^b (0.71)	1.43 ^a (2.34)	-1.93 ^a (1.19)	-0.62 ^a (1.03)

Note: μ S = μ Siemen; bpm = beats per minute.

Means in each column that do not share the same superscript letter are significantly different at $P < 0.05$.

= 5.69, $P < 0.02$, $\epsilon = 0.61$). SCL was equally greater in both shocked groups, relative to controls (Figure 4). Further, women generally had greater SCL increases than men ($M = 4.5$ vs $M = 2.43$). Whereas men's SCL decreased by 2 minutes after shocking and leveled off, women's slowly decreased over time. In sum, SCL was increased by shocks, an effect that persisted for at least 8 minutes.

Heart Rate

Table 3 presents HR data. Change scores were created by subtracting individual-level baseline HR from HR during emotion induction and prior to retests (T3: +2 minutes and T4: +8 minutes). A significant effect was found for trial ($F(1.38, 63.57) = 6.88$, $P = 0.006$, $\epsilon = 0.69$) that was qualified by Trial \times Condition ($F(2.76, 63.57) = 3.32$, $P = 0.03$, $\epsilon = 0.66$), and Trial \times Gender interactions ($F(1.38, 63.57) = 4.03$, $P = 0.04$, $\epsilon = 0.66$). Compared with controls, HR was higher during emotion induction in the fear + humor ($P = 0.04$) and fear only groups ($P = 0.08$); but only moderately so in the latter (Figure 5). However, the fear

+ humor and fear only groups were not different from one another ($P = 0.29$). All groups were equivalent at 2 and 8 minutes later. The Trial \times Gender interaction suggests that, when collapsed across groups, men's HR significantly decreased at 2 minutes relative to other times ($P_s < 0.05$). However, women's collapsed HR did not differ over time ($P_s > 0.05$). In sum, HR was increased by shocks, but quickly decelerated.

Discussion

This study examined whether fear-induced hypoalgesia generalizes to the hand contralateral to a shock. Preliminary examination of manipulation checks found that some participants in the shock condition reported experiencing concurrent humor and fear. Therefore, the shocked group was divided into two groups: Participants experiencing fear only and those experiencing fear and humor (fear + humor). Pain data suggest that participants reacting with fear only became hypoalgesic (increased pain threshold), while those reacting with mixed affect (fear + humor) showed no

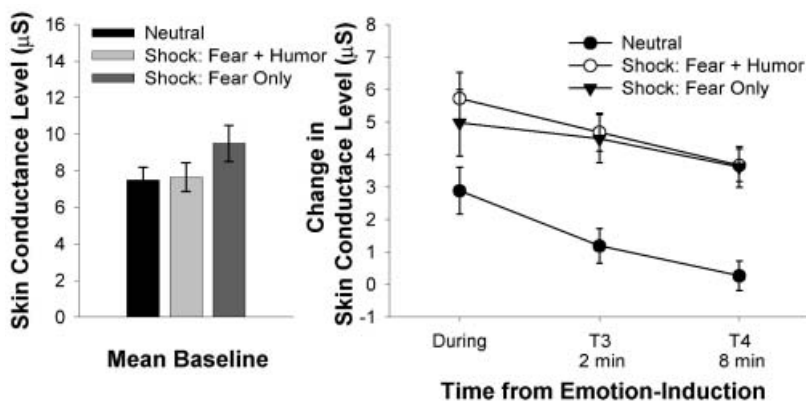


Figure 4 Mean change in SCL during and after emotion induction grouped by condition.

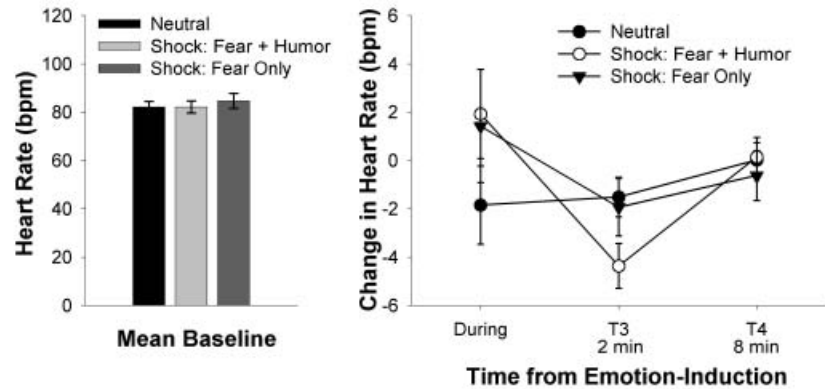


Figure 5 Mean change in HR during and after emotion induction grouped by condition.

change in pain. Manipulation checks indicated that shocks led to increased negative affect (unpleasantness, fear) and arousal (subjective, SCL, HR) in the fear only *and* fear + humor groups. These groups were only distinguished by increased anger in the fear only group and by increased humor and happiness in the fear + humor group. Although participants in the fear + humor group were found to have higher preexisting emotional distress (as measured by the CES-D), this was below the cutoff indicating clinically significant distress [31]. Furthermore, exploratory analyses suggested that distress did not contribute to the change in pain threshold or the mixed reaction in the fear + humor group.

Is Fear-Induced Hypoalgesia Bilateral?

Consistent with prior animal and human data, these findings suggest that fear causes hypoalgesia [2–6,8–11,13,15–22]. In addition, it appears that hypoalgesia occurred on the hand contralateral to fear-inducing shocks. This suggests that pain modulation does generalize to other parts of the body and is bilateral. Although the present study did not test pain on both hands, our laboratory has consistently shown fear-induced hypoalgesia on the hand receiving shocks [13–17]. Further, we have shown that fear-inducing shocks cause hypoalgesia on multiple fingers of the ipsilateral hand [16]. Moreover, in two studies, it was shown that conditioned fear caused hypoalgesia on hands ipsi- and contralateral to shock [17]. Taken together, it appears that fear-induced hypoalgesia is bilateral—not circumscribed to the area most threatened. From an evolutionary perspective, it would be adaptive for fear to cause a whole-body reduction in pain. Fear is an immediate alarm reaction to present threat; thus, system-wide inhibition of nociception would constrain pain

responses that might interfere with fight/flight. This would help ensure survival until safety could be achieved.

Does Humor Inhibit Fear-Induced Hypoalgesia?

It is unclear why some participants in the present study reacted with humor to the shocks. The statistically higher psychological distress in this group may have contributed to this unusual reaction. However, after controlling for between-group differences in distress, these participants still showed a divergent emotional reaction. Furthermore, it would be expected that increased psychological distress should enhance negative reactions, not cause positive affect. Indeed, persons experiencing psychological distress are more likely to interpret stimuli in a negative way [34]. This is the only study of several using the shock-induced fear paradigm that found a significant humor reaction to the shocks [13–14,16–17,35]. The primary difference in the present study is the application of shock to the hand contralateral to pain testing. It is possible that this procedure promoted humor, because participants felt fooled. Prior to testing, stimulating electrodes were applied to the shocked hand; therefore, it was assumed that participants would deduce the location where shocks would be presented. However, they were never explicitly told where shocks would occur. Participants were asked to focus their attention on the nonshocked hand during pain threshold testing in order to assess the painfulness of the heat stimulus. Thus, it is possible that the surprise of receiving the shocks on the contralateral hand promoted humor. Indeed, some participants in the fear + humor group laughed following shock delivery.

Although it is unclear what caused this mixed reaction, it appears that humor inhibited fear-

induced hypoalgesia. When humor was experienced concurrent with fear, no changes in pain were noted. Only those reacting with fear alone had elevated pain thresholds. This is seemingly counterintuitive given the many reports suggesting positive affect induces hypoalgesia [27,36–42]. Given these findings, one might expect that the hypoalgesia caused by positive affect and fear should sum to cause stronger hypoalgesia. However, there is reason to believe this would not be the case.

According to Lang's theory of motivational priming [28], stimuli eliciting negatively valenced affect activate the defensive system (associated with negative affect) while inhibiting the appetitive motivational system (associated with positive affect). Alternatively, stimuli eliciting positively valenced affect activate the approach system while inhibiting the defensive system. Lang and his colleagues have shown that positive affect inhibits the startle reflex (a defensive response), while negative affect augments it [28–30,43]. This theory suggests that the simultaneous activation of dual motives (positive and negative affect) would have a zero net effect on startle [28]. Indeed, our laboratory has previously noted that pain is unchanged when dual motives are activated [27]. Future studies should experimentally test whether positive affect negates the influence of fear by presenting appetitive stimuli following shocks to examine the effect on pain.

The Potential Role of Diffuse Noxious Inhibitory Control

It could be argued that pain resulting from the shocks, and not the emotional state of the participant, caused hypoalgesia. Indeed, evidence suggests that activation of nociceptors (pain receptors) can activate the brainstem (i.e., nucleus raphe magnus), causing descending projections to inhibit nociception in the dorsal horn [44–45]. This phenomenon, known as diffuse noxious inhibitory control (DNIC), is not thought to involve higher brain centers involved with emotion. Although it is clear that pain can be inhibited by directly activating such lower level systems, three lines of evidence suggest that a forebrain-mediated fear state underlies the current hypoalgesia. First, not all shocked participants became hypoalgesic. Those reacting with fear and humor did not show a change in pain. Unfortunately, the present study did not ask participants to rate the painfulness of the shocks. Therefore, it is possible that participants reacting with fear alone found the shocks

more painful than those reacting with fear and humor. On the other hand, physiological reactions to shock (HR, SCL, BP) did not differ between these two groups, suggesting that the painfulness did not differ. Second, our laboratory and others have shown that non-noxious fear-inducing stimuli can cause pain inhibition [15–17,19,22]. Indeed, presentation of a spider, non-noxious noise bursts, threat of shock, and video viewing have all been used to elicit fear-induced hypoalgesia. Third, while La Bars and Villanueva have suggested that emotional/stressful reactions elicited by noxious stimuli do not contribute to DNIC [44–45], it would be surprising if such painful stimuli had no effect on emotion; hence, it remains possible that fear may contribute to the activation of descending pain inhibitory systems. Supporting this, exposure to threatening stimuli has been shown to elicit a forebrain-mediated hypoalgesia through circuits in the amygdala and frontal cortex that, in turn, activate descending pain inhibitory systems in the brainstem [2,10–11,46–47]. Indeed, the hypoalgesia observed in rats after exposure to three brief shocks is eliminated by decerebration and frontal cortex lesions [10–11,48]. Because the present experiment was modeled after those studies, it is likely that the same supraspinal fear circuit underlies the hypoalgesia observed in humans.

The Importance of Valence and Arousal in Pain Modulation

Previously, we have argued that affective valence and arousal interact to determine the outcome of emotion on pain [24]. Negative affect with low-to-moderate arousal (e.g., anxiety) causes hyperalgesia, whereas negative affect with high arousal (e.g., fear) causes hypoalgesia. Conversely, positive affect always causes hypoalgesia as long as a minimal level of arousal is achieved. However, the definitive study has yet to be conducted effectively manipulating valence and arousal across a wide range of each variable. Therefore, it remains possible that arousal is the critical variable, such that arousal is solely responsible for affect-induced hypoalgesia. Indeed, areas of the midbrain and brainstem responsible for arousal are also implicated in pain modulation [23]. However, the present study suggests that arousal is not the critical variable. Shocks led to equivalent levels of subjective and physiological arousal, regardless of whether participants reacted with fear only or fear and humor. But, humor appeared to inhibit the hypoalgesia. These data suggest that valence *and* arousal contribute to pain modulation.

The Role of Attention

Some have argued that attention, not emotion, modulates pain [22,49–52]. Ostensibly, motivationally relevant stimuli, and the affect that follows, shift cognitive resources away from the nociceptive processing, thus dampening pain. The paradigms employed by our laboratory have not systematically evaluated the role of attention; therefore, it is unclear whether changes in attention contribute to fear-induced hypoalgesia. However, we have recently shown that presenting a postshock distractor attenuated fear-induced hypoalgesia [53]. If shock elicits pain reduction through distraction, then it would not be expected that an additional distractor would counter the hypoalgesia. One interpretation is that the postshock distractor displaced the memory of the fear-producing stimulus [53–55], thus diminishing fear-induced hypoalgesia. This suggests that affect, not attention, modulates pain. Moreover, the present study does not suggest that attention mediates hypoalgesia. If it did, then both the fear only and fear + humor groups should have experienced pain reduction, because shocks (and the affect that followed) should have drawn attention away from the processing of the heat stimulus.

Potential Clinical Relevance and Future Directions

From a clinical perspective, these data suggest that patients who experience intense fear in response to unpredictable threatening events will show a reduction in pain perception. In contrast, other patients who experience the same threatening event as both frightening and humorous will not exhibit pain inhibition. These findings may help to explain individual differences in pain perception following traumatic events. For example, some victims of motor vehicle accidents (MVAs) report a decrease in injury-related pain immediately following the accident, whereas others do not [56]. We propose that individual differences in emotional reactivity to the traumatic event itself contribute to differences in pain. To further evaluate this hypothesis, future clinical research should investigate whether individual differences in emotional responses to traumatic events (e.g., MVA, shooting, assault) account for variability in injury-related pain.

Fear-induced hypoalgesia differs from the phenomenon of fear of pain associated with low pain tolerance in clinical studies [57–61]. We propose that fear-induced hypoalgesia and fear of pain are distinct concepts mediated by different mechanisms. Fear-induced hypoalgesia is a temporary

state in which an alarm reaction to a threatening event subsequently alters pain sensitivity. The high levels of fear and arousal elicited by that threat trigger the release of endogenous opioids that inhibit pain. Attentional processing of subsequent painful stimuli may also be reduced, because the fear stimulus is more salient, resulting in decreased pain perception. In contrast, fear of pain is a stable trait characterized by negative affect and apprehensive anticipation/avoidance of pain [57–61]. Unlike fear, high levels of anxiety result in hypervigilance and somatic scanning [1], which should increase attention to pain, and thereby increase its perceived intensity.

Future experimental studies should investigate the effects of dual motives following less-arousing negative and positive affective states. Most experimental and clinical studies examining the influence of affect on human pain perception have tended to use relatively subtle affect manipulations that do not induce heightened levels of physiological arousal. In those studies, participants are typically asked to view emotional visual stimuli (pictures or movies) or to read or listen to affectively charged verbal material. In contrast with the present study, which utilized shock to elicit an intense negative affect state with high arousal, those studies generally observe negative-affect-induced hyperalgesia and positive-affect-induced hypoalgesia [38–42,62–66]. Using picture viewing to induce affect, our laboratory found that the pain-enhancing effects of negative affect with low arousal and the pain-attenuating effects of positive affect with moderate arousal appear to be cancelled out by the simultaneous activation of appetitive and aversive motivational states [27]. Specifically, pain tolerance was reduced by viewing pictures that induced mild fear, but not by disgust slides that evoke a mixed emotional response (fear and pity). Unlike fear, which elicits an avoidance disposition, pity elicits an approach disposition to help others. Thus, we propose that when mixed emotions activate dual motives, the effect on pain modulation is determined by the relative activation of the aversive/avoidance and appetitive/approach motivational systems.

Although the antihypoalgesic effect of humor in the present study appears to conflict with prior clinical and experimental studies demonstrating that humor inhibits pain, it does not. Indeed, our account anticipates that humor alone or other positive emotional states would reduce pain [24,27]. However, when humor is combined with fear, the simultaneous activation of opponent motivational

states would be expected to cancel each other out rather than summate. To further evaluate this issue, future studies should investigate how the dimensions of valence and arousal interact and whether this relationship varies across particular emotional categories (e.g., fear, humor, etc.). These dimensions may interact to produce different outcomes depending on their product (e.g., low products yielding hyperalgesia and high products yielding hypoalgesia) and the particular emotion category.

Conclusions

The present study examined whether fear-inducing shocks cause hypoalgesia on the hand contralateral to shocks. Manipulation checks indicated that shocks elicited negative affect and increased arousal; however, some participants reacted with fear *and* humor to shocks. Pain threshold was unchanged in those participants. Only those participants reacting with fear *only* became hypoalgesic. These data suggest that fear-induced hypoalgesia does occur bilaterally. Furthermore, humor may inhibit fear-induced hypoalgesia.

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