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Negative affect: effects on an evaluative measure of human pain

Jamie L. Rhudy[a], Mary W. Meagher[b,*]

[a]*Department of Psychiatry and Human Behavior, University of Mississippi Medical Center, 2500 North State Street, Jackson, MS 39216, USA*
[b]*Department of Psychology, Texas A&M University, TAMU 4235, College Station, TX 77843-4235, USA*

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Abstract

Prior work indicates that exposure to fear-inducing shock inhibits finger-withdrawal to radiant heat in humans (hypoalgesia), whereas anxiety induced by threat of shock enhances reactivity (hyperalgesia; Pain 84 (2000) 65–75). Although finger-withdrawal latencies are thought to reflect changes in pain sensitivity, additional measures of pain are needed to determine whether pain perception is altered. The present study examined the impact of negative affect on visual analog scale (VAS) ratings of fixed duration thermal stimuli. One hundred twenty-seven male and female human subjects were randomly assigned to one of three emotion-induction conditions: (1) negative affect induced by exposure to three brief shocks; (2) negative affect elicited by the threat of shock without presentation; and (3) neutral affect, with no intervention. VAS ratings were tested before and after emotion-induction. Results suggest that both negative affect manipulations reduced pain. Manipulation checks indicated that the emotion-induction treatments induced similar levels of fear but with different arousal levels. Potential mechanisms for affect induced changes in pain are discussed.

Keywords: Fear; Anxiety; Stress; Analgesia; Pain modulation; Heat

1. Introduction

Several theories predict that negative affect can modulate the experience of pain (Melzack and Wall, 1965; Beecher, 1966; Melzack and Casey, 1968). Although understanding the link between negative emotion and pain may have implications for pain management, relatively few well-controlled studies have examined this issue. The few human studies that have been conducted report mixed results, with some observing decreased pain (Bobey and Davidson, 1970; Willer et al., 1979; Malow, 1981; Pitman et al., 1990, Janssen and Arntz, 1996, Rhudy et al., 1999; Rhudy and Meagher, 2000, 2001a), while others report increased pain (Haslam, 1966; von Graffenried et al., 1978; Schumacher and Velden, 1984; Weisenberg et al., 1984; Malow et al., 1987; Cornwall and Donderi, 1988; Meagher et al., 2001a).

In contrast, animal studies have examined the impact of a variety of stressors on pain, with most reporting pain inhibition (hypoalgesia) after exposure to noxious and non-noxious aversive stimuli (Bodnar et al., 1980; Bolles and Fanselow, 1980; Basbaum and Fields, 1984; Fanselow, 1984, 1986; Watkins and Mayer, 1986; Maier, 1989; Meagher et al., 1989, 1990; Lichtman and Fanselow, 1990). Interestingly, many of these stressors (e.g. shock, predators, cues paired with shock) also elicit behaviors indicative of fear (e.g. freezing, tachycardia, increased startle, decreased social interaction), suggesting that fear mediates stress-induced hypoalgesia (Bolles and Fanselow, 1980; Fanselow, 1984, 1986; Lichtman and Fanselow, 1990). Indeed, under some circumstances the amygdala, a neurological structure implicated in the production of fear responses, can activate descending pain modulatory pathways (Helmstetter, 1992, 1993; Helmstetter and Bellgowan, 1993; Watkins et al., 1993; Fox and Sorenson, 1994; Manning and Mayer, 1995a,b; Manning, 1998; Crown et al., 2000). Taken together, these data suggest that highly arousing negative affective states, such as fear, can inhibit pain. However, under other circumstances pain enhancement (hyperalgesia) is observed following a stressor (Vidal and Jacob, 1986; Illich et al., 1995; King et al., 1996; Meagher et al., 1998a,b, 2001b; King et al., 1999; McLemore et al., 1999).

How can these seemingly divergent findings be resolved?

* Corresponding author. Present address: Department of Psychology, University of Tulsa, Lorton Hall, 600 South College, Tulsa, OK 74104, USA. Tel.: +1-979-845-2564; fax: +1-979-845-4727.
E-mail address: m-meagher@tamu.edu (M.W. Meagher).

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Recent animal studies suggest that the direction of a modulation may depend on the severity of the stress-inducing event (Meagher et al., 1998, 2001a,b; Sieve et al., 2001). Hyperalgesia is typically observed after exposure to less severe stressors or in situations of diffuse threat (e.g. mild shock, novelty, holding), whereas hypoalgesia occurs after exposure to severe stressors (e.g. exposure to a predator, intense shock). Differences in the severity and imminence of the stress-inducing stimulus are likely to result in differences in arousal. Therefore, we have proposed that highly arousing negative affect may cause hypoalgesia in humans, whereas low-to-moderately arousing negative affect results in hyperalgesia (Rhudy and Meagher, 2000, 2001c; Meagher et al., 2001a,b).

This hypothesis was tested in three experiments modeled after animal studies. Rhudy and Meagher (2000) demonstrated that exposure to three brief shocks produced physiological and subjective indicators of fear (negative affect, high arousal), followed by increased finger-withdrawal latencies to radiant heat (hypoalgesia). In contrast, induction of anxiety (negative affect, low arousal) by verbal threat of shock (without actual delivery) decreased withdrawal latencies (hyperalgesia). In a subsequent study, surprising bursts of white noise were substituted for shocks to demonstrate that a non-noxious fear-inducing stimulus could elicit hypoalgesia (Rhudy and Meagher, 2001a). Recently, conditioned hypoalgesia was elicited by exposure to a stimulus (CS +) that had been previously paired with shock relative to a CS − that was unpaired (Rhudy and Meagher, 2001b). Because these studies were modeled after animal studies, pain reactivity was assessed using a method similar to the tail-flick test – finger-withdrawal from radiant heat. Similar to the tail-flick test, the latency from heat onset to finger-withdrawal was used as the index of pain reactivity. To further mimic the animal paradigm, a rapid onset heat stimulus was used that ostensibly elicited a finger-withdrawal reflex rather than a supraspinally mediated, evaluative pain response. Although it was assumed that the observed effects would generalize to other indices of pain, this hypothesis requires testing. This is important because several animal studies indicate that the inhibition of a protective withdrawal reflex is not necessarily associated with a reduction in supraspinally mediated measures of pain sensitivity (Meagher et al., 1989, 2001a,b; Morgan et al., 1994; Illich et al., 1995; King et al., 1996, 1997).

The present study evaluated the impact of negative affect on a pain index that requires supraspinal, evaluative processes. To do so, pain was tested before and after one of three emotion-induction conditions: (1) shock – exposure to three brief shocks intended to produce highly arousing negative affect (e.g. fear), (2) threat of shock – verbal threat of shock without delivery intended to produce moderately arousing negative affect (e.g. anxiety), and (3) neutral – no affect intervention. Pain was tested using subjective VAS ratings of a fixed duration and intensity heat stimulus. We hypothesized that the less severe threat of shock manipulation would produce hyperalgesia while the relatively severe shock manipulation would produce hypoalgesia. Furthermore, to examine whether emotion differentially modulates threshold and suprathreshold pain, two intensities of heat were used. Half of the participants received heat at a fixed duration corresponding to their pain threshold, whereas half received heat with duration 20% longer than their pain threshold.

This study also evaluated whether the magnitude of pain modulation depends on test location by testing two other fingers (middle, ring) of the ipsilateral hand. Although several studies suggest that pain modulation may be localized (Prentice et al., 1996; Benedetti et al., 1999), others report diffuse modulation (Le Bars and Villaneuva, 1988; Villaneuva and Le Bars, 1995). If pain modulation is localized to the site of electrical stimulation (index finger), pain ratings on non-shocked fingers should be unchanged following shock exposure. Alternatively, if pain modulation is diffuse, ratings on non-shocked fingers should vary as a result of shock to the index finger.

2. Method

For a thorough explanation of methods, apparatus, and stimuli see the study by Rhudy and Meagher (2000).

2.1. Subjects

Participants were 127 undergraduate psychology students who received course credit for participation. Thirty-two were excluded due to equipment problems or failure to comply with instructions (i.e. removed finger from heat source before computer turned it off) leaving 48 women and 47 men. Of those, 87% were Caucasian, 9% Hispanic, 2% African American, and 2% Asian. Mean age was 19.01 years (SD = 1.07). Subjects were excluded if they had: circulatory, cardiovascular, or neurological problems, chronic pain, recently used tobacco, analgesics, antidepressants, or alcohol, or experienced a recent psychological trauma.

2.2. Apparatus and physiological recording

All data acquisition and stimulus presentation were computer-controlled. Skin conductance level (SCL) and heart rate (HR) sensors were attached to fingers of the non-dominant hand and sampled at 50 Hz. Blood pressure (BP) was measured pre- and post-experiment using a digital meter (Eckerd Drug Company-Model-E7622).

2.3. Electrocutaneous stimulation

Fear stimuli consisted of three, brief, 12 mA (moderately painful) shocks applied to the index finger of the dominant hand.