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Ethnic differences in the nociceptive flexion reflex (NFR)

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Abstract

A substantial body of literature suggests that the experience of both clinical and experimental pain differs among ethnic groups, with African Americans generally reporting greater sensitivity to chronic and experimentally induced pain when compared to non-Hispanic whites. However, no studies to date have examined nociceptive processes that may underlie these differences. The nociceptive flexion reflex (NFR) is based on the measurement of stimulus-induced spinal reflexes. Prior research suggests that the NFR threshold, or RIII response, is highly correlated with subjective pain thresholds. The current study evaluated responses to the nociceptive flexion reflex in healthy young adults from two different ethnic groups: African Americans ($n = 29$) and non-Hispanic whites ($n = 28$). Perceptual responses (e.g., pain ratings) as well as physiological reflex responses (i.e., biceps femoris EMG) were assessed. Significant ethnic group differences were observed for NFR reflex threshold, with African Americans producing a reflex at lower stimulation intensities relative to non-Hispanic whites. Interestingly, verbal pain ratings at NFR threshold were not significantly different between the groups, suggesting that the lower stimulation intensities required to elicit a reflex in African-American versus non-Hispanic white participants were nonetheless perceived as similar. Psychological Involvement, Positive and Negative Mood, and Rumination were correlated with NFR threshold in a pattern that was consistent across both ethnic groups. These results extend previous research on ethnic differences in self-report measures of pain by demonstrating group differences in a nociceptive muscle reflex.

Keywords

Ethnic differences; Experimental pain; Nociceptive flexion reflex; RIII

1. Introduction

Considerable evidence has demonstrated that both clinical and experimental pain responses differ across ethnic groups, with African Americans (AA) generally demonstrating greater sensitivity to clinical and experimentally induced pain when compared to non-Hispanic whites (Green et al., 2003). Edwards et al. (2001) found higher levels of pain and disability among AA relative to white patients seen in a multidisciplinary pain center. AA with chronic pain have also reported higher levels of pain unpleasantness, greater pain-related emotional distress and increased pain behaviors relative to whites (Riley et al., 2002; Green et al., 2003).

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Laboratory studies have consistently demonstrated increased experimental pain sensitivity among AA compared to whites (Zatzick and Dimsdale, 1990), with AA demonstrating lower heat pain thresholds and tolerances (Chapman and Jones, 1944; Sheffield et al., 2000), and lower cold pressor pain tolerances (Walsh et al., 1989). Additionally, AA reported greater intensity and unpleasantness in response to a modified ischemic task relative to whites, using a standardized rating scale (Campbell et al., 2004), and higher ratings of unpleasantness to heat pain tasks (Edwards and Fillingim, 1999). Therefore, ethnic differences in both clinical and experimental pain have been widely reported, with the most robust differences in experimental pain found for suprathreshold measures (Campbell et al., 2005).

Group differences in pain report could be related to differences in use of pain scales (e.g., Campbell et al., 2004). Thus, electrophysiological alternatives, such as the nociceptive flexion reflex (NFR), may provide important information regarding ethnic differences in nociception (Skljarevski and Ramadan, 2002). The NFR is a protective physiological withdrawal reflex triggered by noxious stimuli (Mylius et al., 2005), and has been widely used as an index of nociceptive threshold (Rhudy and France, 2007). While NFR is based on measurement of stimulus-induced spinal reflexes (Willer, 1977), it may undergo modulation before and after it reaches supraspinal centers (France, 2002) and may be influenced by psychological factors and external stimuli (Rhudy and France, 2007). However, NFR can be administered in a standardized fashion (Skljarevski and Ramadan, 2002), has adequate reproducibility (French et al., 2005) and has been shown to correlate with subjective pain thresholds (Rhudy and France, 2007). While numerous studies have examined differences in pain perception using self-report, no studies to date have examined ethnic differences in NFR.

Psychosocial factors may influence NFR responses. Altering participant's affect has recently been shown to alter NFR and pain ratings (Rhudy et al., 2005). When shock is predictable, affective modulation is observed for pain ratings but not NFR responses (Rhudy et al., 2006). Individual differences in catastrophizing (France et al., 2004) and anxiety (French et al., 2005) have been associated with pain ratings, but not with NFR. In order to determine the contribution of psychosocial factors to NFR and ethnic differences in pain ratings, participants completed several psychological questionnaires.

This study was designed to further elucidate the nature of ethnic differences in pain perception by investigating electrophysiological (i.e., NFR) and perceptual (i.e., verbal pain rating) responses to electrical stimulation, and the contribution of psychological factors to NFR was examined.

2. Research design and methods

The total study sample consisted of 58 healthy young adults (29 African Americans, 29 non-Hispanic whites). The University of Florida Institutional Review Board approved all study procedures. Women were scheduled during their follicular phase (i.e., days 4–9 of the menstrual cycle) to reduce variability associated with menstrual cycle effects (Riley et al., 1999; Tassorelli et al., 2002). Verbal and written informed consent were obtained, after which participants completed a health history questionnaire, which indicated that all were in good health and had no prior history of pain problems. Next, they completed a series of questionnaires assessing demographic information, mood, catastrophizing, and hypervigilance (described in detail below). Ethnicity was determined using self-report. Following completion of the questionnaires, participants were instrumented (see procedure below) and rested for 10 min, after which time blood pressure was measured for 5 min using an automated blood pressure cuff, then their NFR threshold was determined.

2.1. Nociceptive flexion reflex assessment

To measure the nociceptive flexion reflex, electromyographic (EMG) activity was recorded from the biceps femoris muscle of the left leg using a DelSys, Bagnoli-2 differential amplifier. The active electrode was placed over the left biceps femoris muscle 10 cm superior to the popliteal fossa, and a reference electrode attached over the lateral epicondyle of the femur. EMG was recorded and processed using a CED Micro1401 analog-to-digital converter and Spike2 software. A Nicolet bar electrode (anode inferior) was attached to the left leg over the retromalleolar pathway of the sural nerve and electrical stimulation was delivered using a Digitimer, DS7A constant-current stimulator (Hertfordshire, UK). All stimulating and recording sites were cleaned and gently abraded to achieve an impedance of less than 10,000 Ohms prior to electrode placement.

Participants were then comfortably seated in a recliner chair with a foam cushion placed under the participant's knee in order to maintain a 60-degree angle of the left knee. Electrical stimulation was applied in a series of ascending and descending steps according to Levitt's staircase methodology (1971). A 5-pulse train (1 ms pulse duration, 3 ms inter-pulse interval) was administered approximately once every 30 s. The nociceptive flexion reflex was operationally defined as a mean rectified EMG response in the 90–150 ms post-stimulus interval that exceeded mean rectified EMG activity during a 60 ms pre-stimulus baseline interval (–65 to –5 ms) by at least 1.5 SD. This interval has been shown to circumvent contamination by low-threshold cutaneous flexor reflex, startle reactions and voluntary movements (Page and France, 1997; France and Suchowiecki, 2001; France et al., 2002; French et al., 2005). Stimulation intensity began at 0 mA and increased in 4 mA steps until the nociceptive withdrawal reflex was obtained (or a maximum intensity of 40 mA, or termination was requested by the participant). Stimulus intensity was then decreased in 2 mA steps until the reflex was no longer observed. From this intensity, the procedure was repeated using 1 mA steps so that the nociceptive withdrawal reflex appeared and subsided three times in total. Reflex threshold was defined according to Rhudy and France (2006) as the average of the second and third peaks and troughs using this methodology. During reflex assessment, participants rated the perceived intensity of each pulse using a 0–100 rating scale (0 = no sensation and 100 = strongest imaginable sensation of any kind). Pain ratings corresponding to the second and third peaks and troughs were averaged to determine the pain reported at the NFR threshold intensity.

2.2. Psychological measures

In order to determine the contribution of psychosocial factors to the NFR and potential group differences in experimental pain responses, subjects completed the following psychological questionnaires.

The Pain Catastrophizing Scale (PCS) (Sullivan et al., 1995) consists of 14 items rated on a 5-point scale ranging from 0 (not at all) to 4 (all the time). Participants are instructed to indicate the degree to which they have specified thoughts and feelings when experiencing pain. The measure assesses three dimensions of catastrophizing: Rumination, Magnification, and Helplessness. The PCS has been validated for both clinical and nonclinical samples (Sullivan et al., 1995; Osman et al., 2000).

The Kohn Reactivity Scale (Kohn, 1985) consists of 24 items that assess an individual's level of reactivity or central nervous system arousability and has been used as a measure of hypervigilance (McDermid et al., 1996). This measure has been shown to correlate negatively with pain tolerance (Dubreuil and Kohn, 1986) and has been reported to have adequate internal consistency, ranging from alpha of 0.73 to 0.83 (Kohn, 1985).

The Frid Scale (FRID) is a 10-item, 5-point Likert scale assessing expectancies and attitudes regarding experimental pain procedures. The measure consists of three subscales including Psychological Involvement in the experiment, Negative Expectancies regarding the experiment, and Efficacy and Control beliefs (Frid et al., 1979).

The Visual Analogue Mood Scale (VAMS) consists of 8 horizontal 100 mm VAS scales representing different aspects of mood. Participants were asked to mark their current mood by placing a vertical mark on the line. The VAMS has been shown to have adequate validity (Killgore, 1999).

3. Results

The NFR was not detected in five (3 African Americans, 2 non-Hispanic whites) of the 58 subjects. Compared to those showing a reflex, individuals not exhibiting a reflex were significantly older (36.0 vs. 23.3 years) and provided significantly higher pain ratings for the maximum stimulus intensity tolerated (mean rating of 86.0 vs. 52.3). All subsequent analyses are based on the 53 subjects who produced a reflex. A *t*-test revealed significant ethnic group differences in NFR threshold ($t(51) = 2.23, p = .030$, Cohen's $d = .61$), with African Americans ($M = 14.99$ mA, $SD = 8.98$) demonstrating a reflex at a lower stimulus intensity relative to non-Hispanic whites ($M = 20.95$ mA, $SD = 10.45$). Interestingly, verbal pain ratings corresponding to NFR threshold level were variable, but in the mild to moderate range, and not significantly different between the groups, suggesting that the lower stimulation intensities required to elicit a reflex in African-American ($M = 40.00$, $SD = 22.07$) versus non-Hispanic white participants ($M = 44.95$, $SD = 25.5$) were nonetheless perceived as similar on the 0–100 pain rating scale. Also, the relationship between NFR reflex and pain ratings corresponding to this stimulus was significant in both non-Hispanic whites ($r = .718, p < .001$) and African Americans ($r = .561, p = .003$). Finally, there were no ethnic group differences observed for sex, age, body mass index, blood pressure, or any of the psychological variables (see Table 1).

Correlational analyses were conducted examining associations between responses to psychological questionnaires and NFR threshold. Psychological Involvement (a subscale of the FRID scale) and Positive Mood were positively correlated with NFR threshold, and the Rumination subscale of the PCS and Negative Mood were negatively correlated with NFR threshold (see Table 2). The pattern of correlations was consistent across both ethnic groups. In order to determine whether these psychological variables contributed to ethnic group differences in pain responses, ANCOVAs were performed. Group differences in the NFR remained significant after controlling for Psychological Involvement and Positive Mood. When controlling for Rumination ($p = .066$) and Negative Mood ($p = .063$), the ethnic difference in the reflex remained marginally significant. Thus, psychological variables do not seem to have contributed significantly to the group differences in the NFR reflex. No significant correlations emerged between psychological factors and NFR ratings (all p 's $> .10$).

4. Discussion

The findings of this study indicate ethnic differences in the NFR, such that African Americans required less electrical stimulation to produce a nociceptive muscle reflex. Because the NFR is an electrophysiological, rather than self-report, nociceptive reflex measure, the current findings extend the existing evidence that African American individuals demonstrate greater sensitivity to noxious stimuli compared to whites. The pain report corresponding to the NFR was similar across ethnic groups, and on average reflected mild to moderate pain levels, comparable to previous research (France et al., 2004, 2005; French et al., 2005). The mechanisms underlying ethnic group differences in the NFR cannot be determined from this study; however, it is tempting to speculate that descending pain modulation may differ across

ethnic groups, which could contribute to group differences in the nociceptive reflex. In this regard, Mechlin et al. (2005) recently demonstrated that stress-induced pain regulatory mechanisms involving blood pressure, norepinephrine, and cortisol functioned more effectively among whites than African Americans. The present study was not designed to permit assessment of stress-induced physiological responses; therefore, we cannot determine the extent to which similar processes contributed to the observed differences.

While typically conceptualized in biological terms, descending pain modulation can be driven by psychosocial factors (e.g., expectations, pain beliefs). Indeed, our findings demonstrate modest associations between the NFR and psychological variables. Specifically, greater Psychological Involvement and higher Positive Mood correlated positively with NFR, whereas higher scores on the Rumination subscale of the Pain Catastrophizing Scale predicted lower NFR. While these psychological variables did not account for the group differences we observed in the NFR, it is important to recognize that the magnitude of these associations was low, generally accounting for less than 10% of the variance in the NFR. Thus, it is plausible that other psychological processes not assessed in this study may show more robust associations with the NFR and could contribute to the group difference via descending modulatory influences. Previous research exploring psychological correlates of NFR has provided mixed results. For example, neither catastrophizing nor anxiety was found to be associated with NFR, while both variables predicted subjective pain ratings in response to electrical stimulation (France et al., 2002, 2004; French et al., 2005). It may be important to note that France et al. (2002) used the catastrophizing subscale of the Coping Strategies Questionnaire (CSQ), while we assessed catastrophizing with the PCS, which yields separate subscales, one of which (i.e., the Helplessness subscale) is equivalent to the CSQ catastrophizing scale. Indeed, our results showed an association only between the Rumination subscale of the PCS, and the Helplessness subscale was not associated with the NFR. Similar to France et al. (2004), we found no association between total PCS scores and the NFR. Thus, it may be that only specific components of catastrophizing predict NFR thresholds. Other investigators have reported significant psychological modulation of NFR thresholds. For example, induction of Positive and Negative Mood decreased and increased NFR activity, respectively (Rhudy et al., 2005, in press), consistent with our associations of the NFR with Positive Mood. Moreover, a brief session of pain coping skills training significantly increased NFR thresholds among patients with osteoarthritis of the knee (Emery et al., 2006), and hypnotic analgesia also increased NFR thresholds (Kiernan et al., 1995). Thus, both previous research and the present findings provide evidence that psychological processes may modestly influence NFR thresholds, and whether psychosocial factors contribute to ethnic group differences in the NFR merits additional investigation.

There are several noteworthy limitations when interpreting the results of the present study. First, the electrical stimuli used to elicit the NFR produce an acute, controlled painful experience in which all participants are aware that testing may be stopped at any time. Therefore, it is unknown how these findings relate to a clinical population; however, the NFR is an electrophysiological method for assessing nociceptive responses, which may provide insight into possible group differences in pain processing. In addition, all subjects were generally well-educated, healthy, young adults; therefore, the degree to which the current findings generalize to other populations, such as elderly or more poorly educated samples, is unknown. Finally, potential differences in pain perception among subgroups within larger ethnic categories have been reported (Chapman and Jones, 1944; Sternbach and Tursky, 1965), albeit not consistently (Lipton and Marbach, 1984; Granot et al., 2003), but were not investigated in the present study.

In sum, the current study provides evidence of ethnic differences in a spinally mediated nociceptive reflex. Future studies may clarify how ethnic differences in NFR relate to clinical

pain conditions or pursue a better understanding of the underlying mechanisms involved in ethnic differences by examining the contributions of descending pain modulatory systems.

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Table 1
Demographic information, means (SD) and effect sizes for cardiovascular and psychological measures by ethnicity

| Variable | African Americans (<i>n</i> = 26) | Whites (<i>n</i> = 27) | Effect size |
|-----------------------------------|------------------------------------|-------------------------|-------------|
| Age (SD) | 22.0 (2.3) | 24.6 (7.9) | .44 |
| Sex (% female) | 51.7 | 57.7 | – |
| BMI (SD) | 23.4 (5.4) | 23.1 (2.6) | .07 |
| Systolic blood pressure | 108.3 (10.1) | 108.7 (8.4) | .01 |
| Diastolic blood pressure | 62.1 (7.6) | 61.2 (6.9) | .06 |
| Heart rate | 67.3 (11.3) | 64.6 (9.4) | .28 |
| VAMS Positive Mood | 168.9 (66.7) | 165.8 (64.9) | .05 |
| VAMS Negative Mood | 40.5 (56.7) | 22.7 (24.7) | .41 |
| FRID Negative Expectancies | 5.7 (1.9) | 5.1 (1.5) | .35 |
| FRID Efficacy and Control Beliefs | 11.5 (1.9) | 12.5 (2.0) | .51 |
| FRID Psychological Involvement | 11.9 (2.0) | 12.1 (2.1) | .10 |
| PCS Rumination | 3.6 (3.3) | 3.3 (3.1) | .09 |
| PCS Magnification | 1.4 (1.8) | 1.4 (1.4) | 0 |
| PCS Helplessness | 2.9 (3.5) | 2.3 (2.8) | .19 |
| PCS total | 7.9 (7.4) | 7.0 (5.8) | .14 |
| Kohn Reactivity Scale | 68.2 (15.4) | 66.0 (9.6) | .17 |

VAMS, Visual Analogue Mood Scale; PCS, Pain Coping Scale.

Table 2

Correlation matrix of psychological factors and NFR Threshold by ethnic group

| Variable | NFR threshold | | |
|-----------------------------------|-------------------|--------|---------------|
| | African Americans | Whites | Combined |
| VAMS Positive Mood | .381* | .345 | .337* |
| VAMS Negative Mood | -.255 | -.248 | -.273* |
| FRID Negative Expectancies | .051 | -.165 | .098 |
| FRID Efficacy and Control Beliefs | .019 | .119 | .142 |
| FRID Psychological Involvement | .214 | .329 | .285* |
| PCS Rumination | -.230 | -.291 | -.295* |
| PCS Magnification | -.037 | -.134 | -.089 |
| PCS Helplessness | -.038 | .019 | -.072 |
| PCS total | -.134 | -.224 | -.210 |
| Kohn Reactivity Scale | -.360 | .197 | -.135 |

VAMS, Visual Analogue Mood Scale; PCS, Pain Coping Scale.

* $p < .05$.