Ethnic Differences in Pain Tolerance: Clinical Implications in a Chronic Pain Population

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Objective: Although numerous studies have independently examined ethnic differences in clinical and experimental pain, few have investigated differences in both sensitivity to controlled noxious stimuli and clinical pain reports in the same sample. The present experiment examined the effects of ethnicity (African American vs. white) on experimental pain tolerance and adjustment to chronic pain. **Methods:** Three hundred thirty-seven (68 African American and 269 white) patients with chronic pain referred to a multidisciplinary treatment center participated in the study. In addition to completing a number of standardized questionnaires assessing adjustment to chronic pain, participants underwent a submaximal effort tourniquet procedure. This experimental pain procedure yields a measure of tolerance for a controlled noxious stimulus (ie, arm ischemia). **Results:** African American subjects reported higher levels of clinical pain as well as greater pain-related disability than white participants. In addition, substantial group differences were observed for ischemic pain tolerance, with African Americans demonstrating less tolerance than whites. Correlational analyses revealed a small but significant inverse relationship between ischemic pain tolerance and the reported severity of chronic pain. **Conclusions:** Collectively these findings support previous research revealing ethnic differences in responses to both clinical and experimental pain. Moreover, the present results suggest that enhanced sensitivity to noxious stimuli on the part of African Americans may be associated with ethnic differences in reported clinical pain, although the magnitude of ethnic differences was much greater for ischemic pain tolerance than for clinical pain measures. **Key words:** race, ethnicity, ischemic pain, chronic pain, pain sensitivity.

BDI = Beck Depression Inventory; IPTO = ischemic pain tolerance; MPI = Multiphasic Pain Inventory; MPQ = McGill Pain Questionnaire; ODQ = Oswestry Disability Questionnaire; STAI = State-Trait Anxiety Inventory.

INTRODUCTION

Since Zborowski's pioneering work on ethnic and cultural differences in the experience of pain (1–3), numerous clinical studies investigating a wide variety of painful conditions have noted ethnic differences in the prevalence and severity of pain. Specifically, investigators have recently indicated that African Americans report greater levels of pain than whites for such conditions as glaucoma (4), AIDS (5), migraine headache (6), jaw pain (7), postoperative pain (8, 9), myofascial pain (10, 11), angina pectoris (12), joint pain (13), nonspecific daily pain (14), and arthritis (15, 16). Interpretations of such findings remain difficult, however, because of potential group differences in disease severity (6) and physician management (17, 18). Indeed, several studies have suggested that physicians tend to prescribe less analgesic medication for African Americans than for whites (19–21). However, although ethnic differences in pain reports may often be confounded with ethnic variations in health professionals' management of pain, the finding that African Americans report greater pain than whites seems to be quite robust across a wide variety of age groups and populations. Specifically, findings of greater self-reported clinical pain in African Americans relative to whites appear in young children with signs of temporomandibular disorders (6), healthy college students (14), and community samples of disabled elderly individuals (13).

Although the underlying mechanisms remain unclear, it has been suggested that enhanced pain sensitivity on the part of African Americans might partially explain the observed ethnic differences in the reported frequency and severity of clinical pain (14). That is, if African Americans were more sensitive to noxious stimuli, then they would be expected to experience relatively greater clinical pain. However, although the clinical literature on this topic is expanding, only a handful of experimental studies have examined differences between African Americans and whites in responses to experimental pain. In a review of this literature, Zatzick and Dimsdale (22) identified three relevant studies, each of which reported increased pain tolerance among whites relative to African Americans. In a large-sample study ($N > 40{,}000$), Woodrow et al. (23) reported greater pressure pain tolerance among whites relative to African Americans. Similarly, Walsh et al. (24) noted greater pain tolerance among whites on the cold pressor test. Finally, Chap-

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man and Jones (25) reported reduced heat pain thresholds and tolerances among African American subjects in comparison to white subjects. These studies, widely separated in time and using diverse methodologies, collectively suggest differences between African Americans and whites in responses to a variety of experimental noxious stimuli. In addition, we recently reported that in a sample of healthy college students, African American subjects demonstrated lower thermal pain tolerances and higher ratings of pain unpleasantness in response to noxious thermal stimuli than did white subjects (14). In addition, Sheffield et al. (26) recently reported ethnic differences in ratings of the unpleasantness of noxious thermal stimuli, with African Americans rating the thermal stimuli as more unpleasant than whites.

The present study sought to extend previous research by investigating ethnic differences in both reported clinical pain and in experimental pain tolerance in a population of patients with chronic pain. Few studies have examined ethnic differences in both clinical and experimental pain in a single sample; use of this methodology permits a comparison of the relative size of ethnic differences as well as an evaluation of relationships between clinical and experimental pain. In particular, this study attempted to determine whether African American and white chronic pain patients differed on a measure of tolerance for a controlled noxious stimulus (ie, ischemic pain tolerance using a submaximal effort tourniquet procedure) or on their reported adjustment to chronic pain (eg, report of pain-related distress, pain severity, and disability).

METHODS

Participants

Participants in the present study were 337 consecutive chronic pain patients (68 African Americans, 20%) completing an interdisciplinary pain treatment program (27). Patients were referred by physicians, rehabilitation nurses, or insurance companies for evaluation and treatment of chronic noncancer pain. Inclusion criteria were not restricted according to etiology or location of pain because the rather general measures used in the present study were designed for use with a variety of chronic pain and other populations. Primary sites of pain included the lower back (54%), leg (11%), shoulder or arm (11%), neck (6%), and other (18%). The "modal" patient reported primary pain in the lower back with radiation into one or both legs. The mean age of participants, duration of pain, total number of pain locations reported, and number of prior surgeries for pain are reported in Table 1. In addition, data were collected on medication use at the time of admission to the program. Percentages of subjects taking opioids, antidepressants, benzodiazepines, and muscle relaxants are also reported in Table 1.

TABLE 1. Demographic and Other Information for African American and White Participants (N = 337)

Variable	Total Sample	African American	White	p
Age (y)	40.1	40.6	40.0	NS
Sex (% male)	60	52	63	<.1
Duration of pain (mo)	29.5	27.0	30.1	NS
Previous surgeries for pain (N)	1.3	.8	1.4	<.01
Pain locations (N)	2.6	2.6	2.4	NS
Opioids[a] (%)	50	56	49	NS
Benzodiazepines[a] (%)	23	16	25	NS
Muscle relaxants[a] (%)	28	37	26	<.1
Antidepressants[a] (%)	39	31	41	NS

[a] Patients taking medication on admission to the program.

Measures

Before entry into the treatment program, each subject completed the following self-report measures and behavioral assessment procedures, all of which are routinely performed as part of the evaluation and treatment of patients entering the pain management program.

Multidimensional Pain Inventory. The MPI (28) consists of 13 scales divided into three sections assessing the impact of pain on the patient's life, responses of significant others to the communication of pain, and the daily activity level of the patient. The reliability and validity of the MPI are well-established (28, 29). In the present study, the MPI subscales of pain severity, pain interference, affective distress, perceived life control, and general activity level were used as an index of adjustment to chronic pain. MPI data were scored using the MPI computerized scoring program; subscale scores are presented as T scores.

Beck Depression Inventory. The BDI (30) is a widely used, 21-item, self-report measure assessing common cognitive, affective, and vegetative symptoms of depression. Research evaluating the psychometric properties of the BDI suggests that it has excellent reliability and validity as an index of depression (31).

Oswestry Disability Questionnaire. The ODQ (32) is a 10-item, self-report scale assessing the extent to which functional daily activities are restricted by pain. Prior psychometric evaluations of the ODQ have revealed that it has adequate reliability and validity (33); furthermore, the ODQ has been recommended as a standard tool for the assessment of pain-related disability (34).

McGill Pain Questionnaire. The MPQ (35) consists of 20 groups of single-word pain descriptors with the words in each group increasing in rank-order intensity. The sum of the rank values for each descriptor based on its position in the word set results in a score termed the Pain Rating Index; the Pain Rating Index was the summary score used in the present study. The MPQ is among the most widely used measures for rating pain; it is used extensively in many countries and has taken on the status of a gold standard against which other, newer instruments are compared (36).

State-Trait Anxiety Inventory. The state version of the STAI (37) is a self-report scale consisting of 20 statements evaluating recent levels of anxiety. Each item is scored on a four-point Likert scale, with higher scores representing increasing levels of state anxiety (recent, situationally derived symptoms of anxiety). The STAI has consistently demonstrated adequate psychometric properties and is among the most commonly used measures of anxiety (37–39).

Pain ratings. Subjective pain ratings were obtained by asking each patient to record hourly ratings of pain severity on a scale of 0

(no pain) to 10 (the most severe pain imaginable) (27). For the purposes of statistical analysis, hourly pain ratings for the first 2 days of treatment were averaged to yield a single overall pain rating.

100-yard timed walking test. Although self-report measures of physical function are more commonly used, behavioral measures of physical function have been widely recommended for use in assessment of patients with chronic pain (40, 41). Simple, standardized tests of physical function, such as timed walking tests, have repeatedly demonstrated good reliability, validity, and predictive utility in a variety of rehabilitation populations (40, 41). In the present study, patients were required to walk once around a 100-yard-long indoor circular track as fast as they could. Performance was measured as the number of seconds required to complete the task.

Ischemic pain tolerance. IPTO was assessed using the modified submaximal tourniquet procedure as described previously (42, 43). This procedure involves exercising the hand as blood flow to the arm is occluded, evoking ischemic pain. After determination of subjects' maximum grip strength, the arm was exsanguinated by elevating it above heart level for 30 seconds, after which a standard blood pressure cuff was positioned proximal to the elbow of the dominant arm and inflated to 200 mm Hg. Subjects then performed 20 handgrip exercises of 2 seconds duration at 4-second intervals at 50% of maximum grip strength. Subjects were instructed to continue until the pain became intolerable; thus, all subjects were free to discontinue the procedure at any time. The procedure was terminated by the experimenter if pain tolerance had not been achieved at 20 minutes. IPTO was operationally defined as the number of seconds to pain tolerance after commencement of handgrip exercises. Patients with a history of hypertension, coronary artery disease, or upper extremity pain of unknown etiology were excluded from the test. Patients who were not excluded for these criteria completed the procedure before beginning the interdisciplinary treatment program.

Data Reduction and Analysis

Data are presented as means and standard errors. Because data points were missing for a number of subjects, the number of individuals included in the analysis, as well as the ethnic distribution of available subjects, is included in each table. Because of distributional nonnormality, as well as group differences in sample sizes and variability, the significance of simple group differences was determined using the Wilcoxon rank-sum test as recommended by Wilcox (44, 45). Group differences on nominal variables were assessed using χ^2 tests. To reduce the number of variables to a smaller, coherent aggregate and to reduce the likelihood of Type I error, an exploratory principal components analysis with varimax rotation was performed on all available questionnaire responses as well as 100-yard walking times. The scree test (46, 47) was used to determine the number of factors retained. The derived factors were then used in further analyses. Relationships between continuous variables were examined using Pearson correlations. Significance was set at $p < .05$ for each analysis. All analyses were performed using SAS software.

RESULTS

Demographics

No ethnic group differences emerged for the following variables: age, primary pain location, pain duration, total number of reported pain locations, or percentage of individuals taking opioids, benzodiazepines, or antidepressants (p values $> .1$) (Table 1). A significant difference was noted in the number of previous surgeries, with whites reporting more previous pain-related surgeries than African Americans (Table 1). In addition, marginally significant differences were noted in the sex ratios of the two groups (the African American group consisted of a slightly higher percentage of women than the white group) and in the percentage of individuals taking muscle relaxants (African Americans were somewhat more likely to be taking muscle relaxants on admission to the program) (Table 1).

Factor Analysis

The following variables were subjected to a principal-components factor analysis with varimax rotation (47): the MPI subscales of pain severity, pain interference, affective distress, life control, and general activity level; BDI scores; STAI state anxiety scores; ODQ scores; MPQ scores; pain ratings (0–10); and 100-yard walking times. Before reducing these data, Wilcoxon rank-sum tests were performed on individual variables to determine the extent of group differences for specific measures. Results of these tests are given in Table 2.

The factor analysis yielded a three-factor solution (ie, only the first three factors had eigenvalues >1) that accounted for 57% of the variance in these variables. The first factor had high loadings on the following variables: BDI, STAI, and the MPI subscales of affective distress and life control (negative loading). This factor had an eigenvalue of 2.4, explained 22% of the variance, and seemed to reflect the degree of reported emotional distress. Therefore, we labeled this factor "emotional distress," with higher scores on this factor representing increasing levels of emotional distress. The second factor had high loadings on the MPI pain severity subscale, the MPQ, and the pain ratings. This factor had an eigenvalue of 2.0, explained 18% of the variance, and seemed to be constituted by variables reflecting perceived pain severity. This factor was labeled "perceived pain severity"; again, higher factor 2 scores reflect increased reports of pain severity. Finally, the third factor had high loadings on the ODQ, 100-yard walking times, and the MPI subscales of pain interference and general activity level (negative loading). The eigenvalue for the third factor was 1.8, and this factor explained 17% of the variance. This factor comprised variables measuring pain-related physical disability; it was therefore labeled "pain-related disability." As with the two previous factors, increasing scores on factor 3 represent greater levels of pain-related disability. Table 3 shows the factor structure of the rotated three-factor solution.

TABLE 2. Group Comparison (African American vs. White) for Scores on Individual Variables

Variable	Total Subjects (% African American)	African American Mean (SD)	White Mean (SD)	Z Score (Difference)	*p*
Pain severity[a]	317 (21%)	55.7 (9.1)	53.4 (8.0)	2.4	<.01
MPQ[b]	306 (20%)	36.3 (13.2)	36.5 (12.2)	0.1	NS
Average pain rating	330 (20%)	6.9 (1.5)	6.5 (1.3)	2.1	<.05
ODQ[c]	318 (20%)	58.2 (12.3)	53.3 (12.2)	3.1	<.01
Walk time(s)	311 (20%)	80.2 (21.9)	73.3 (21.2)	2.8	<.01
Pain interference[a]	316 (21%)	56.0 (5.9)	55.1 (6.0)	1.2	NS
General activity[a]	315 (21%)	42.6 (7.5)	43.9 (8.1)	−1.1	NS
Affective distress[a]	316 (21%)	52.9 (7.6)	52.8 (7.8)	−0.4	NS
Life control[a]	316 (21%)	50.9 (9.9)	48.4 (9.3)	2.1	<.05
BDI[d]	327 (21%)	23.6 (10.0)	22.2 (10.0)	0.9	NS
STAI (state)[e]	325 (20%)	53.8 (9.9)	54.6 (12.1)	−0.7	NS

[a] MPI subscale variables are measured as *T* scores (mean = 50, SD = 10).
[b] MPQ scores range from 0 to 78. The mean for Melzack's (35) original sample was 23.0.
[c] Scores range from 0 to 100. Scores of 40 to 60 represent substantial disability; scores >60 represent severe disability.
[d] Scores ≥18 reflect clinically significant levels of depression.
[e] The norm for working adults is 36; mean scores between 45 and 55 are typical of psychiatric patients with symptoms of anxiety.

TABLE 3. Factor Loadings for Principal Axis, Varimax-Rotated, Three-Factor Solution

Variable	Factor 1	Factor 2	Factor 3
Pain severity (MPI)	0.09	0.75[a]	0.25
MPQ	0.23	0.58[a]	−0.10
Pain ratings	0.10	0.80[a]	0.16
ODQ	0.06	0.46	0.51[a]
Walk time	−0.07	0.20	0.72[a]
Pain interference (MPI)	0.40	0.29	0.55[a]
General activity (MPI)	−0.25	0.15	−0.71[a]
Affective distress (MPI)	0.67[a]	0.29	−0.07
Life control (MPI)	−0.79[a]	−0.02	−0.08
BDI	0.66[a]	0.18	0.25
STAI (state)	0.75[a]	0.07	0.11

[a] Primary factor loadings.

Ethnic Differences in Clinical and Experimental Pain

Next we examined potential ethnic differences in clinical pain presentation (as assessed by the three factors indicated above) as well as differences in ischemic pain tolerance. Although no group differences were evident for the factor tapping emotional distress ($p > .1$), statistically significant effects of ethnic group were observed for perceived pain severity, pain-related disability, and IPTO (p values $< .05$). African American participants reported significantly greater perceived pain severity and pain-related disability than whites; in addition, whites demonstrated greater tolerance for the ischemic pain stimulus. These effects remained statistically significant (p values$< .05$) after other variables that differed between the groups (ie, number of previous surgeries, sex, and the percentage of individuals taking muscle relaxants) were entered as covariates. Results of these analyses are presented in Table 4.

Clinical Relevance of IPTO

The clinical relevance of IPTO was assessed by examining correlations between IPTO and clinical pain measures. Pearson product-moment correlation coefficients for the overall sample, as well as within ethnic groups, are shown in Table 5. In general, correlations between IPTO and individual variables were sporadic; the three significant correlations (with BDI, MPQ, and ODQ scores) are presented in Table 5. When correlations between IPTO and factor scores were examined, a significant inverse relationship was observed between IPTO and factor 2, perceived pain severity. No

TABLE 4. Group Differences in Factor Scores and in Ischemic Pain Tolerance

Variable	Total (% African American)	African American Mean (SD)	White Mean (SD)	Z Score	*p*
Emotional distress, factor 1	242 (20%)	2.0 (9.6)	4.3 (10.0)	−1.2	NS
Perceived pain severity, factor 2	242 (20%)	66.9 (6.8)	64.1 (7.3)	2.4	<.01
Pain-related disability, factor 3	242 (20%)	29.4 (14.1)	23.9 (13.4)	2.5	<.01
IPTO (s)	254 (19%)	296 (194.3)	525 (331.9)	−4.6	<.0001

TABLE 5. Pearson Correlations Between IPTO and Clinical Pain Measures (Selected Individual Variables and Factor Scores) Across and Within Ethnic Groups

Variable	Full Sample	African American	White
Factor 1 (distress)	−0.09	−0.02	−0.11
Factor 2 (pain severity)	−0.20**	−0.05	−0.18*
Factor 3 (disability)	−0.09	0.19	−0.11
BDI	−0.17**	−0.12	−0.14*
MPQ	−0.18**	−0.41**	−0.20*
ODQ	−0.14*	0.25	−0.08

* $p < .05$; ** $p < .01$.

significant relationships between IPTO and the distress or disability factors were observed.

DISCUSSION

The results of the present study suggest ethnic differences in the reported severity of chronic pain, in chronic pain–related disability, and in tolerance for a controlled noxious stimulus. Specifically, after data reduction to a set of three empirically derived and theoretically meaningful factors, ethnic group differences were observed on the factors measuring the reported severity of chronic pain and chronic pain–related disability, with African Americans reporting a somewhat greater severity of pain and slightly more pain-related disability than whites. In addition, African Americans demonstrated lower ischemic pain tolerance than whites using a standard experimental pain procedure. On average, although white participants tolerated the ischemic arm pain for nearly 9 minutes, African American participants terminated the procedure at approximately 5 minutes. Collectively, the present results are consistent with previous investigations reporting ethnic differences in reported severity of chronic myofascial pain (10, 11) and tolerance for experimental pain stimuli (14, 22–26). Additional findings suggested the clinical relevance of an experimental pain procedure; an inverse relationship emerged between IPTO and the perceived severity of clinical pain, suggesting that individuals reporting greater clinical pain tend to demonstrate lower ischemic pain tolerances. To our knowledge, the present findings are the first to document ethnic differences in both chronic pain (reported severity and impact of pain) and experimental pain sensitivity (ischemic pain tolerance) within a single study. Furthermore, these results parallel previous findings among healthy college students (14), providing similar results from a large sample of individuals with chronic pain.

The observed ethnic differences cannot be attributed to demographic factors, pain characteristics (pain duration, pain location, number of pain sites, and number of previous surgeries), or patterns of medication usage because the two groups were either comparable on these variables or the observed effects remained present after statistically controlling for group differences. Furthermore, ethnic differences in clinical pain reports and experimental pain tolerance did not seem to be due to mood or emotional distress because African American and white participants did not differ on measures of depression, anxiety, or overall affective state. Collectively, as noted by Zatzick and Dimsdale (22), the greatest difficulties seem to lie not in the measurement of ethnic differences in pain but in the explanation of these differences. It may be that measures of both experimental pain tolerance and chronic pain severity tap an underlying construct such as pain sensitivity, which differs across ethnic groups. However, the magnitude of ethnic differences in ischemic pain tolerance was substantially greater than the rather small magnitude of group differences in reported clinical pain, suggesting the possibility that different mechanisms may operate to produce these effects.

Laboratory pain measures (eg, IPTO) are influenced by a wide array of factors, including peripheral transduction mechanisms, central nervous system mechanisms of pain modulation, personality, sociodemographic characteristics, and psychological variables (22, 48–51). It has been suggested that ethnic differences in pain responses may emerge as a consequence of ethnic differences in one or more of these other variables (48, 49). However, prior studies have suggested that the following factors do not account for differences between African Americans and whites in pain responses: personality (10), anxiety (11), education (9, 14), family history of pain (14), attentional variables (14), and peripheral mechanisms of nociception (22, 48). Furthermore, the present study indicates that mood and other affective factors, as well as general efficacy beliefs (measured by the MPI life control scale), are unlikely to account for ethnic differences in IPTO. However, other potentially important variables, such as coping, social learning, or attitude, may have influenced the present pattern of findings. At least one prior study has suggested that use of specific coping strategies may differ across ethnic groups (52). Moreover, Moore and Brodsgaard (53) note that coping styles generally vary widely across cultures and that cultural differences in use of pain coping strategies may be at least as important as differences in the prevalence or reported severity of pain. Alternatively, ethnic differences in attitudinal variables related to measurement of pain may have played a role in these findings. Pervasive mistrust of the medical research community has been documented among African

Americans (54, 55), and it is certainly possible that a less trusting attitude among African Americans might have contributed to greater report of pain or more rapid termination of the ischemic pain procedure.

One other possibility may merit consideration here. Specifically, ethnic differences in central pain-regulatory systems might account for the observed findings. In one prior study, subgroups of African Americans and whites evidenced differences in circulating β-endorphins in response to stress (56), with African American hypertensives demonstrating significantly lower β-endorphin levels. Because exogenous opioids have been reported to attenuate the experience of both laboratory and clinical pain (57), ethnic differences in pain-related endogenous opioid release could produce the effects observed in this and other studies. An alternative explanatory mechanism involving central factors relates to cardiovascular and associated adrenergic responses to stress. The ischemic pain task is a potent cardiovascular challenge, as is the experience of pain in general (43). Pain, as a stressor, is associated with enhanced epinephrine release and cardiovascular reactivity (58), both of which are associated with increased report of pain (59–62). Because African Americans demonstrate greater adrenergic and vascular responses to stress than do whites (63, 64), such effects could potentially contribute to the observed ethnic differences in clinical and experimental pain. However, this possibility remains speculative in the absence of direct evidence implicating physiological mechanisms.

The present study includes a number of limitations that may restrict the generalizability of the results. First, specification of differing ethnic subgroups or cultures within the broad categories of "African American" and "white," which might allow for subtle intraethnic distinctions, was not possible. Several previous investigations have reported differences in laboratory pain responses among several ethnic subgroups within overarching racial categories (25, 65). Moreover, degree of cultural affiliation and degree of acculturation, variables that have previously been demonstrated to have relationships to pain responses (50), were not measured in this investigation. Second, the present study suffers from the nearly universal limitations identical to those noted in most investigations of patients presenting to multidisciplinary pain clinics, that is, multiple selection biases. That is, little is known about referral patterns from various healthcare personnel or about individual patient decisions to accept (or reject) such referrals. It is certainly possible that the ethnic differences observed in this study are partially a consequence of unintended differential referral or referral acceptance patterns among whites and African Americans. Third, no underlying mechanisms producing ethnic differences in pain responses could be identified, and our hypotheses remain speculative. Fourth, the present study did not assess experimenter characteristics such as race and sex, which may potentially have contributed to the observed results. Zatzick and Dimsdale (22) note that the ethnicity of the experimenter may be an important, and understudied, variable in explaining group differences in responses to laboratory pain procedures. Although we have previously documented the absence of such experimenter effects (14), we cannot rule out the possibility that they may have influenced the present pattern of findings. Fifth, no measures of coping with pain were included in the present study. Future investigations may benefit from assessment of coping strategies as a potential mediator or moderator of relationships between ethnicity and pain responses. Sixth, no patient data on psychiatric diagnoses were available in this sample; thus, we cannot rule out the possibility that group differences in psychiatric symptoms influenced ethnic differences in pain. Seventh, relationships between IPTO and clinical pain variables in the present study were rather inconsistent and did not explain large proportions of variance, leaving much of the individual variability in chronic pain severity and chronic pain–related disability unaccounted for.

In conclusion, although explanations underlying ethnocultural differences in pain perception remain elusive, these results suggest that African Americans demonstrate greater reported severity of chronic pain, more pain-related disability, and decreased tolerance for a controlled noxious stimulus than whites. It should also be noted that although substantial ethnic differences were observed for ischemic pain tolerance, differences associated with clinical pain variables, although significant, were quite modest. In addition, a small but significant inverse relationship was observed between ischemic pain tolerance and the reported severity of chronic pain, although the much larger magnitude of ethnic differences in pain tolerance may suggest that different mechanisms operate to produce these effects. Future studies examining ethnic differences in painful clinical conditions may benefit from additional measurement of responses to a controlled noxious stimulus. The implications of the present findings for clinical populations presenting with pain remain unclear. However, although an emerging body of evidence documents undertreatment of pain among African Americans (17–21), these results suggest slightly greater severity and interference of chronic pain in this ethnic group. Collectively, the bulk of available evidence has supported the findings of differential responses to pain among various ethnic

groups. The present study extends this research and highlights the potentially valuable role of laboratory pain stimuli in elucidating ethnic group differences in clinical pain.

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