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# Differences in Clinical Pain and Experimental Pain Sensitivity between Asian Americans and Whites with Knee Osteoarthritis

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## Abstract

**Objective—**Ethnicity has been associated with clinical and experimental pain responses. While ethnic disparities in pain in other minority groups compared to whites are well described, pain in Asian Americans remains poorly understood. The purpose of this study was to characterize differences in clinical pain intensity and experimental pain sensitivity among older Asian American and non-Hispanic White (NHW) participants with knee osteoarthritis (OA).

**Methods—**Data were collected from 50 Asian Americans ages 45-85 (28 Korean, 9 Chinese, 7 Japanese, 5 Filipino, and 1 Indian) and compared to 50 age- and gender-matched NHW individuals with symptomatic knee OA pain. The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and Graded Chronic Pain Scale (GCPS) were used to assess the intensity of clinical knee pain. In addition, quantitative sensory testing was used to measure experimental sensitivity to heat- and mechanically-induced pain.

**Results—**Asian American participants had significantly higher levels of clinical pain intensity than NHW participants with knee OA. In addition, Asian American participants had significantly higher experimental pain sensitivity than NHW participants with knee OA.

**Discussion—**These findings add to the growing literature regarding ethnic and racial differences in clinical pain intensity and experimental pain sensitivity. Asian Americans in particular may be at risk for clinical pain and heightened experimental pain sensitivity. Further investigation is needed to identify the mechanisms underlying ethnic group differences in pain between Asian Americans and non-Hispanic Whites, and to ensure that ethnic group disparities in pain are ameliorated.

### Keywords

Racial/ethnic differences; quantitative sensory testing; osteoarthritis; Asian American

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## Introduction

Arthritis is one of the leading causes of pain, impairments of activities in daily life, and disability in people aged 45 and above.[1-3] Of the 53 million adults diagnosed with arthritis, more than 22 million (42%) reported trouble performing their daily activities due to arthritis.[3] Osteoarthritis (OA) is the most common of the arthritic conditions, with the knee being the most commonly affected joint,[2,4,5] and some studies show that the prevalence and severity of knee OA differs across ethnic groups. For example, compared to non-Hispanic whites (NHWs), a greater proportion of African Americans not only have knee OA,[5,6] but also greater pain-related disabilities.[7,8] In addition, OA prevalence is higher among Asian Americans compared to NHWs.[9,10] For example, Zhang and colleagues reported that symptomatic knee OA in Chinese women is higher by 16 – 75% than among age-matched white women.[9] However, few studies have examined whether the severity of clinical pain in Asian Americans with knee OA differs from that of their NHW counterparts. Asian American is the fastest growing ethnic group in the United States, increasing by 46% between 2000 and 2010, and is estimated to be the fastest growing ethnic group over the next 40 years. Thus, understanding pain experiences of Asian Americans warrants increased empirical attention.

In addition to ethnic group differences in clinical pain responses, several studies have documented ethnic differences in responses to experimentally induced pain.[11] The most common comparisons have involved African Americans and NHWs.[12,13] These studies have revealed that African Americans may have higher experimental pain sensitivity compared to NHWs.[13,14] While it is generally believed that Asian Americans' pain experiences do not differ from those of non-Hispanic Whites,[15] several studies in healthy young Asian American participants have reported greater experimental pain sensitivity in Asian Americans compared to NHWs.[16-21] Whether similar differences in experimental pain sensitivity exist in older Asian Americans has not been determined. Thus, more research needs to be conducted to elucidate the differences in pain between ethnic groups and to reveal more about the underlying mechanisms of pain and disability in Asian Americans. This knowledge may lead to the development of targeted interventions that optimize pain management in this understudied population.

The primary aim of this study was to examine ethnic differences in clinical pain intensity and experimental pain sensitivity among older Asian Americans compared to age- and gender-matched NHWs with knee OA. We hypothesized that Asian Americans would display (1) higher levels of self-reported clinical pain intensity, (2) a lower pain threshold and tolerance for heat- and mechanically-induced pain, and (3) a greater temporal summation of pain, suggesting greater pain facilitation among Asian Americans compared to NHWs with knee OA.

## Materials and Methods

### Study Participants

Participants in this study were individuals residing in north-central Florida with knee OA pain. Fifty Asian American participants for this project were recruited via posted fliers and

an email advertisement sent to Asian community listservs between June 2014 and October 2014. Participants from 45 – 85 years old were considered eligible if they (1) were Asian American by self-report, (2) could speak and read English, (3) could provide written informed consent prior to enrollment, and (4) had self-reported unilateral or bilateral knee OA pain. Participants were excluded if they had concurrent medical conditions that could confound symptomatic OA-related outcome measures or coexisting diseases that could hinder the completion of the protocol including: (1) prosthetic knee replacement, (2) serious medical illness, such as uncontrolled hypertension (blood pressure > 150/95), heart failure, or a history of acute myocardial infarction, (3) peripheral neuropathy, (4) systemic rheumatic disorders including rheumatoid arthritis, systemic lupus erythematosus, and fibromyalgia, (5) daily use of opioids, (6) cognitive impairment (Mini-Mental Status Examination [MMSE] < 23/30), and (7) hospitalization within the preceding year for psychiatric illness. The comparison group data were obtained from gender- and age-matched NHW individuals randomly selected from a previous study completed between January 2010 and October 2013, “Understanding Pain and Limitations in Osteoarthritic Disease (UPLOAD).” Members of this group were recruited with methods and underwent testing protocols similar to those of the present study.[12] Four age groups: 45-55 years, 56-65 years, 66-75 years, and 76-85 years were used for age-matching. The random selection of comparison individuals was performed using SAS software (version 9.3) to reduce selection bias.

## General study procedures

The study was approved by the Institutional Review Board of the affiliated university prior to commencement. After informed consent was obtained, the study participants completed a general health and demographic questionnaire, including age, gender, education level, height, weight, and employment status. Participants then completed clinical questionnaires, followed by quantitative sensory testing (QST), including thermal and mechanical testing. Thermal and mechanical test order was counterbalanced and randomized. Recorded instructions were played prior to commencement of each QST procedure. To control experimenter bias in both this and the UPLOAD study, two experimenters conducted each experimental session, one of whom was of the same race/ethnicity as the participant. Also, to maintain consistency one of the experimenters was kept constant within each ethnic group. All participants listened to digitally recorded instructions, and the experimenter provided information only when asked by participants. The study procedure for Asian American participants was the same as the UPLOAD study[12] for NHW participants.

## Clinical pain and functional impairments

The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)[22] was used to assess symptoms of knee OA in the past 48 hours using a 4-point Likert scale. WOMAC yields subscales relating to pain during activities (5 items), stiffness during the day (2 items), and impairments of physical function (17 items), with higher scores indicating worse pain, stiffness, and impairments of physical function. WOMAC has been reported to have Cronbach's alpha coefficient of 0.86 – 0.89 among patients with knee and hip OA.[22] Also, the Graded Chronic Pain Scale (GCPS)[23] was used to measure global pain intensity and interference with activities within the previous six months. With a 0 – 10 numeric scale, participants were asked to rate their current pain, and the worst and average pain during the

past 6 months. These 3 items were averaged and multiplied by 10 to generate pain intensity scores. Using the same scale, participants were asked to rate the degree to which their knee pain interfered with daily activities (3 items) during the past 6 months, and these 3 items were averaged and multiplied by 10 to generate a disability score. The GCPS scores of pain intensity and disability show excellent reliability with Cronbach's alpha coefficient at 0.94 – 0.95 among persons with chronic pain.[24]

## QST procedures

**Thermal testing procedures—**Contact heat stimuli were delivered using a computer-controlled Medoc Pathway Neurosensory Analyzer to measure heat pain thresholds and heat pain tolerances on both the index knee and the ipsilateral ventral forearm using an ascending method of limits. The thermode position was moved among 3 sites between trials to avoid sensitization and/or habituation of cutaneous receptors. From a baseline of 32°C, the thermode temperature increased at a rate of 0.5°C per second until the participants responded by pressing a button on a handheld device. In order to assess heat pain threshold, participants were instructed to press the button when the sensation “first becomes painful.” Similarly, to assess heat pain tolerance, participants were instructed to press the button when they “no longer feel able to tolerate the pain.” The results of the 3 individual trials were averaged to generate an overall heat pain threshold temperature and heat tolerance temperature, which were used for analysis.

Five minutes following the assessment of heat pain threshold and tolerance, temporal summation of thermal pain was assessed on both the index knee and the ipsilateral ventral forearm by having the participant verbally rate the intensity of peak pain evoked by each of the 5 brief, repetitive, suprathreshold heat pulses on a scale of 0 (no pain sensation) to 100 (the most intense pain sensation imaginable). Three target temperatures (44°C, 46°C, and 48°C) were delivered 5 times by a contact heat-evoked potential stimulator thermode for less than 1 second, with an approximately 2.5-second interpulse interval during which the temperature of the contactor returned to the baseline temperature (32°C). The procedure was terminated if the participants wanted to stop the procedure or rated the thermal pain at 100. Participants were asked to rate the pain when they asked to stop the procedure. The average rating over the 5 trials, an index of overall sensitivity to suprathreshold heat-pain, and the maximum increase in pain, a measure of temporal summation, were calculated by subtracting the first trial rating from the maximum rating provided at each temperature. The average rating and temporal summation were used in the analyses.

**Mechanical testing procedure—**A handheld Medoc digital pressure algometer (Algomed) was applied at a constant rate of 30 kPa per second to measure the pressure pain threshold at 5 sites: the medial aspect of the index knee, lateral aspect of the index knee, ipsilateral quadriceps, trapezius, and dorsal forearm. The order of testing sites was counterbalanced and randomized. For assessing the pressure pain threshold, participants were instructed to press the button when the sensation “first becomes painful,” and the pressure was recorded. The results of the 3 individual trials were averaged to generate an overall pressure pain threshold, which was used for analysis.

Five minutes following the assessment of the pressure pain threshold, sensitivity to punctate mechanical stimuli was assessed on both the index patella and back of the ipsilateral hand by obtaining verbal ratings of the intensity of pain evoked by a calibrated nylon monofilament delivering a target force of 300 grams. The order of testing sites was counterbalanced and randomized. Participants verbally rated pain intensity on a scale of 0 (no pain sensation) to 100 (the most intense pain sensation imaginable) following a single contact and after 10 contacts, at a rate of 1 contact per second. The procedure was repeated twice, and the ratings for a single contact and for 10 contacts were averaged to generate a measure of temporal summation of punctate pain, which was calculated by subtracting the pain ratings of the single contact from the pain rating after 10 contacts. The temporal summation of mechanical pain was used in the analyses.

### Statistical analyses

All analyses were conducted with SAS version 9.4.[25] Descriptive statistics appropriate for measurement level were used to validate values and evaluate variable distributions and missing data patterns. To accommodate the matched pairs design, a general linear mixed model approach, employing SAS PROC MIXED[25] was used to test for differences between Asian and NHW participants. This approach appropriately handles the dependencies introduced by the matched pairs design and the repeated measurements of individuals (e.g.: testing at different positions on the body).[26-28] The form for the within covariance structure was evaluated by examination of the information criteria, with a selection of the best-fitting form. PROC MIXED default denominator degrees of freedom were used for the models tested. Conformance to statistical model assumptions and the presence of influential observations were evaluated using available diagnostic and influence plots. Where required, Box-Cox transformations or deletion of influential observations was employed. Body mass index [BMI], education level, and employment status were included within the model based on statistically significant differences between Asian Americans and NHWs with knee OA. Interactions with Race were evaluated and retained in the model if statistically significant. Simple main effects analysis[25,29] was performed to characterize those moderation effects. Strength of association was measured by calculating $R^2$ using Vonesh's GOF SAS macro for mixed models.[26] Statistical significance for all tests was set at .05.

## Results

In total, 100 individuals (50 Asian American participants and 50 age- and gender-matched NHW participants) with knee OA were included in this study. The study participants had a mean age of 55 years (SD = ± 8 years), and the majority of each ethnic group was female (n = 31; 62%). Asian American participants originally came from Korea (n = 28; 56%), China (n = 9; 18%), Japan (n = 7; 14%), Philippines (n = 5; 10%), and India (n = 1; 2%), and on average had lived in the United States for about 21 ± 15 years. Asian Americans had a lower mean BMI than NHWs (Asian American 24.16 ± 3.03, NHW 28.63 ± 6.05, $p < .001$) and a higher education level than NHWs ($p = .043$) (Table 1). Based on residual distribution, Box-Cox family transformation was applied to four variables: a natural log transformation was applied to WOMAC, Pressure Pain Threshold, and Temporal Summation of Punctate Pain, while a -0.2 power transformation was applied to Temporal Summation of Heat Pain.

Influential observations were identified in models for Heat Pain Tolerance, Pulse Pain, and transformed Temporal Summation of Heat Pain, but they were retained in the models as their removal did not change decisions about the null hypothesis. Variance Components covariance structures fit best for WOMAC Pain, GCPS Pain, and GCPS Disability models, while unstructured forms fit best for the other models.

### Racial/ethnic differences between Asian Americans and NHWs

Results for each of the general linear mixed models, including simple main effects (SME) analyses, appear in Table 2. Model-estimated (that is, least square) means for NHW and Asian participants appear in Table 3. Strength of association, based on the average model adjusted $R^2$, ranged from very weak for GCPS Disability (0.03) to moderate (0.48) for Heat Pain Tolerance, with a majority (5) being in the moderate (.25-.49) range.

**Natural Log Transformed WOMAC Total—**The final model for WOMAC Total demonstrated a weak association between the set of independent variables and WOMAC Total (average model adjusted $R^2$ = .21). The lack of statistically significant interactions with race indicated that differences between Asians and NHW were not moderated by other variables in the model. Controlling for other variables in the model, Asians had higher (p<.001) WOMAC values (Least Square Means [LSM] of natural log transformed WOMAC score = 2.17) than NHW (LSM of natural log transformed WOMAC score = 1.67).

**GCPS Pain—**There was a weak association (average model adjusted $R^2$ = .21) for the GCPS Pain mixed model. The Race by Gender interaction (p=.02) indicates that race group differences in GCPS Pain varied between males and females. SME results indicated that, for males, NHW (LSM=40.89) values were similar (p=.335) to Asian (LSM=46.42) values. NHW females (LSM=37.21), however, had lower (p<.001) GCPS values than Asians (LSM=59.06).

**GCPS Disability—**This model demonstrated the lowest average model adjusted $R^2$ (0.03), and the only statistically significant effect in the model was race (p=0.013). Asian participants demonstrated a higher GCPS disability score (LSM=41.92) than NHWs (LSM=29.73).

**Heat Pain threshold—**There was a moderate association between the set of independent variables in the mixed model and the Heat Pain Threshold (average model adjusted $R^2$ = .41). Controlling for all other variables in the model, NHW showed higher (p<.001) values (LSM=43.38) than Asian participants (LSM=38.29).

**Heat Pain Tolerance—**The average model adjusted $R^2$ was 0.48, a moderately strong association. The statistically significant race by location interaction (p=.003) indicated that differences between NHW and Asian participants varied between the measured locations. SME analyses demonstrated that the model-estimated mean Heat Pain Tolerance for NHW and Asian participants was different for both forearm (p<.001) and knee (p<.001) locations. While NHW had higher LSM values than Asians at both sites, the difference was larger at the forearm (5.98) than at the knee (4.98).

**Temporal Summation of Heat Pain—**For mean pain ratings during the temporal summation procedure, the average model adjusted $R^2$ was 0.44, a moderately strong association. The statistically significant race by location interaction (p<.001) indicated that differences between NHW and Asian participants varied between the measured locations. SME analyses demonstrated that model-estimated mean heat pain ratings for NHW and Asian participants was different for both forearm (p<.001) and knee (p<.001) locations. NHW had lower LSM values than Asians at both sites, and the difference was larger at the knee (8.64) than at the forearm (6.77).

For the maximum increase in pain across the five trials, the average model adjusted $R^2$ was 0.11, a weak association. The statistically significant race by temperature interaction (p=0.021) indicated that differences between NHW and Asian participants varied between the temperatures employed. SME analyses demonstrated that the model-estimated mean temporal summation of heat pain for NHW and Asian participants was different at both 44 (NHW LSM=0.44; Asian LSM=1.13; p<.001) and 46 degrees Celsius (NHW LSM=0.74; Asian LSM=1.28; p=.007), but were similar at 48 degrees (NHW LSM=1.29; Asian LSM=1.53; p=.238).

**Natural Log Transformed Pressure Pain Threshold—**The average model adjusted $R^2$ was 0.36, a moderate association. Two interaction effects were statistically significant and retained in the final model. The BMI by race interaction (p=.027) resulted from the slope of the association between BMI and pressure pain being different for Asians $\beta\hat{}$= .026 and NHW $\beta\hat{}$= −.030. In Asian individuals, pressure pain threshold tended to increase as BMI increased, while in NHW individuals, pressure pain threshold tended to decrease as BMI increased. The race by location interaction indicates that differences in LSM between NHW and Asian participants varied by location measured. Specifically, NHW participants tended to have larger LSM values (Forearm=5.59; Medial Knee=5.89; Lateral Knee=5.83; Quadriceps=6.13; Trapezius=5.67) than Asians (Forearm=4.86; Medial Knee=5.19; Lateral Knee=5.30; Quadriceps=5.19; Trapezius=5.05).

**Natural Log Transformed Temporal Summation of Mechanical Pain—**The average model adjusted $R^2$ was 0.333, a moderate association. No interaction effects were statistically significant within the final model. Controlling for other variables in the model, NHW participants (LSM=2.53) had lower (p<.001) temporal summation of mechanical pain values than Asian (LSM=3.21).

## Discussion

This study is among the first to compare differences in clinical pain intensity and experimental pain sensitivity between older Asian Americans and NHWs with knee OA. We found several important ethnic group differences in this study. First, Asian American participants with knee OA had significantly higher levels of clinical pain and functional impairment than age- and gender-matched NHW participants, even after adjusting for covariates (e.g., BMI, education level, and employment status). In addition, Asian American participants with knee OA displayed significantly greater sensitivity to heat-induced pain and mechanically-induced pain, including temporal summation of both heat and mechanical

pain, than NHW participants. This greater responsivity to experimentally induced pain among Asian American individuals was observed at both the affected knee and unaffected body sites, suggesting widespread hyperalgesia among Asian American participants with knee OA, perhaps reflecting central pain amplification. Finally, correlations between clinical and experimental pain were found to be weak at best among both Asian Americans and NHWs ($|r| < .3$), consistent with other reports in the literature.[30]

A handful of recent studies have compared experimental pain sensitivity between Asians and NHWs using healthy young participants, and results from these studies are consistent with our findings.[16-21] For example, Lu and colleagues[16] reported that among healthy children ages 8 to 18 in the Los Angeles metropolitan area, Asians demonstrated greater heat pain unpleasantness than whites. Similarly, Rowell and colleagues[17] found that among healthy college students in North Carolina, Asians from South Korea, China, and India demonstrated significantly lower cold pain threshold and tolerance than NHWs. Hsieh and colleagues[18] reported that Chinese participants displayed lower cold pain tolerance than NHWs among college students in Canada. Also, Japanese participants had a significantly lower electric pain threshold than age- and gender-matched NHWs among university students and staff in Belgium,[19] and Asian participants showed lower heat pain thresholds than young healthy male NHW participants in the United Kingdom.[20] Collectively, findings from these studies of healthy children and young adults indicate greater pain sensitivity among Asians compared to Whites, and our study extends these findings to older adults with knee OA.

The mechanisms underlying these differences in pain sensitivity are not fully understood; however, it is well documented that multiple biological factors contribute to pain.[31-33] Indeed, emerging evidence suggests that genetic factors are related to clinical pain and experimental pain sensitivity. For example, the catechol-O-methyltransferase gene (COMT) and mu-opioid receptor gene (OPRM1) have been associated with pain-induced mu-opioid receptor binding,[34] experimental pain sensitivity,[35,36] and risk for developing chronic pain.[32] Ethnic group differences in allele frequencies for polymorphisms of pain-related genes could contribute to ethnic group differences in pain responses. For example, the 118G allele of *OPRM1* polymorphism shows significantly higher frequency in Asian populations[37,38] and has been associated with increased experimental pain sensitivity in Hispanic Americans,[35] in contrast to its association with lower experimental pain sensitivity in non-Hispanic Whites.[35,36] These findings suggest that genetic associations with pain phenotypes may differ as a function of ethnic group, as recently reported,[35] which may contribute to ethnic group differences in pain sensitivity.

In addition to biological factors, psychosocial factors (e.g., pain coping, depression, and stress) are associated with an increase in clinical pain and experimental pain sensitivity.[31,39] It is possible that Asian Americans, an ethnic minority group with a limited history of immigration to the United States, may deal with cross-cultural stresses and develop more sensitivity to pain as they acculturate to the new environment. However, few studies have examined the possibility of underlying psychosocial mechanisms of pain and disability in this population, and more research is needed in this area of inquiry.

Our study showed that, in addition to increased basal experimental pain sensitivity, Asian Americans experienced greater temporal summation of both heat and mechanical pain compared to NHWs. This suggests the possibility of increased central sensitization in Asian Americans with knee OA. Taken together, our findings regarding pain-facilitatory mechanisms extend the data that support increased experimental pain sensitivity in Asian Americans with knee OA. Despite these novel findings, there were some study limitations. First, the comparison data for the NHWs were obtained from a previous study.[12] However, we used the same study procedures as the previous study and the random selection of comparison individuals was performed to reduce selection bias. Second, Asian Americans in our study were limited to English-speaking individuals. However, it is reasonable to conjecture that racial and ethnic group differences might be even greater in non-English-speaking individuals in the United States. Third, the cross-sectional nature of our study to describe racial or ethnic group differences in clinical pain and experimental pain sensitivity provides a snapshot rather than a view of pain over time. Fourth, we included patients with symptomatic osteoarthritis without radiographic examination. Finally, we did not include a control group of Asian Americans and NHWs without OA pain; therefore, we cannot ascertain whether the greater experimental pain sensitivity in our Asian American group is restricted to older adults with clinical pain.

Findings from this study provide an important foundation for future research. First, more research with larger sample sizes and a comprehensive quantitative sensory testing battery needs to be conducted to further elucidate the differences in pain between Asian Americans and NHWs. This would allow an examination of the different types of experimental pain sensitivity (e.g., cold-induced pain) including the pain inhibitory function (e.g. conditioned pain modulation). Second, the underlying biological, cultural, or psychosocial mechanisms that contribute to increased pain and disability in Asian Americans need to be further assessed. These results could lead to targeted interventions that optimize pain management and mobility performance in this understudied population. Third, studies promoting culturally sensitive pain management that considers racial or ethnic group differences in response to pain treatment are needed. Despite growing awareness that pain experience may vary by ethnicity, this knowledge has not yet reached a level of clinical application.

In conclusion, these findings add to the growing literature regarding ethnic and racial differences in clinical pain and experimental pain sensitivity among individuals with knee OA. Further investigation is needed to identify the mechanisms underlying these differences as well as to ensure that ethnic group disparities in pain are ameliorated.

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## Table 1

**Sociodemographic characteristics of participants with knee osteoarthritis by racial/ethnic group**

| Characteristic | Asian American (n=50) | NHW*(n = 50) | P-value |
|---|---|---|---|
| Age, M (SD), years | 55 (8) | 55 (8) | |
| Female, n (%) | 31 (62) | 31 (62) | |
| BMI, M (SD), kg/m$^2$ | 24.16 (3.03) | 28.63 (6.05) | <.001 |
| Education, n (%) | | | .043 |
| Less than high school | 1 (2) | 0 (0) | |
| High school | 7 (14) | 15 (30) | |
| Some college | 8 (16) | 14 (28) | |
| Bachelor's degree | 14 (28) | 12 (24) | |
| Graduate degree | 20 (40) | 9 (18) | |
| Employment Status, n (%) | | | .398 |
| Employed | 35 (70) | 31 (62) | |
| Non-employed | 15 (30) | 19 (38) | |

Note.

*NHW (non-Hispanic White) is age- and gender-matched with Asian American. BMI = body mass index.

## Table 2
## General linear mixed model analysis results

| Effect (Comparison Group) | β (SE) | F (df) | p |
|---|---|---|---|
| WOMAC Pain Scale * | | | |
| Race (White) | -0.51 (0.11) | 20.00 (1,46) | <.001 |
| Gender (Female) | 0.13 (0.11) | 1.45 (1,48) | 0.235 |
| Education (HS or less) | 0.26 (0.13) | 4.34 (1,18) | 0.052 |
| Age | -0.01 (.01) | 0.59 (1,46) | 0.446 |
| BMI | 0.03 (.01) | 7.94 (1,46) | 0.007 |
| GCPS Pain Scale | | | |
| Race (White) | -5.53 (5.67) | 13.50 (1,45) | <.001 |
| Gender (Female) | 12.60 (4.74) | 1.81 (1,48) | 0.185 |
| Education (HS or less) | 3.45 (4.02) | 0.74 (1,18) | 0.402 |
| Age | -0.20 (0.22) | 0.84 (1,45) | 0.364 |
| BMI | 0.81 (0.34) | 5.69 (1,45) | 0.021 |
| Race *Gender | -16.30 (6.72) | 5.89 (1,45) | 0.019 |
| Race\|Female | N/A | 26.20 (1,45) | <.001 |
| Race\|Male | N/A | 0.95 (1,45) | 0.335 |
| GCPS Disability Scale | | | |
| Race (White) | -12.20 (4.71) | 6.70 (1,46) | 0.013 |
| Gender (Female) | 6.75 (4.38) | 2.37 (1,48) | 0.130 |
| Education (HS or less) | 6.54 (5.20) | 1.58 (1,18) | 0.225 |
| Age | -0.19 (0.28) | 0.44 (1,46) | 0.509 |
| BMI | 0.58 (0.45) | 1.70 (1,46) | 0.199 |
| Heat Pain Threshold | | | |
| Race (White) | 5.08 (0.66) | 58.80 (1,48) | <.001 |
| Gender (Female) | -1.50 (0.52) | 8.39 (1,48) | 0.006 |
| Education (HS or less) | 0.16 (0.64) | 0.06 (1,48) | 0.804 |
| Age | -0.001 (0.03) | 0.08 (1,48) | 0.785 |
| BMI | 0.01 (0.05) | 0.01 (1,48) | 0.925 |
| Location (Forearm) | -0.54 (0.26) | 4.38 (1,48) | 0.042 |
| Heat Pain Tolerance | | | |
| Race (White) | 4.49 (0.63) | 78.70 (1,48) | <.001 |
| Gender (Female) | -1.85 (0.50) | 13.80 (1,48) | <.001 |
| Education (HS or less) | 0.14 (0.56) | 0.06 (1,48) | 0.804 |
| Age | -0.02 (0.03) | 0.38 (1,48) | 0.540 |
| BMI | 0.02 (0.05) | 0.29 (1,48) | 0.593 |
| Location (Forearm) | -1.03 (0.31) | 7.04 (1,48) | 0.011 |
| Race *Location | 0.99 (0.32) | 9.95 (1,48) | 0.003 |
| Race\|Forearm | N/A | 84.50 (1,48) | <.001 |
| Race\|Knee | N/A | 63.70 (1,48) | <.001 |
| Mean Pulse Pain | | | |

| Effect (Comparison Group) | β (SE) | F (df) | p |
|---|---|---|---|
| Race (White) | -40.60 (4.70) | 61.10 (1,48) | <.001 |
| Gender (Female) | 6.23 (3.48) | 3.96 (1,48) | 0.052 |
| Education (HS or less) | -5.86 (3.92) | 2.24 (1,48) | 0.141 |
| Age | -0.12 (0.22) | 0.27 (1,48) | 0.607 |
| BMI | 0.23 (0.32) | 0.51 (1,48) | 0.478 |
| Location (Forearm) | -2.05 (1.12) | 7.76 (1,48) | 0.008 |
| Temperature | | 83.50 (2,48) | <.001 |
| (44 Celsius) | -17.24 (1.39) | | |
| (46 Celsius) | -9.88 (0.85) | | |
| Race *Location | 8.08 (1.66) | 23.60 (1,48) | <.001 |
| Race\|Forearm | N/A | 45.80 (1,48) | <.001 |
| Race\|Knee | N/A | 74.70 (1,48) | <.001 |
| Temporal Summation Heat Pain † | | | |
| Race (White) | -0.24 (0.20) | 7.72 (1,48) | 0.008 |
| Gender (Female) | 0.14 (0.13) | 1.18 (1,48) | 0.283 |
| Education (HS or less) | 0.16 (0.15) | 1.13 (1,48) | 0.292 |
| Age | -0.02 (0.01) | 3.57 (1,48) | 0.065 |
| BMI | 0.02 (0.01) | 3.51 (1,48) | 0.067 |
| Location (Forearm) | 0.06 (0.05) | 1.06 (1,48) | 0.309 |
| Temperature | | 23.70 (2,48) | <.001 |
| (44 Celsius) | -0.40 (0.13) | | |
| (46 Celsius) | -0.24 (0.09) | | |
| Race *Temperature | | 4.19 (2,48) | 0.021 |
| White, 44 Celsius | -0.45 (0.16) | | |
| White, 46 Celsius | -0.31 (0.13) | | |
| Race\|44 Celsius | N/A | 12.80 (1,48) | <.001 |
| Race\|46 Celsius | N/A | 7.96 (1,48) | 0.007 |
| Race\|48 Celsius | N/A | 1.43 (1,48) | 0.238 |
| Pressure Pain Threshold * | | | |
| Race (White) | 1.92 (0.56) | 13.10 (1,48) | <.001 |
| Gender (Female) | -0.38 (0.09) | 15.30 (1,48) | <.001 |
| Education (HS or less) | -0.03 (0.11) | 0.10 (1,48) | 0.750 |
| Age | -0.0003 (0.01) | 0.00 (1,48) | 0.966 |
| BMI | 0.02 (0.02) | 0.34 (1,48) | 0.564 |
| Location | | 17.80 (4,48) | <.001 |
| (Forearm) | | | |
| (Medial Knee) | 0.14-.20 (0.07) (0.06) | | |
| (Lateral Knee) | 0.25 (0.07) | | |
| (Quadriceps) | 0.13 (0.07) | | |
| Race *Location | | 8.80 (4,48) | <.001 |
| (White, Forearm) | 0.12 (0.09) | | |

| Effect (Comparison Group) | $\hat{\beta}$ (SE) | F (df) | p |
|---|---|---|---|
| (White, Medial Knee) | 0.09 (0.09) | | |
| (White, Lateral Knee) | -0.09 (0.09) | | |
| (White Quadriceps) | 0.33 (0.09) | | |
| Race\|Forearm | N/A | 44.80 (1,48) | <.001 |
| Race\|Medial Knee | N/A | 42.30 (1,48) | <.001 |
| Race\|Lateral Knee | N/A | 19.90 (1,48) | <.001 |
| Race\|Quadriceps | N/A | 50.90 (1,48) | <.001 |
| Race\|Trapezius | N/A | 26.60 (1,48) | <.001 |
| Race *BMI | | 5.21 (1,48) | 0.027 |
| (White) | -0.05 (0.02) | | |
| Temporal Summation Punctate Pain * | | | |
| Race (White) | -0.68 (0.10) | 43.60 (1, 48) | <.001 |
| Gender (Female) | 0.32 (0.11) | 8.55 (1, 48) | 0.005 |
| Education (HS or less) | 0.23 (0.12) | 3.64 (1, 18) | 0.062 |
| Age | 0.01 (0.01) | 3.90 (1, 48) | 0.054 |
| BMI | -0.01 (0.01) | 0.22 (1, 48) | 0.638 |
| Location (Forearm) | -0.21 (0.05) | 20.90 (1, 48) | <.001 |

Note.

*Natural log transformed scale.

†-0.2 power transformed scale. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index. GCPS: Graded Chronic Pain Scale. Average Model Adjusted $R^2$: Natural Log WOMAC Pain Scale (.151), GCPS Pain Scale (.179), GCPS Disability Scale (.031), Heat Pain Threshold (.406), Heat Pain Tolerance (.483), Mean Pulse Pain (.444), -0.2 Power Temporal Summation Heat Pain (.110), Natural Log Pressure Pain Threshold (.110), Natural Log Temporal Summation Punctate Pain (.333).

## Table 3
**Model-estimated (least squares) means and standard errors by racial groups**

| Variable | Non-Hispanic White | Asian American |
|---|---|---|
| WOMAC Pain | 1.67 (0.08) * | 2.17 (0.09) * |
| | 5.12 (0.54) ** | 8.34 (0.58) ** |
| GCPS Pain | | |
| Female | 37.21 (3.08) | 59.06 (3.16) |
| Male | 40.89 (3.88) | 46.42 (4.19) |
| GCPS Disability | 29.73 (3.38) | 41.92 (3.63) |
| Heat Pain Threshold | 43.38 (0.39) | 38.29 (0.52) |
| Heat Pain Tolerance | | |
| Forearm | 47.63 (0.36) | 41.65 (0.63) |
| Knee | 47.66 (0.33) | 42.67 (0.54) |
| Mean Pulse Pain | | |
| Forearm | 29.46 (2.98) | 61.98 (3.54) |
| Knee | 23.42 (2.79) | 64.02 (3.80) |
| Temporal Summation Heat Pain | | |
| 44 Celsius | 0.44 (0.11) † | 1.13 (0.16) † |
| | 2.70 (1.18) ** | 14.19 (2.59) ** |
| 46 Celsius | 0.74 (0.11) † | 1.28 (0.16) † |
| | 3.99 (1.56) ** | 14.79 (2.33) ** |
| 48 Celsius | 1.29 (0.11) † | 1.53 (0.16) † |
| | 8.77 (1.83) ** | 17.49 (2.59) ** |
| Pressure Pain Threshold | | |
| Forearm | 5.59 (0.09) * | 4.86 (0.10) * |
| | 303.48 (23.44) ** | 158.14 (16.43) ** |
| Medial knee | 5.89 (0.08) * | 5.19 (0.09) * |
| | 389.92 (23.14) ** | 206.71 (17.43) ** |
| Lateral knee | 5.83 (0.09) * | 5.30 (0.09) * |
| | 386.11 (24.85) ** | 229.36 (17.71) ** |
| Quadriceps | 6.13 (0.10) * | 5.19 (0.10) * |
| | 507.87 (35.62) ** | 217.16 (20.57) ** |
| Trapezius | 5.67 (0.09) * | 5.05 (0.10) * |
| | 322.94 (24.74) ** | 188.61 (18.24) ** |
| Temporal Summation Punctate Pain | 2.53 (0.08) * | 3.21 (0.08) * |
| | 7.67 (1.49) ** | 20.61 (2.11) ** |

Note.

* Natural log transformed scale.

** Non-transformed raw scale.

† -0.2 power transformed scale. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index. GCPS: Graded Chronic Pain Scale.