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Multiple-levels of suffering: Discrimination in health care settings is associated with enhanced laboratory pain sensitivity in sickle cell disease

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Abstract

Objective—People living with sickle cell disease (SCD) experience severe episodic and chronic pain and frequently report poor interpersonal treatment within health care settings. In this particularly relevant context, we examined the relationship between perceived discrimination and both clinical and laboratory pain.

Methods—Seventy-one patients with SCD provided self-reports of experiences with discrimination in health care settings and clinical pain severity, and completed a psychophysical pain testing battery in the laboratory.

Results—Discrimination in health care settings was correlated with greater clinical pain severity and enhanced sensitivity to multiple laboratory-induced pain measures, as well as stress, depression, and sleep. After controlling for relevant covariates, discrimination remained a significant predictor of mechanical temporal summation (MTS, a marker of central pain facilitation), but not clinical pain severity or suprathreshold heat pain response. Furthermore, a

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significant interaction between experience with discrimination and clinical pain severity was associated with MTS; increased experience with discrimination was associated with an increased correlation between clinical pain severity and temporal summation of pain.

Discussion—Perceived discrimination within health care settings was associated with pain facilitation. These findings suggest that discrimination may be related to increased central sensitization among SCD patients, and more broadly that health care social environments may interact with pain pathophysiology.

Keywords

clinical pain; quantitative sensory testing; patient-provider interaction; temporal summation; racial discrimination

Introduction

People living with sickle cell disease (SCD) experience severe episodic and chronic pain[1] and psychosocial sequelae associated with the disease.[2] Although they report high levels of daily pain that is frequently managed at home,[1] the experience of severe pain often leads to frequent engagement with the health care system.[3] Unfortunately, despite the availability of SCD pain treatment guidelines,[4] SCD patients report under-treatment of pain and poor interpersonal treatment in health care settings.[5–6]

SCD patients are often perceived as “difficult patients,”[6] and may be disproportionately exposed to biased and discriminatory treatment in health care settings due to a number of historical, cultural, and social factors. While SCD affects people from various ethnic backgrounds worldwide, in the United States it is largely associated with and perceived to only affect African Americans.[7] A recent survey found that many patients and providers at a SCD clinic believed that patient race affected treatment and pain management.[8] Moreover, because of severe and undermanaged pain, SCD patients are often perceived as drug-seekers or addicts[9] and might display behaviors in interactions with providers that are misperceived as being characteristic of substance abuse.[10]

Discrimination in health care settings may directly affect multiple health outcomes, including pain management, clinical pain, and pain sensitivity. Several researchers have suggested that discrimination plays a role in the inadequate treatment and pain management in SCD.[7, 11, 12] Negative interpersonal experiences contribute to frequent at-home management of vaso-occlusive crisis[1] and self-discharge from hospitals,[13] suggesting that interpersonal treatment factors, such as discrimination, impede adequate pain management for SCD patients.[14] A few studies have assessed the relationship between discrimination and clinical pain in SCD[15, 16] and reveal that SCD patients experience more race-based discrimination in health care settings than African Americans in general, and additionally experience disease-based discrimination that is associated with increased clinical pain.[16] If also associated with increased pain sensitivity, discrimination within health care settings may increase the burden on SCD patients several-fold.

Here, for the first time, we examine the relationship between perceived racial discrimination within health care settings and both clinical pain and laboratory pain sensitivity among adults with SCD. While clinical pain and laboratory pain sensitivity are related,[17] examining both may provide valuable insight in characterizing the pathophysiology and sensory dimensions of pain due to SCD. Specifically, we include tests of central sensitization (e.g. temporal summation) as this has been proposed as a mechanism and marker of pain chronification.[18] Central sensitization is an amplification of central nociceptive processes that leads to altered pain responses following repeated exposure to pain.[19] The majority of investigations on SCD pain have focused on the acute pain of vaso-occlusive crisis.[20] However, adults with SCD also typically experience chronic pain that occurs every day in about one-third of patients[1] and is independent of vaso-occlusion.[21] The problem of chronic pain in patients with SCD remains largely unexplored and undertreated.[20] To date, despite suggestion that central sensitization may lead to chronic pain and hyperalgesia among SCD patients,[4] no clinical studies have tested pain sensitization among adults with SCD.[22] We hypothesized that discrimination would be associated with increased clinical pain and facilitation of laboratory pain.

Materials and Methods

Participants

SCD patients were recruited for participation from the Sickle Cell Center for Adults at Johns Hopkins and through posted advertisements. Seventy-one volunteers (68 African American/Black, 3 Multiracial) with SCD participated in this study (see Table 1 for demographic data), which is part of an ongoing larger study on pain in SCD (n = 82). Major inclusion criteria included age ≥18 years, formal diagnosis of SCD (hemoglobinopathy genotype (Hb SS, Hb SC, Hb S/β-thalassemia)), and on a stable dose (if any) of NSAIDs, acetaminophen, or opioids one month prior to pain testing. Exclusion criteria included current alcohol or substance abuse/dependence; delirium, dementia, or cognitive impairment; and unstable psychiatric illness. All participants who provided responses to the discrimination subscale of the Interpersonal Processes of Care Survey (IPC-18)[23] were included in the present analyses.

Procedure

Prior to study participation, participants completed an initial phone screen in which they provided a brief medical history to ensure study criteria were met. All study-related procedures were approved by the Johns Hopkins University School of Medicine Institutional Review Board, and informed written consent was obtained from each participant. The in-person visit was scheduled on days when patients were experiencing typical SCD pain at the level of 5 or lower on a 0–10 pain rating scale and had not experienced a vaso-occlusive crisis in at least the previous 3 weeks. After the consent process, participants completed the surveys described below, and a psychophysical pain testing battery lasting approximately one hour. Participants also completed questions related to their situational responses to and evaluations of the pain. Participants were allowed to stop or refuse any procedure at any time.

Survey Measures

Participants completed a number of surveys prior to commencing pain testing, including a demographic and health history questionnaire, and the following previously validated surveys.

Discrimination—The discrimination subscale of the short form of the Interpersonal Processes of Care Survey (IPC-18)[23] was used to assess experience with discrimination in health care settings that is attributed to race or ethnicity. The discrimination subscale consists of two questions (*How often did doctors pay less attention to you because of your race or ethnicity? How often did you feel discriminated against by doctors because of your race or ethnicity?*) Participants answered each item using a 5-point scale (1 – *Never* to 5 – *Always*). For conceptual clarity, the scale was transformed to a 0–4 scale so that a response of *Never* corresponds to a score of 0. This discrimination subscale has been validated across diverse racial groups within clinical populations[23] and has previously been shown to be associated with decreased quality of life[24] and physiological assessments of disease severity[25] in different patient populations.

Clinical Pain—Self-reported clinical pain severity was assessed as the average of patients' current pain as well as their worst, least, and average pain over the previous week using an 11-point scale (0 – *No Pain* to 10 – *Pain as bad as it could be*). The ten items from the extended[26, 27] Brief Pain Inventory (BPI)[28] pain interference subscale assessed functional interference caused by pain during the previous week in the areas of mood, sleep, relationships with others, and various daily activities and were scored on an 11-point scale (0 – *Does not interfere* to 10 – *Completely interferes*).

Potential Covariates—To more fully characterize the independent relationship between discrimination and pain, we include several well-validated measures of various behavioral and psychological constructs known to also be associated with pain sensitivity.

Stress: Baseline stress level (*How much stress do you feel right now?*) was assessed using an 11-point scale (0 - *none* to 10 - *extreme*), with higher scores representing a higher degree of stress. Stress is associated with altered pain sensitivity[29] as well as discrimination[30], and was collected as a potential mediator of the relationship between discrimination and pain.

Depression: The Center for Epidemiological Studies Depression Scale (CES-D)[31] measures depressive symptomatology. In the current study, we asked participants to respond based on frequency of feelings/experiences during the last week on a 5-point scale (0 - *rarely/less than one day* to 4 - *most of the time/5–7 days*). Depression is consistently associated with clinical pain[32] and modulates sensitivity to laboratory pain.[33]

Catastrophizing: The Pain Catastrophizing Scale (PCS)[34] assesses exaggerated negative cognitive and affective response to pain, and is a powerful predictor of clinical pain across chronic pain populations.[35] This standard version of the scale assesses trait-like responses to pain in general and consists of 13 items rated on a 5-point scale (0 - *not at all* to 4 - *all the time*) with higher scores indicating greater pain catastrophizing. Situational catastrophizing

(sitCAT)[36], which is predictive of laboratory pain,[37] was assessed during the pain testing session, and was scored on the same 5-point scale as the PCS.

Pain Anxiety: The short form of the Pain Anxiety Symptoms Scale (PASS)[38] measures fear and anxiety responses to pain in general, is related to enhanced clinical pain, and consists of 20 items rated on a 6-point scale (0 - *never* to 5 - *always*) with higher scores indicating greater pain anxiety.

Poor Sleep Quality: The Pittsburgh Sleep Quality Index (PSQI)[39] assesses subjective sleep quality and continuity for the previous month. The global score takes into account sleep quality, latency, duration, efficiency, disturbance, medication, and daytime dysfunction. Possible scores range from 0 to 21, with higher scores indicating poorer sleep quality. Poor sleep quality was examined as a potential covariate due to its relationship with clinical pain[40] and discrimination.[41,42]

Ethnic Identification: The Multigroup Ethnic Identity Measure (MEIM)[43] measures degree of identification with one's own ethnic group using a 4-point scale (1 - *strongly disagree* to 4 - *strongly agree*), with higher scores corresponding to greater identification. Ethnic identification has previously been shown to be associated laboratory pain among healthy African Americans[43] and was therefore included as a potential mediator of the relationship between discrimination and pain.

Psychophysical Pain Testing

Participants completed at least one trial of each of the below described procedures. All available data are included in subsequent analyses.

Pain Ratings—A numerical rating scale ranging from 0 (no pain) to 100 (worst pain imaginable) was used for each of the pain testing procedures.

Thermal Stimuli—All contact heat stimuli were delivered using a Contact Heat-Evoked Potential Stimulator (CHEPS, Medoc Ltd., Ramat Yishai, Israel) system, a peltier-element-based stimulator with a 9 cm^2 rapidly heating/cooling probe.

Heat Pain Threshold/Tolerance—Heat pain threshold (HPTh) and tolerance (HPTo) were calculated as the average of two corresponding trials administered to participants' dominant ventral forearm using an ascending method of limits paradigm. On each trial, the contact thermode gradually increased in temperature, from a baseline of 30°C at a 0.5°C/second rate of increase, until the subject indicated via button press the stimulus first felt painful (HPTh) or when the stimulus became intolerable (HPTo). Between trials, the thermode was moved up the arm slightly to avoid overlapping stimulation sites.

Pressure Pain Threshold—An electronic algometer (Somedic, Sollentuna, Sweden) was used to assess pressure pain threshold using a 1 cm^2 probe covered with a 1 mm polypropylene material.[45] Pressure was applied to the muscle belly and increased steadily at a rate of 30 kPa/sec until the subject verbally indicated the pressure first felt painful (PPTh). Pressure pain thresholds were assessed twice at each of four body sites, bilaterally (trapezius

muscle, interphalangeal joint of the thumb, the proximal third of the brachioradialis muscle (forearm), and middle of the quadriceps insertion point), for a total of 16 PPTh assessments. A minimum one minute interval was maintained between applications at the same site. The final PPTh was calculated as the average across all sites and repetitions[a] (Table 1).

Conditioned Pain Modulation—Conditioned pain modulation (CPM) was assessed using pressure applied to the trapezius as the test stimulus, and hot water bath as the conditioning stimulus. First, PPTh was again assessed (separate from PPTh above) twice at the non-dominant trapezius. The dominant hand was then submerged in a hot water bath for 20 seconds, at which time PPTh was reassessed. If participants removed their hands before 20 seconds, PPTh was assessed immediately upon withdrawal. The hot water temperature was determined early in the pain testing session as the temperature at which patients rate their pain as a 60–70 out of 100 after 20 seconds of hand submersion. Hot water temperature was first tested at 40°C. Subsequent tests with increasing temperatures were conducted as needed until the target pain intensity was achieved. CPM was calculated as the difference between the PPThs during and before water submersion. This procedure was repeated a second time, and final scores reflect an average of the difference score obtained during each trial. Participants revealed a significant increase in PPTh in the presence of the conditioning stimulus ($M_{baseline}$ = 228.81 kPa, $M_{hot\ water}$ = 302.23 kPa, $t(69) = 9.29$, $p<.001$), indicating our procedure successfully elicited CPM.

Suprathreshold Heat Pain Response, Thermal Temporal Summation, Pain Unpleasantness, and After Sensations—Ten repetitive thermal stimuli were applied rapidly, to participants' dominant ventral forearm, in a series of identical pulses. A pain rating was obtained for each pulse. The thermode remained in a fixed position during administration of each sequence of 10 heat pulses (0.5 sec each, with a 2.5-sec inter-pulse interval). A practice trial with pulses at participants' warmth detection threshold was conducted to familiarize participants with the procedure. Experimental trials were conducted at tailored temperatures (HPTh-2°C, HPTh, HPTh+2°C), and at a standard temperature of 45°C. The thermode was moved slightly between trials to avoid overlapping stimulation sites.

The pain rating on the fifth pulse was used as a measure of suprathreshold heat pain response (SHPR) as has been used by others.[46] Participants rated the fifth pulse in each series as painful (suprathreshold) across temperatures (M_{HPTh-2}: 17.12, SE_{HPTh-2}: 2.50; M_{HPTh}: 24.88, SE_{HPTh}: 2.86; M_{HPTh+2}: 34.52, SE_{HPTh+2}: 3.33; M_{45}: 46.82, SE_{45}: 3.51), and the pattern of response was similar across temperatures. An average of the SHPR at the four[b] experimental temperatures was used for all analyses.

Thermal temporal summation (TTS) was calculated as the difference between maximum pain rating within each trial and the pain rating on the first pulse. An average TTS across the four experimental temperatures was used for all analyses.

[a]The pattern of response was similar across all sites and repetitions. Results remain similar when site-specific PPThs were used in primary analyses.
[b]Results remain similar when the response at HPTh-2 was excluded from the average composite score.

Pain unpleasantness (0 to 100) was assessed immediately following each trial. Pain unpleasantness was averaged across the four experimental temperatures.

Residual pain was queried following each trial and these "after sensations" were rated 15 seconds after the final "pulse" of each trial. After sensations were similarly averaged across the four temperatures and used in all analyses.

Mechanical Temporal Summation—Mechanical temporal summation (MTS) was calculated as the difference between pain ratings in response to a single punctuate stimulus compared to a sequence of ten identical punctuate stimuli. Weighted pinprick stimulators with a flat contact area of 0.2 mm diameter were used to deliver stimuli at a 1/sec rate to the middle phalange of the middle finger. A practice trial was conducted with a stimulator that produced 32 mN force. Experimental trials were conducted at 128 mN and 256 mN. An average MTS at the two experimental weights was used for all analyses.

Data Analysis

The goal of the first level of analysis was to assess the relationship between discrimination and clinical and laboratory pain as well as related behavioral, psychological, and physiological variables. Descriptive statistics were evaluated and guided first level inferential statistical analyses.

The goal of the second level of analysis was to determine the statistical effect of current clinical pain and discrimination on laboratory pain sensitivity. Discrimination was included as a continuous, not dichotomous, variable in all multivariate models. Second level analyses were not conducted on factors that were not significantly correlated with individual differences on the discrimination scale. We conducted hierarchical multiple regression to determine the relationship between discrimination and pain after controlling for demographic data and correlated covariates (constructs that were correlated with dependent variables of interest). When covariates were highly correlated and overlapping with each other (i.e., depression and anxiety, dispositional and situational catastrophizing), we included the variable that was more strongly correlated with the dependent variable in the models. When clinical pain was correlated with laboratory pain dependent variables, potential clinical pain x discrimination interactions were also examined. Finally, we probed significant interactions using moderation models. The Johnson-Neyman technique was used to identify the region of significance of the moderator.[47] All data analyses were conducted using SPSS (version 21, Armonk, NY: IBM Corp.) and moderation was tested using Hayes' PROCESS macro[47] implemented in SPSS.

Missing Data—Participants were not excluded due to partially missing data, and the majority of participants (N=65; 91.5%) completed all procedures. Though some participants did not complete every trial of each psychophysical pain testing procedure due to voluntary discontinuance or rating the maximum (100) before the completion of a procedure, average ratings are available for all participants on each procedure with the exception of CPM (missing N=1) and SPTH/TTS (missing N=2). All participants (N=71) completed the discrimination, clinical pain, stress, pain catastrophizing, pain anxiety, and sleep surveys;

however, a few participants chose not to respond to the depression (missing N=3) and ethnic identification (missing N=1) surveys.

Results

Discrimination in Health Care Settings

Participants responded similarly to both discrimination items; mean scores = 0.51(doctors paid less attention) and 0.42 (patients felt discriminated). Thirty-eight percent (n=27) of participants reported some experience with discrimination in health care settings. Most of these (n=21) reported that doctors paid less attention to them because of their race (range of reports from “rarely” to “always”) and (n=22) that they *felt* discriminated against by doctors because of their race or ethnicity (range of reports from “rarely” to “sometimes”). Items were correlated ($R = .58$, $p < .001$), and the subscale was reliable (Spearman-Brown Coefficient = .73) within our sample.

Descriptive statistics revealed two distinguishable groups[c] of patients – those who reported experiences of discrimination in health care settings and those who reported no experience with discrimination in health care settings. In order to evaluate the differences in pain sensitivity between patients reporting no discrimination and those reporting any discrimination, these groups were compared on all study variables of interest using independent t-tests (Table 1). There were no group differences in age, sex, or education level, pain catastrophizing, pain anxiety, ethnic identification, nor in heat pain threshold (HPTh), heat pain tolerance (HPTo), pressure pain threshold (PPTh), conditioned pain modulation (CPM) or thermal temporal summation (TTS). Patients who reported experience with discrimination in health care settings reported greater clinical pain severity and interference (Table 1); these patients also demonstrated greater suprathreshold pain ratings (SHPR), mechanical temporal summation (MTS), and after sensations, all indicators of central sensitization.[18] Patients who experienced discrimination within health care settings also reported more pain unpleasantness, greater stress, marginally more depressive symptomatology, and worse sleep quality.

In order to identify potential covariates for multivariate analyses, correlations were examined between potential covariates and pain measures (both clinical pain and markers of central sensitization) that showed a difference across discrimination groups. Across all participants, individual differences in experiences of discrimination in health care settings were associated with greater SHPR, MTS, and clinical pain severity and interference but not with pain unpleasantness or after sensations (Table 2). Pain after sensations and MTS correlated with clinical pain severity. As expected, situational catastrophizing correlated with all psychophysical pain measures, and clinical pain severity was related to stress, depression, catastrophizing, pain anxiety, and poor sleep quality.

[c]The split was identical whether a mean split, median split, or all-or-nothing split was chosen.

Discrimination, Clinical Pain, and Laboratory Pain Sensitivity

Regression models were used to probe the relationships between discrimination and clinical pain, and discrimination and laboratory pain sensitivity, focusing on the two markers of central sensitization - SHPR and MTS - that were consistently associated with individual differences in experiences of discrimination in health care settings.

Clinical Pain—Hierarchical multiple regression revealed that discrimination did not predict clinical pain severity over and above other factors. Age, depression and poor sleep quality were significant predictors of clinical pain severity (Table 3).

Laboratory Pain

Suprathreshold Heat Pain Response: Discrimination did not remain a significant predictor of SHPR over and above situational catastrophizing and poor sleep quality (Table 4).

Mechanical Temporal Summation: Clinical pain severity was no longer a significant predictor of MTS after controlling for situational catastrophizing. Discrimination as well as the interaction between discrimination and clinical pain severity significantly predicted MTS, independently accounting for 10% and 9% of the variance in MTS respectively, even after controlling for demographic variables and correlated covariates (Table 4).

Moderation analysis conducted to further probe the interaction between clinical pain severity and discrimination on MTS indicated that for patients experiencing greater racial discrimination, greater clinical pain severity was associated with significantly greater MTS. There was no significant relationship between clinical and laboratory pain among those with no experience with discrimination (Table 5). Further decomposition of the interaction using the Johnson-Neyman technique[47] revealed that clinical pain severity was positively associated with MTS at discrimination frequencies above 0.65 (Figure 1). A discrimination score of 0.50 corresponds to any report of discrimination (a response above “never” on one of the items). Therefore, the moderating effect of discrimination on the relationship between clinical pain severity and MTS is significant for most participants reporting any discrimination at all.[d]

Discussion

SCD patients suffer both severe and poorly managed pain as well as the social harm of discriminatory interpersonal treatment. Compared to patients who report no discrimination within health care settings, patients who experienced such discrimination show a profile of increased pain sensitivity that includes greater clinical pain severity, heightened sensitivity to suprathreshold thermal stimuli, increased after sensations, greater mechanical temporal summation of pain, and greater pain unpleasantness. While discrimination did not remain significantly associated with clinical pain severity when other pain-related covariates were included in the multivariate models, discrimination was independently associated with acute pain processing in the laboratory, particularly measures of pain facilitation. Health care

[d]The results of the moderation model remained significant, and the pattern unchanged, when discrimination was included as dichotomous (discrimination vs. no discrimination), rather than continuous, variable.

discrimination was also associated with a variety of symptoms of distress, including greater depressive symptomatology, poorer sleep, and higher stress ratings. Overall, these findings are consistent with the literature demonstrating positive associations between life-time discrimination and pain,[48–50] stress,[30] depression,[51] and poor sleep.[41, 42] Importantly, discrimination among African American adults is associated with delays in seeking medical care and lower adherence to doctor recommendations.[52] Our results extend these findings by demonstrating that discrimination within health care environments is independently correlated with increased pain and poorer psychological outcomes among SCD patients.

Health care discrimination showed a fairly large association with mechanical temporal summation, accounting for an additional 10% of variance even after controlling for numerous factors known to be associated with pain. These results indicate that measures of discrimination should be included in future studies of pain sensitivity in SCD and more broadly suggest that interpersonal experiences may influence physiological processes underlying pain processing and central sensitization. We also find a significant interaction between discrimination and clinical pain severity that is independently associated with MTS. Patients who report any discrimination in health care show increased clinical pain severity-related mechanical sensitization, a relationship absent in patients who do not report discrimination. The mechanisms underlying this interaction are unclear; however, one plausible explanation is that discrimination alters the physiological environment such that heightened clinical pain facilitates central sensitization. This may occur through neuroendocrine responses to discrimination[53,54] or other mechanisms related to social exclusion[55] (discussed in more detail below). Furthermore, greater central sensitization may have a bi-directional effect with clinical pain, maintaining and even worsening pain over time.

Discrimination is one type of social stressor and future studies should directly compare discrimination with other stressors, including other social stressors. Current evidence suggests that discrimination may have unique effects on pain processing, over and above that of stress broadly defined. In the experimental social laboratory, direct comparisons of performance stress and discrimination suggest that discrimination produces significantly more risky health behaviors than performance stress or control conditions.[56] Prior research has not found an effect of cognitive stress inductions on temporal summation of pain among healthy or chronic pain populations.[57,58] Social exclusion in the lab increases the unpleasantness of acute heat pain[55] and social support decreases pain intensity ratings in response to cold pressor[59] and heat[60] pain among healthy volunteers. Taken together, our results and the experimental literature among healthy volunteers suggest that negative social experience contributes to enhanced pain sensitization. The ways in which experiences with discrimination are similar, and different from that of other stressors, and the influence of discrimination on the neuroendocrine system, have yet to be fully explored (see initial examinations of the relationship between discrimination and functioning of the HPA system[53,54]). Future research should examine whether intervening at the level of interpersonal interactions may potentially lessen some of the deleterious effects of discrimination.

This is the first study to find that the social experience of discrimination is associated with pain facilitation processes. Prior findings have demonstrated that cognitive-affective psychological processes such as pain catastrophizing[61,62] and fear of movement[63] are associated with temporal summation and other indices of central sensitization within other chronic pain conditions. The current findings extend this evidence and suggest that social factors may also contribute to pain facilitation and perhaps central sensitization above the influence of clinical pain on central sensitization. Importantly, this relationship is also independent of the influence of previously identified cognitive-affective processes such as situational catastrophizing. Thus, it will be important in future research to investigate how and when these social experiences translate into increased central sensitivity to pain. Our pattern of findings do not suggest an overall heightened sensitivity resulting from the experience of health care discrimination, since increased discrimination did not correlate with all pain outcomes (e.g., heat or pressure pain thresholds). Furthermore, this demonstration of the relationship between discrimination and pain sensitization has important broader implications for the study of pain disparities. Numerous studies have demonstrated heightened pain sensitization among African Americans in the laboratory relative to white Americans, but the mechanisms underlying this disparity are not understood.[64, 65] Our results suggest social mechanisms, such as the influence of discrimination on pain sensitization, should be investigated in future studies of pain disparities.

While we propose that the social experience of discrimination modulates pain sensitivity and facilitates central sensitization, we have considered a number of alternative hypotheses that warrant further exploration in future research. One plausible alternative explanation is that discrimination decreases health care utilization, treatment seeking, and/or adherence to medical advice which all may increase pain. Among people with SCD, who are already resistant to engaging the medical system and prefer to manage their pain at home when possible,[1] experiences of discrimination may provide further discouragement from seeking medical care. Being distrusted by hospital staff, and having difficulty convincing providers of one's pain has also been associated with self-discharge from the hospital, an indicator of dissatisfaction with pain management.[13] However, the relationship between discrimination and health care utilization depends on SCD patient optimism,[15] suggesting this relationship may not be simply a function of discrimination-evoked reduction in health care utilization.

Another plausible explanation of our findings is that the patients who experience heightened sensitization may also be engaging the health care system more, and therefore may have more opportunity to experience discrimination within these settings. More health care visits might increase exposure to specific settings and/or providers who may be biased or have negative attitudes about SCD patients. Controlled laboratory studies have demonstrated that perceiver bias and patient factors such as race and medication status can alter pain perception, empathy, and treatment decisions.[66–68] Negative attitudes among providers toward SCD patients are a consistent and significant barrier to SCD treatment and pain management across studies.[14] Future studies should seek to examine the effect of discrimination, particularly within health care settings, on treatment seeking and health care utilization over time.

A final consideration is that some participants may have a response bias to report greater sensitivity to a wide variety of challenges and insults, including laboratory pain and discrimination. However, the lack of association between discrimination and some measures of pain sensitivity (e.g., heat pain threshold and tolerance and pressure pain threshold) does not suggest a consistent response bias. One might expect that if these results are due to response bias, controlling for other similar constructs, such catastrophizing, might nullify the relationship. Nonetheless, longitudinal studies that examine the impact of insults over time will likely provide additional insight into the progression and cause of this finding.

Limitations of the current study include our use of a single subscale to assess discrimination. Discrimination is a complex construct, and patients with SCD likely experience other forms of discrimination, including disease-based discrimination[16]. However, by examining discrimination within health care settings, a specifically relevant context to patients with a chronic and complicated illness, this study importantly advances current knowledge about the relationship between discrimination and health outcomes. The inclusion of multidimensional discrimination measures will enable future studies to directly compare the predictive value of the various dimensions of discrimination. Additionally, discrimination was not associated with static markers of sensitization (e.g. pain thresholds) or pain inhibition, and did not remain significantly associated with clinical pain severity, over and above highly correlated covariates, which suggests that discrimination may be most influential in impacting central pain sensitizing mechanisms. Future studies are needed to further parse these findings. Finally, our sample size may have limited our ability to detect smaller effects of discrimination on pain.

Given the prevalence and severity of both pain and discrimination experienced by people living with SCD, and the importance of avoiding additional pain and barriers to treatment embedded in patient care, we suggest future studies test whether interventions that reduce discrimination within health care settings also reduce clinical pain, and whether this reduction occurs independently or through mediating effects of other variables, such as depression. Prior studies have demonstrated the effectiveness of brief interventions on provider attitudes toward SCD patients,[69] but whether these interventions also reduce perceptions of discrimination on the part of patients receiving care from these providers following such interventions needs to be established. Other research shows brief training in cognitive coping skills reduces laboratory-induced pain, increases coping attempts and decreases negative thinking in SCD patients.[70] Future studies should examine the potential effects of such interventions on the relationship between perceived discrimination and pain sensitivity and severity.

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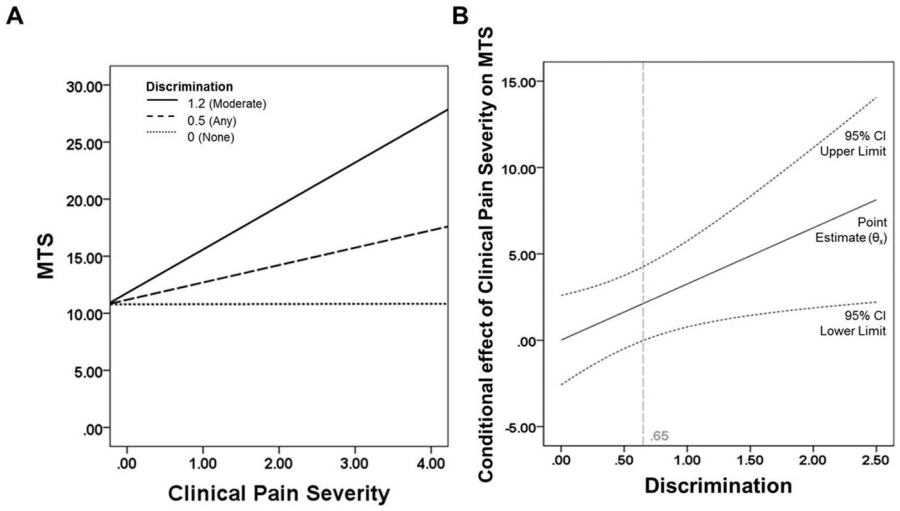

Figure 1. Racial discrimination moderates the relationship between clinical pain severity and mechanical temporal summation

A) Regression lines for the association between clinical pain severity and mechanical temporal summation as moderated by experience with discrimination. For the purpose of demonstration, values are not adjusted for the covariate situational catastrophizing. B) Conditional effect of clinical pain severity on TS ($\theta_{X\rightarrow Y}$) as a function of perceived discrimination in health care settings.

Table 1

Descriptive statistics by level of experience with discrimination in health care settings

	Discrimination	No Discrimination
N	27	44
Sex		
Female	22 (81%)	29 (66%)
Age		
Mean (SD)	40.73(10.79)	37.25(12.53)
Range	26–61	19–64
Highest Education		
High school or less	4 (15%)	8 (18%)
Some college	12 (44%)	20 (46%)
Bachelor's degree or more	11 (41%)	16 (36%)
Discrimination	1.22(.12) *	0.00(.00)
Laboratory Pain		
HPTh	40.69(.53)	40.97(.43)
HPTo	43.55(.34)	44.25(.32)
PPTh	332.19(24.34)	333.85(18.15)
CPM	76.72(12.24)	71.45(10.42)
SHPR	40.64(4.71) *	25.61(2.42)
TTS	5.74 (1.68)	3.67 (.79)
Unpleasantness	35.17(4.98) *	22.21(3.65)
After Sensations	15.74(3.53) *	7.24(1.75)
MTS	21.24(4.00) *	11.07(1.81)
Clinical Pain		
Severity	2.50(.32) *	1.49(.25)
Interference	3.74(.48) *	1.84(.35)
Stress	1.63(.35) *	.70(.19)
Depression	17.99(2.20) †	13.42(1.61)
Catastrophizing		
Dispositional	13.59(1.84)	12.63(1.50)
Situational	1.12(.18)	.92(.12)
Pain Anxiety	49.13(2.76)	46.79(2.83)
Poor Sleep Quality	9.30(.81) *	6.88(.56)

	Discrimination	No Discrimination
Ethnic Identification	3.00(.10)	3.02(.08)

$^{*}p \leq .05$;

$^{\dagger}p \leq .10$;

Standard error in parentheses unless otherwise noted.

HPTh=Heat pain threshold, HPTo=Heat pain tolerance, PPTh=Pressure pain threshold, CPM = Conditioned Pain Modulation, SHPR=Suprathreshold heat pain response, TTS = Thermal temporal summation, MTS=Mechanical temporal summation.

Table 2

Correlations between laboratory pain sensitivity, clinical pain, and related covariates

		Laboratory Pain Sensitivity				Clinical Pain	
		Suprathreshold Heat-Pain Response	Pain Unpleasantness	Pain After Sensations	Mechanical Temporal Summation	Clinical Pain Severity	Clinical Pain Interference
Related Covariates	Clinical Pain Severity	.10	.10	.45*	.26*	-	
	Clinical Pain Interference	.01	.02	.32*	.17	.77*	-
	Discrimination	.26*	.15	.18	.36*	.23*	.40*
	Stress	−.13	−.09	−.13	−.01	.07	.22†
	Depression	.06	.13	.25*	.12	.43*	.47*
	Dispositional Catastrophizing	−.12	−.01	.02	−.0	.30*	.31*
	Situational Catastrophizing	.28*	.58*	.57*	.41*	.29*	.23*
	Pain Anxiety	.18	.24*	.20†	.07	.25*	.35*
	Poor Sleep Quality	.33*	.13	.26*	.20†	.51*	.43*
	Ethnic Identification	.04	−.02	0	−.11	.09	.10

*$p \leq .05$;

†$p \leq .10$.

Correlations within full sample (N = 71).

Table 3

Results of regression model predicting clinical pain severity

Variable	B	SE	*β*	*t*	R^2	ΔR^2
Step 1: Demographics					.09	.09
Sex	.15	.46	.04	.32		
Age	.04	.02	.27*	2.18		
Education	−.19	.16	−.14	−1.19		
Step 2: Depression	.07	.02	.44*	3.84	.26*	.18*
Step 3: Dispositional Catastrophizing	.02	.02	.13	1.01	.27*	.01
Step 4: Poor Sleep Quality	.18	.05	.41*	3.73	.41*	.14*
Step 5: Discrimination	−.15	.29	−.06	−.50	.41*	.003

* p≤.05.

Statistics are presented in sequential fashion, such that the first step includes coefficients when only step 1 is executed. Subsequent steps show adjusted coefficients controlling for the predictors entered in previous steps. B, unstandardized coefficient; *β*, standardized beta coefficient

Table 4

Results of regression models predicting laboratory pain sensitivity

Suprathreshold heat pain	**B**	**SE**	***β***	***t***	R^2	ΔR^2
Step 1: Demographics					.01	.01
Sex	3.28	5.44	.08	.60		
Age	.01	.21	.01	.07		
Education	−1.37	1.89	−.09	−.72		
Step 2: Situational Catastrophizing	5.99	3.08	.25†	1.94	.07	.06†
Step 3: Poor Sleep Quality	1.45	.58	.30*	2.49	.15†	.09*
Step 4: Discrimination	3.68	3.73	.13	.99	.17†	.01
Mechanical temporal summation						
Step 1: Demographics					.06	.06
Sex	5.55	4.41	.15	1.26		
Age	.25	.17	.18	1.51		
Education	−1.69	1.52	−.13	−1.11		
Step 2: Situational Catastrophizing	7.74	2.33	.39*	3.32	.20*	.14*
Step 3: Clinical Pain Severity	1.19	1.15	.12	1.04	.21*	.01
Step 4: Discrimination	7.78	2.62	.33*	2.97	.31*	.10*
Step 5: Clinical Pain Severity x Discrimination	4.00	1.35	.85*	2.98	.39*	.09*

*p≤.05;

†p≤.10.

Statistics are presented in sequential fashion, such that the first step includes coefficients when only step 1 is executed. Subsequent steps show adjusted coefficients controlling for the predictors entered previous steps. B, unstandardized coefficient; *β*, standardized beta coefficient

Table 5

Moderation analysis: The effect of discrimination on the relationship between clinical pain and mechanical temporal summation

Moderator value		*b*	SE_b	*t*	*p*	95% CI
Moderate discrimination (+ 1 SD)	1.17	3.81	1.39	2.75	.008	1.04, 6.58
Any discrimination (Mean[a])	0.46	1.52	1.06	1.43	.16	−.60, 3.64
No discrimination[b]	0	.01	1.30	.01	.99	−2.58, 2.60

The complete model was significant (R^2= .23), $F(3, 67) = 6.48$, $p < .001$. The change in R^2 as a result of the interaction was also significant (ΔR^2= .06), $F(1,67) = 5.32$, $p = .02$. *b*, unstandardized regression coefficient; CI, confidence interval.

[a]This is also equivalent to any discrimination (patient responded higher than “never” on one of the two discrimination items)

[b]The minimum was used instead of 1 SD below the mean because 1 SD below the mean is outside of the range of the data.