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Race and Histories of Mood Disorders Modulate Experimental Pain Tolerance in Women

Rebecca R. Klatzkin, B.S.[b], **Beth Mechlin, B.S.**[b], **Robertas Bunevicius, M.D., Ph.D.**[a], and **Susan S. Girdler, Ph.D.**[a,b,*]

a*Department of Psychiatry, University of North Carolina at Chapel Hill, CB #7175, Medical Research Bldg A, Chapel Hill, NC 27599-7175, USA*

b*Department of Psychology, University of North Carolina at Chapel Hill, CB #7175, Medical Research Bldg A, Chapel Hill, NC 27599-7175, USA*

Abstract

Thirty-two African American and 23 non-Hispanic White women were compared for experimental pain threshold and tolerance to thermal, ischemic, and cold pressor pain. Approximately half of each group had prior mood disorders (17 African Americans, 13 non-Hispanic Whites), though all were free of current mood disturbance. Women with prior mood disorders were less sensitive to ischemic pain than women with no prior mood disorders ($p<.05$), while African Americans were more sensitive to ischemic pain than non-Hispanic Whites, though only at pain tolerance ($p<.001$). For cold pressor pain, the effects of race were only seen in women with prior mood disorders, since African Americans with prior mood disorders were more sensitive than non-Hispanic Whites with prior mood disorders ($p<.05$). These results indicate that experimental pain sensitivity in women is influenced by both race and histories of mood disorders.

Perspective: We examined the association of race and histories of mood disorders with experimental pain sensitivity in an exclusively female sample. Our findings for racial differences in pain sensitivity may have implications for greater clinical pain in African American women. Persistent disturbance in pain modulatory mechanisms in women with a history of mood disorders may also have implications for the development of subsequent mood disturbances.

Keywords

Mood disorders; Race; Pain Tolerance

INTRODUCTION

It is well established that clinical pain and somatic symptoms are an associated feature of depression [16, 52]. Since laboratory based methods to assess pain sensitivity are predictive of clinical pain [24] investigations of experimental pain sensitivity in individuals with depression and other mood disorders have been undertaken in order to elucidate the depression-pain relationship [3, 4, 5, 19, 20, 30, 42].

*Corresponding Author: Susan S. Girdler, Ph.D., CB # 7175, Medical Research Bldg A, University of North Carolina at Chapel Hill, Chapel Hill, NC 27599-7175. Telephone: (919) 966-2544; FAX: (919) 966-0708; Email: susan_girdler@med.unc.edu

While results of these studies have been mixed [3, 4, 5, 19, 20, 30, 42], which may be due, in part, to the different pain modalities employed [3, 7, 28, 32], a systematic review and meta-analysis of studies comparing patients with current depression versus non-depressed controls for experimental pain perception concluded that experimental pain thresholds were higher (i.e. reduced sensitivity) in depressed individuals [19]. However, only 2 of the 6 studies included in the meta-analysis assessed pain tolerance, which may be especially relevant for mood disorders, since pain tolerance reflects the affective experience of pain, while pain threshold reflects the sensory experience [43].

What is relatively understudied to date is whether euthymic individuals with a history of mood disorders experience alterations in pain sensitivity. This may be especially relevant to women since they experience significantly more clinical pain [55] and are twice as likely to experience lifetime depression relative to men [58]. This is also of clinical relevance, given that a recent study in women with a history of depression showed that clinical pain (e.g. headaches, backaches) persists beyond the remission of a depressive episode [10].

The studies included in the meta-analysis on experimental pain sensitivity and current depression did not address racial differences in pain sensitivity. African Americans have more clinical pain [22, 34, 46] and experience more pain related disability [21] than primarily Caucasian samples. There is a growing and consistent literature that African Americans have lower experimental pain tolerance, though not pain thresholds, relative to primarily Caucasian samples [13, 23, 36, 50, 60]. Recent work in our laboratory indicates that hyperalgesia to experimental pain stimuli in African Americans may be a result of alterations in endogenous pain regulatory mechanisms, including systolic blood pressure, since African Americans failed to show the expected blood pressure-related hypoalgesia that was seen in the primarily Caucasian sample [36]. Moreover, it is important to emphasize that the studies that do exist on racial differences in pain sensitivity are based exclusively on men or on mixed gender samples with no gender-based analyses. Consequently, it is unknown whether these same racial differences in pain sensitivity would be evident in an exclusively female sample.

Although exceptions exist, there is also evidence that African Americans may have a higher prevalence of mood disorders, including depression [8, 56, 59]. For example, a recent study documented that even after controlling for demographic factors, histories of depression , and other predictors, African American women were more than twice as likely to experience postpartum depressive symptoms than non-Hispanic White women [27]. The greater rates of mental illness in African American populations have been attributed to greater psychosocial stress associated with poverty, crime, and racism [47, 48].

Consequently, the purpose of this study was to examine the association of race and a history of mood disorders with threshold and tolerance to experimental pain in women. Specifically, we hypothesized that African American women would show increased pain sensitivity compared to non-Hispanic White women, while women with histories of mood disturbance would show decreased pain sensitivity compared to women with no prior mood disorder. Moreover, since pain sensitivity in patients with depression is influenced by the pain modality [3], we employed a variety of noxious stimuli differing in the quality and sensation of the painful percept.

METHODS

Subjects

A total of 72 women were recruited via advertisements. A proportion of our advertisements specifically stated that African American women and women with histories of depression were needed for a research study. Approximately half of the subjects were African American (n=32),

whereas the other half (n=30) included 'Other' racial groups (77% Non-Hispanic White, 10% Hispanic, 7% Asian, 3% Native American, and 3% Multi-racial heritage). One purpose of the study was to examine the association of psychosocial stress measures with cardiovascular and neuroendocrine stress reactivity in a multi-racial sample (to be reported elsewhere). An additional purpose of the study, which comprises the focus of this report, was to examine racial differences in the association of prior mood disorders with experimental pain sensitivity. Given the evidence that Hispanic populations differ in clinical pain symptoms [26] and are more sensitive to experimental pain than non-Hispanic Whites [31], and since the numbers of Hispanics (n = 3), Native Americans (n = 1), Asians (n = 2), and multi-racial women (n=1) in our study did not allow for valid analyses, the present report compares African Americans with non-Hispanic Whites only. Therefore, 32 African American women and 23 non-Hispanic White women are included in the present report.

All subjects were in good health, reported regular menstrual cycles, and were free of any current psychiatric Axis I disorder, as determined by structured interview (see below). Based on self report, subjects were not pregnant, nursing, or taking any prescription medication, including oral contraceptives. The protocol was approved by the University of North Carolina at Chapel Hill Biomedical Institutional Review Board. Subjects provided written informed consent before participation and each received $100 compensation.

Procedures

During an initial screening session, blood pressure readings were obtained and a medical history questionnaire was administered. The Perceived Stress Scale [15](PSS;), the most widely used psychological instrument for measuring the degree to which situations in one's life are appraised as stressful, was also administered at this time, along with the Beck Depression Inventory [6] (BDI) and the Spielberger Trait Anxiety Scale [53] (STAI-Y2). The BDI is a self-rated scale to evaluate depressive symptoms (cognitive, behavioral, and somatic) [6], while the STAI-Y2 is a self-report assessment of long-term/chronic anxiety [53]. Following, the Mini International Neuropsychiatric Interview [49] (M.I.N.I.), which is based on DSM-IV criteria for Axis I disorders, was administered by a trained interviewer (RK or BM). The reliability and validity of the MINI have been assessed in studies of psychiatric subjects [49], showing high inter-rater and retest reliability along with good diagnostic concordance of the MINI against the Structured Clinical Interview for DSM-III (SCID-P) [54] diagnoses. When compared with both the SCID-P and the Composite International Diagnostic Interview [61] (CIDI), the sensitivity of the MINI for major depression is 96% and specificity is 88% [49].

All diagnoses were based on a consensus diagnostic session with a psychiatrist (RB). In the present study, we defined prior mood disorders to include the diagnoses of major depressive disorder (MDD), minor depressive disorder, or a bipolar mixed episode, since patients with bipolar disorder do not differ from patients with major depression in thermal pain sensory discrimination and response criteria [20]. No subject met criteria for current or past dysthymia. For prior mood disorders, 3 months in full remission was required before testing. For other Axis I disorders, 3 years in full remission was required for all subjects. Women classified as having no history of mood disorder met criteria for no lifetime major or minor depression, dysthymia, and bipolar disorder. Women with prior mood disorders did not differ from women with no prior mood disorders in proportions with history of anxiety disorders (4% vs. 2%), eating disorders (4% vs. 0%), or substance abuse disorders (4% vs. 3%).

Based on these criteria, 17 of the 32 (53%) African American women and 13 of the 23 (57%) non-Hispanic White women were classified with having a prior mood disorder. African Americans did not differ from non-Hispanic Whites in the proportion with prior MDD (34% vs. 39%), minor depression (9% vs. 13%) or bipolar mixed episode (9% vs. 4%). We operationally defined prior minor depression as having experienced 3 or 4 (as opposed to 5 or

more) of the criterion symptoms for MDD and requiring the inclusion of either anhedonia or down/depressed mood. African Americans also did not differ from non-Hispanic Whites in months since full remission of the mood disorder (11 vs. 18 months, respectively), or in proportion with histories of anxiety disorders (6% vs. 4%), bipolar depression (0% vs. 0%), bipolar mania (0% vs. 0%), eating disorders (6% vs. 0%), or substance abuse disorders (9% vs. 0%).

Experimental procedures

All testing was conducted in the follicular phase of the menstrual cycle based on self-report (days 2-12 of the menstrual cycle). The experiment was conducted by a non-Hispanic white female experimenter. Studies on the effects of experimenter race have yielded inconsistent results regarding racial differences in pain sensitivity [57, 65]. Subjects were asked to refrain from caffeine and all over-the-counter medications for 24 hours. After instrumentation for cardiovascular monitoring (results to be reported elsewhere), subjects were escorted to a sound-attenuated testing chamber and seated in a comfortable chair.

Pre-test rest

Immediately following cardiovascular instrumentation and administration of the Spielberger State Anxiety Scale [53], 5 minutes of quite rest ensued. Blood pressure and heart rates were taken at minutes 1, 3, and 5 and averaged to constitute baseline levels.

Pain Testing Procedures

Immediately following the pre-test rest, each subject was exposed to three pain procedures. One of three task orders (i.e., 1. tourniquet, thermal, cold; 2. thermal, cold, tourniquet; or 3. cold, tourniquet, thermal) was randomly assigned to each subject, ensuring, however, that the number of subjects in each race and mood group having each of the three orders was roughly equivalent. The dominant arm was used for all pain testing. Since only three subjects were left-handed (all non-Hispanic White subjects, and two with histories of mood disorders), the likelihood that cerebral lateralization associated with handedness impacted the findings is minimal. Pain intensity and unpleasantness ratings were obtained for each of the three pain tasks. Subjects were instructed that at the point of tolerance for each pain test, they would be asked to rate the intensity and unpleasantness of their pain using separate visual analogue scales (0 – 100). Thus, immediately prior to deflating the tourniquet cuff, immediately prior to removal of the hand from the ice bath, and immediately after the third thermal tolerance temperature was delivered, the experimenter held up one visual analogue scale for intensity rating, with the 100 cm line anchored by the words ‘Not at all Intense’ and ‘The Most Intense Pain Imaginable’. Next, the experimenter held up the scale for unpleasantness, with the 100 cm line anchored by the words ‘Not at all Unpleasant’ and ‘The Most Unpleasant Pain Imaginable’.

The Submaximal Effort Tourniquet Procedure—In this procedure [40], a tourniquet cuff was positioned on the subject's dominant arm and the arm placed to the side. Before inflating the tourniquet cuff to 200 mm Hg (Hokanson E20 Rapid Cuff Inflator), the subject's arm was raised for 30 seconds to promote venous drainage, and then the cuff was inflated, the experimenter's stopwatch started, and the arm returned to the side. To promote forearm ischemia, subjects engaged in 20 handgrip exercises at 30% of their maximum force with an intersqueeze interval of 2 seconds. Subjects were instructed to indicate when the sensations in their arm first became painful (pain threshold) and when they were no longer willing or able to tolerate the pain (pain tolerance). After the subjects indicated their pain tolerance, but before the cuff was deflated, subjects indicated their pain intensity and unpleasantness ratings. A

maximum time limit of 20 minutes was enforced, though subjects were not informed of this limit.

Hand Cold Pressor—The apparatus for the cold pressor consisted of a container filled with ice and water that was maintained at 4°C as recorded immediately before initiating the test. The use of a water circulator prevented the water from warming near the subject's hand. At the onset of the test, subjects were instructed to submerge their dominant hand to the marked line on their wrist and to remain still. Subjects were instructed to indicate to the experimenter when the sensations in their hand first became painful (pain threshold) and to also indicate when they were no longer willing or able to tolerate the pain by saying “stop” (pain tolerance). After the subjects indicated their pain tolerance, but before removing their hand from the ice water bath, subjects indicated their pain intensity and unpleasantness ratings. A maximum time limit of 5 minutes was imposed, though subjects were not informed of this limit.

Thermal Pain Testing—Thermal pain threshold and tolerance were determined by an ascending method of limits using a 1-cm-diameter contact thermode with the capability for a rise time of 10°C/second. The thermode was controlled by a personal computer, and thermal probe applied to the dominant volar forearm. During the pain testing, an adapting temperature of 38°C was maintained for 10 seconds. Then, the temperature increased directly to 41.5°C and from that point on, increased 0.5°C every 5 seconds until it reached 53°C or until the subject reached her tolerance. To determine thermal pain onset (threshold), subjects were instructed to press a mouse button (which terminated the stimulus) when the thermal percept first became painful. This was repeated three times and averaged to calculate thermal pain thresholds. Then, three series to determine average thermal pain tolerance were conducted by instructing the subject to press a mouse button when they were no long able to tolerate the pain. Immediately after the subjects indicated their pain tolerance on the third pain tolerance series, subjects indicated their pain intensity and unpleasantness ratings.

A 5 minute recovery period followed each pain procedure, since it has been suggested that 5 minutes should be the minimum amount of elapsed time between pain measurements [38].

Following pain testing, subjects were exposed to a mental stressor battery (results to be reported elsewhere).

DATA ANALYSIS

Group differences in demographic factors, psychiatric histories, months in full remission from the mood disorder, body mass index (BMI), pre-test rest systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), Perceived Stress Scale (PSS), Beck Depression Inventory (BDI), and State and Trait Anxiety were examined using a 2 (History of Mood Disorders: yes or no) × 2 (Race: Non-Hispanic White or African American) analysis of variance (ANOVA) or Chi-Square analyses.

Since African Americans had higher SBP levels (see Results), and since higher blood pressure is associated with reduced pain sensitivity [9, 11, 33, 35, 51, 64], we examined differences in pain intensity and unpleasantness ratings for each pain test, using a 2 (History of Mood Disorders) × 2 (Race) analysis of covariance (ANCOVA), with pre-test rest SBP as the covariate. Analyses did not indicate any relationship of BMI to any measure of pain sensitivity for any group. Thus, analyses were not adjusted for BMI. In addition, pain sensitivity was assessed using a 2 (History of Mood Disorders) × 2 (Race) repeated measures ANCOVA, with pre-test rest SBP as the covariate and time point (pain threshold and tolerance) as the repeated factor. Where significant interactions emerged, subsequent simple effects analyses were conducted in order to explore those effects.

RESULTS

Baseline and Demographic Factors (Table 1)

Significant main effects of Race were seen for BMI ($F(3,54) = 7.41$, $p<.01$), SBP ($F(3,54) = 3.88$, $p=.05$), and PSS ($F(3,54) = 5.48$, $p<.05$), since African Americans had greater BMI, PSS, and SBP than non-Hispanic Whites. Significant main effects for a History of Mood Disorders were seen for Age ($F(3,54) = 4.67$, $p<.05$) and Trait Anxiety ($F(3,54) = 5.26$, $p<.05$), since women with prior mood disorders were older (by approximately 2.5 years) and had greater Trait Anxiety (an associated feature of depression) than women with no prior mood disorders. No significant main or interactive effects involving a History of mood disorders or Race were obtained for DBP, HR, BDI, or State Anxiety.

Pain Intensity and Unpleasantness Ratings

There were no differences between African Americans and non-Hispanic White women in pain intensity ratings (ranges = 10-95 and 5-100, respectively, ps > .10) and pain unpleasantness ratings (ranges = 0-100 and 5-100, respectively, ps > .10) for any pain test. Similarly, there were no differences between women with and without prior mood disorders in pain intensity ratings (ranges = 5-100 and 10-90, respectively, ps > .10) and pain unpleasantness ratings (ranges = 0-100 and 5-100, respectively, ps > .10) for any pain task. Analyses also failed to reveal any significant Race × Prior Mood Disorder interactions.

Associations Between Race, History of Mood Disorders, and Pain Threshold and Tolerance

A main effect of Prior Mood Disorders was seen for ischemic pain (Figure 1) since, on average, women with prior mood disorders had higher threshold and tolerance to ischemic pain than women with no prior mood disorder ($F(1,50) = 4.1$, $p<.05$). A Race × Time Point interaction was also found ($F(1,50) = 9.2$, $p<.01$), since African American women had lower ischemic pain tolerance ($p<.001$), but not lower pain threshold (p=NS; Fig. 1) than non-Hispanic White women. As seen in Figure 2, a main effect of Race ($F(1,50) = 16.7$, $p<.001$) was seen for the cold pressor pain task, since on average, African American women had lower cold pressor threshold and tolerance than non-Hispanic White women. A Race × Prior Mood Disorder × Time Point interaction was also found ($F(1,50) = 9.4$, $p<.01$), since African Americans with prior mood disorders showed reduced tolerance to cold pressor pain than non-Hispanic Whites with prior mood disorders ($p<.0001$), while no ethnic differences in tolerance were evident in women with no history of mood disorders (see Fig. 2). While a similar pattern of effects was observed for thermal pain, with African Americans showing decreased pain tolerance than non-Hispanic Whites (48.3 and 49.3 degrees Celsius, respectively) and women with histories of mood disorders showing increased pain tolerance than women with no histories of mood disorders (49.4 and 48.2, degrees Celsius respectively), these differences did not reach statistical significance. Thus, no significant main effects or interactions were present involving thermal pain.

DISCUSSION

The results of our study indicate that experimental pain threshold and tolerance are influenced by both race and histories of mood disorders in women. Our study is among the first to show that histories of mood disorders are associated with decreased sensitivity to experimental pain in euthymic women. Bar et al. [5] assessed thermal pain sensitivity in women who were in full clinical recovery from major depressive disorder and found significantly increased pain threshold and tolerance in women with prior depression compared to controls. However, most of the women in recovery from depression were taking anti-depressant medication, while our study included only women not taking any prescription medication. Nonetheless, similar findings emerged in our study, since all women with histories of mood disorders, regardless

of race, showed increased pain threshold and tolerance to tourniquet ischemic pain, and only in women with prior mood disorders were the more pronounced racial differences in cold pressor pain tolerance evident.

Thus, our results, along with those of Bar et al. [5], suggest that persistent alterations in pain sensitivity may last beyond the remission of the mood disorder, and extend previous studies showing reduced sensitivity to experimental pain in subjects with current mood disorders [3, 4, 5, 19, 20, 30]. Consistent with the meta-analysis and review by Dickens et al. [19] that showed increased pain thresholds in patients with current depression, our results indicate that in women in full remission from a previous mood disorder there is also increased pain threshold and tolerance to experimental pain. Several hypotheses have been proposed to explain the association between mood disorders and reduced pain sensitivity, such as the presence of a more stoic behavior or affective indifference in depression [17], a true sensory deficit in psychiatric patients [12, 14], and slower reaction time to experimental pain stimuli in depression [1]. Although the mechanism(s) by which mood disorders influence pain sensitivity has not yet been identified, alterations in experimental pain sensitivity are likely to reflect alterations in autonomic and neuroendocrine mechanisms [18], disturbances associated with depression [41, 62, 63]. Thus, persistent alterations in pain sensitivity in women with a history of mood disorders may have implications for risk for a subsequent mood disorder episode. Longitudinal studies will be needed to address this issue.

Despite the conclusions of the meta-analysis [19] and the results of the present report, it is important to point out that other studies have yielded opposing results for ischemic pain sensitivity, finding either no differences in ischemic pain threshold [42], or reduced ischemic pain threshold [3] and tolerance [3, 42] in women with current mood disorders compared to controls subjects. Although the reasons for the discrepancies in the literature remain unknown, one possibility is that women who recover from depression and other mood disorders may be fundamentally different from those who do not recover. The inclusion of women with current mood disorders as well as those with histories of mood disorders in future studies would shed light on this issue.

Given the well documented evidence that women have increased clinical pain [55] and also show decreased experimental pain tolerance [45, 50, 60], it may seem paradoxical that women with prior mood disorders, who also have increased clinical pain [10], show increased experimental pain tolerance (i.e. reduced pain sensitivity). Lautenbacher and Krieg [29] have addressed this paradox of increased clinical pain complaints and reduced experimental pain sensitivity in depression, hypothesizing that diminished processing of painful stimuli could be responsible for both phenomena. The authors argue that reduced processing of nociceptive stimuli at both spinal and subcortical stages may cause hypoalgesia to phasic experimental pain, and at the same time cause hyperalgesia to clinical pain due to deficient activation of inhibitory systems [29]. Although Lautenbacher et al. [30] failed to find a significant correlation between clinical pain complaints and pain threshold in depressed patients, this does not rule out the possibility that alterations in central and peripheral pain processing contribute to both phenomena. Additional studies on underlying mechanisms contributing to alterations in pain sensitivity in mood disordered individuals are clearly indicated.

Regarding the racial differences observed, African American women showed decreased experimental pain tolerance to the tourniquet ischemic and cold pressor pain tasks compared to non-Hispanic White women, even after controlling for racial differences in resting blood pressure. However, it is important to note that the racial differences in cold pressor pain tolerance were only seen in women with histories of mood disorders. Our results indicating reduced pain tolerance in African American women are consistent with other reports [13, 23, 36, 50, 60], though this is the first study to examine racial differences in pain sensitivity in an

exclusively female sample. Although the mechanisms underlying racial differences in pain tolerance are unknown, the consistency of results across numerous studies underscores the robustness of the effect. Additionally, since pain intensity ratings reflect the sensory-discriminative aspect of pain, while pain unpleasantness ratings reflect the affective/emotional aspect of pain [44], our results revealing no significant ethnic differences in intensity and unpleasantness ratings for any of the pain tasks suggests that, for women, racial differences in pain sensitivity do not appear to be a function of either perceptual differences or affective interpretation of pain. While we did observe ethnic differences in perceived stress, with African American women reporting more stress in the month preceding testing then non-Hispanic White women, our study was not powered to examine whether perceived stress mediated the relationship between race and pain sensitivity. Future studies examining both biological and psychosocial factors that may contribute to racial differences in experimental and clinical pain are indicated, and may have implications for the racial disparities that exist in the management of clinical pain. Some of these studies are currently underway in our laboratory [36, 37].

Limitations to our study must be acknowledged. First, we did not assess chronic and current pain symptoms in our subjects, though all reported themselves to be in good health. Given that chronic pain has been shown to influence experimental pain sensitivity [39] and that African Americans [21, 34, 46] and individuals with depression [16, 52] have high rates of clinical pain, this would be important in future studies designed to address the paradox of increased clinical pain complaints but reduced experimental pain sensitivity in depression. Another limitation to our study is that we did not assess neuroendocrine measures, since alterations in plasma norepinephrine and hypothalamic-pituitary-adrenal axis factors are associated with both experimental pain sensitivity [2, 25] and depression [41, 62, 63]. Thus, studies examining neuroendocrine factors and pain perception in women with mood disorders are indicated. Limitations in regards to the pain intensity and unpleasantness ratings also exist. Although ratings did not differ by race or history of mood disorders, subjects endorsed pain intensity and unpleasantness ratings as low as 0, 5, and 10 out of 100 during pain tolerance, a time when subjects were instructed to indicate when they were no longer willing or able to tolerate the pain. It is possible that the subjects giving these low ratings did not understand the instructions, or that they simply had no motivation to continue further with the pain tasks, regardless of the low levels of intensity and unpleasantness. Therefore, it may be beneficial for future studies to assess indices of motivation, as well as to ensure that subjects fully understand the pain rating scale. Finally, the possibility exists for the presence of stress-induced analgesia with respect to differential carryover effects from one pain test to another, despite randomizing order of pain testing.

In summary, our results suggest that experimental pain threshold and tolerance in women is influenced by both race and lifetime histories of mood disorders. Women with prior mood disorders, regardless of race, were less sensitive to ischemic pain than women with no history of mood disorders. On the other hand, African American women, regardless of psychiatric history, were more sensitive to ischemic pain than non-Hispanic White women. The interplay between race and prior mood disorders was most evident during cold pressor pain where racial differences in pain tolerance were only evident for women with histories of mood disorders.

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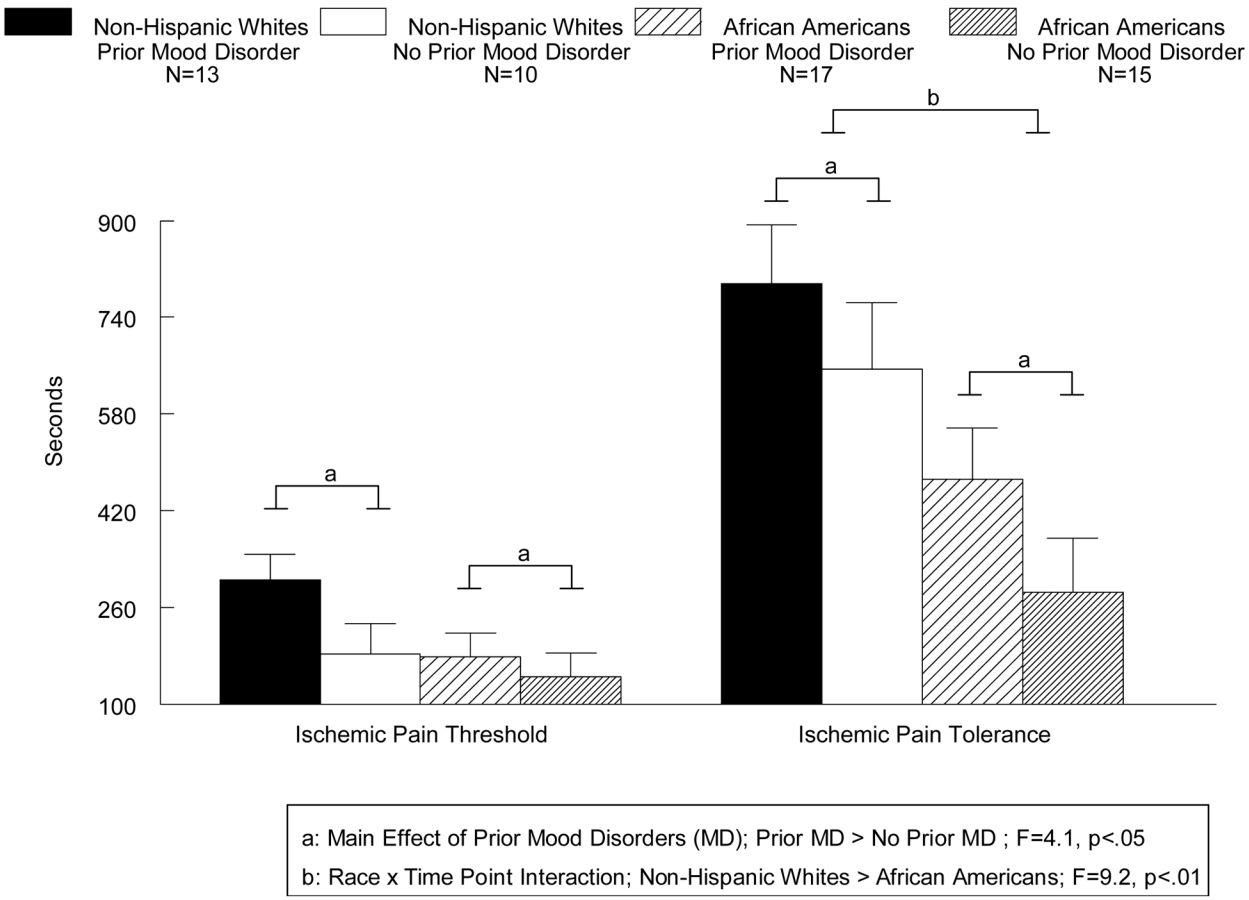

Fig. 1.
Mean (+SEM) ischemic pain tolerance (seconds) as a function of prior mood disorders and race in women.

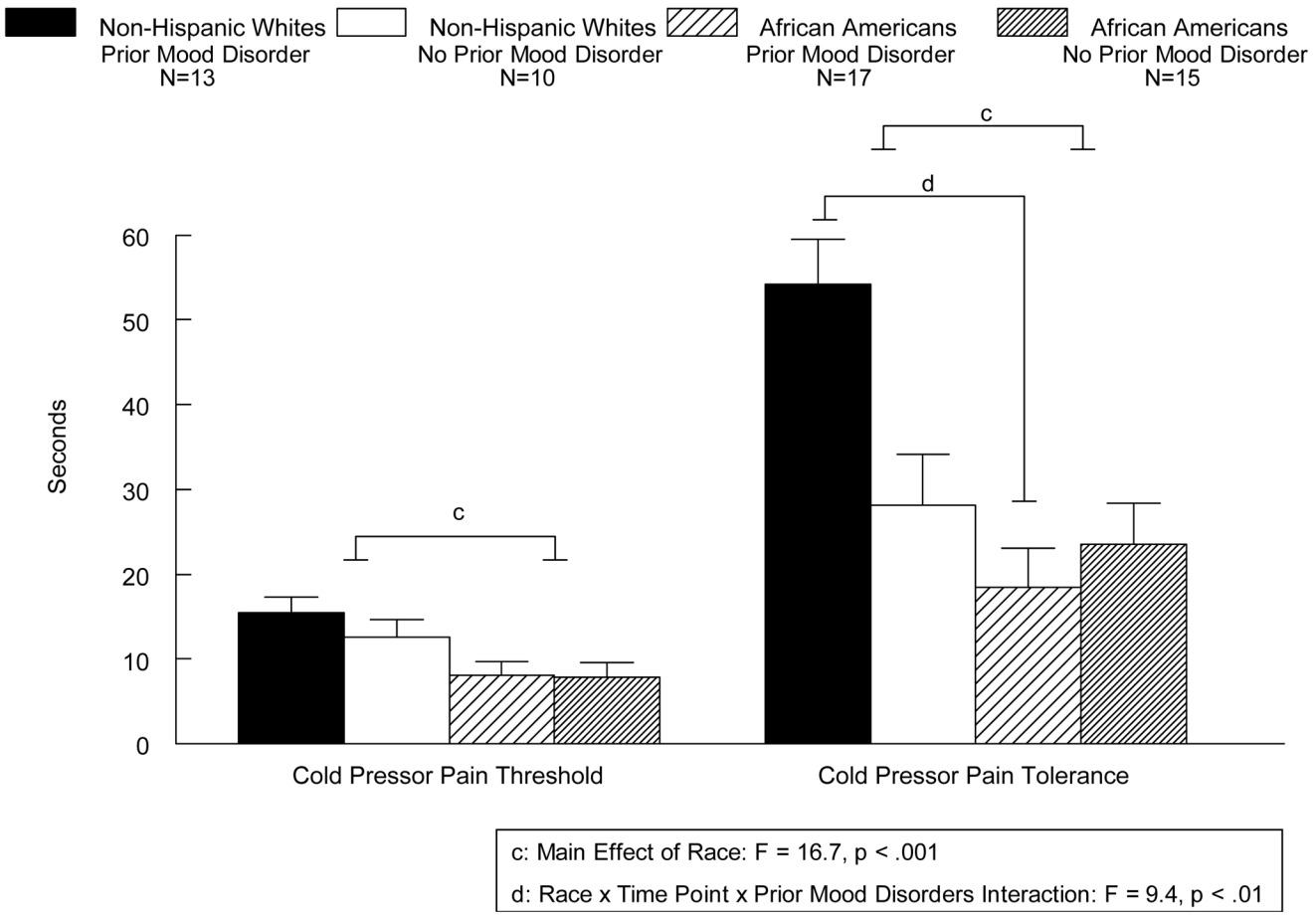

Fig. 2.
Mean (+SEM) cold pressor pain tolerance (seconds) as a function of prior mood disorders and race in women.

Table 1

Mean (+SEM) Baseline and Demographic Factors as a Function of Prior Mood Disorder and Ethnicity

	Non-Hispanic White Women		African American Women	
	Prior Mood Disorder (n = 13)	**No Prior Mood Disorder (n = 10)**	**Prior Mood Disorder (n = 17)**	**No Prior Mood Disorder (n = 15)**
Age[A]	26.5 (1.2)	23.2 (1.4)	25.9 (1.0)	24.0 (1.1)
BMI[B]	23.0 (1.6)	22.1 (1.9)	27.2 (1.4)	26.8 (1.5)
[C] Baseline Systolic Blood Pressure	104.6 (2.2)	105.5 (2.5)	109.6 (1.9)	108.9 (2.0)
Baseline Diastolic Blood Pressure	65.9 (2.2)	64.3 (2.5)	71.5 (1.9)	65.7 (2.1)
Baseline Heart Rate	64.5 (2.7)	65.3 (3.1)	66.0 (2.4)	68.3 (2.5)
[D] Perceived Stress Scale	16.4 (1.9)	14.4 (2.1)	20.1 (1.7)	19.5 (1.8)
Beck Depression Inventory	4.08 (1.1)	3.40 (1.2)	5.76 (0.9)	3.87 (1.0)
State Anxiety	26.9 (1.8)	27.1 (2.0)	26.9 (1.5)	30.2 (1.6)
Trait Anxiety[E]	33.5 (2.3)	31.3 (2.7)	36.9 (2.0)	29.6 (2.2)

African Americans > Non-Hispanic Whites:

Prior Mood Disorder > No Prior Mood Disorder:

[B] $p<.01$

[C] $p=.05$

[D] $p<.05$

[A] $p<.05$

[E] $p<.05$