Racial differences in presentations and predictors of acute pain following motor vehicle collision

Francesca L. Beaudoin a,b, Roee Gutman c, Wanting Zhai c, Roland C. Merchant a,d, Melissa A. Clark d,e, Kenneth A. Bollen f, Phyllis Hendry g,h, Michael C. Kurz i, Christopher Lewandowski j, Claire Pearson k, Brian O’Neil l, Elizabeth Datner m, Patricia Mitchell n, Robert Domeier o, Samuel A. McLean o,p

a Emergency Medicine, Alpert Medical School of Brown University; b Health Services Policy and Practice, School of Public Health, Brown University; c Biostatistics, School of Public Health, Brown University; d Epidemiology, School of Public Health, Brown University; e Quantitative Health Sciences, University of Massachusetts Medical School; f Department of Psychology and Neuroscience, University of North Carolina Chapel Hill; g Department of Sociology, University of North Carolina Chapel Hill; h Department of Emergency Medicine, University of Florida College of Medicine-Jacksonville; i Department of Emergency Medicine, University of Alabama School of Medicine; j Department of Emergency Medicine, Henry Ford Health System; k Department of Emergency Medicine, Wayne State University School of Medicine; l Department of Emergency Medicine, Einstein Healthcare Network; m Department of Emergency Medicine, Boston Medical Center; n Department of Emergency Medicine, St. Joseph Mercy Hospital; o Anesthesiology, University of North Carolina Chapel Hill; p Emergency Medicine, University of North Carolina Chapel Hill

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Manuscript Correspondence:
Francesca L. Beaudoin, MD, PhD
Department of Emergency Medicine
593 Eddy Street – Claverick 2
Providence, Rhode Island 02903
(401) 444 – 2577
Abstract

African-Americans experience a greater burden of acute pain than non-Hispanic white individuals across a variety of acute medical conditions, but it is unknown if this is the case following trauma. We evaluated pain, pain-related characteristics (e.g. peri-traumatic distress), and analgesic treatment in two cohorts of individuals (African-American (n=931) and non-Hispanic white (n=948)) presenting to the emergency department after a motor-vehicle crash (MVC). We performed a propensity-matched analysis (n=796 in each group) to assess racial differences in acute pain in the ED. In multivariable models conducted within the matched sample, race was associated with moderate to severe axial pain (OR 3.2; 95% CI 2.1, 5.0, p<0.001) and higher average NRS scores (1.3; 95% CI: 1.1, 1.6; p=0.001). After adjustment for pain and other covariates, Non-Hispanic white patients were: more likely to receive an opioid analgesic in the ED (OR 2.0; 95% CI 1.4, 3.0, p<0.001) or at discharge (OR 4.9; 95% CI 3.4, 7.1, p<0.001), and also less likely to receive an NSAID in the ED (OR 0.54; 95% CI 0.38, 0.78; p=0.001) or at discharge (0.31; 95% CI 0.43, 0.84). Racial differences in the severity of acute post-traumatic pain following a motor vehicle collision are not explained by factors such as socio-economic status or crash characteristics. Despite a higher burden of acute pain, African-Americans were less likely to receive opioid analgesics and more likely to receive NSAIDs. Further work is needed to understand the relationship between pain severity, disparities in analgesic treatment and longer-term outcomes, such as post-MVC chronic pain.

Keywords: Acute pain; post-traumatic pain; racial differences; motor-vehicle collision; propensity matching
1. Introduction

Motor Vehicle Collisions (MVCs) are one of the most common life-threatening traumatic stress exposures in industrialized countries, resulting in over 4 million U.S. emergency department (ED) visits each year.[3] An MVC includes exposure to a life-threatening event (psychological stress) and tissue trauma (physical stress), two factors that when combined, heighten the risk for chronic pain development, post-traumatic stress disorder (PTSD), and impaired physical functioning.[12; 21; 32; 34; 35; 37; 56] Some populations are at higher risk than others: we recently found that rates of moderate to severe axial pain after MVC in a cohort of African-Americans was over twice that previously reported in another ED-based cohort of non-Hispanic white MVC patients.[25; 35] Severity of acute pain is highly predictive of persistent pain.[21; 35; 47] Therefore it is important to understand racial differences in acute pain after trauma, and specifically an MVC. In addition, racial differences in acute pain may have important implications for treatment and in particular, disparities in pain management. To our knowledge, racial differences in acute post-traumatic pain have not been previously examined.

In both various medical conditions (e.g. chronic pancreatitis, osteoarthritis) and under experimental pain conditions, African-Americans experience a greater burden of acute pain than non-Hispanic white individuals.[7; 10; 33; 39] While it is tempting to attribute these differences to socioeconomic disadvantage, data suggest that disparities in pain experienced by African-Americans are not accounted for by socioeconomics alone.[14] Contrary to this and relative to non-Hispanic white individuals, African-Americans are perceived to have lower levels of pain[20] and often receive less analgesia than other racial and ethnic groups being treated for the same conditions.[18; 24; 36] It is important to examine racial differences in acute pain, not just because of the implications for pain management, but also because of the potential impact on chronic pain development and related negative sequelae, such as PTSD.

To examine racial differences in acute post-traumatic pain following MVC, our primary objective was to compare pain (e.g. severity, location) and pain-related characteristics (e.g. catastrophizing, somatic symptoms) immediately following MVC in a propensity-matched cohort of African-American and non-Hispanic white patients presenting to the ED. Secondarily, we aimed to provide potential insights in biologic or
socio-cultural mechanisms of pain by exploring whether or not predictors of pain severity (pre-MVC and MVC characteristics) following MVC are modified by race.

2. Methods

2.1 Study Design and Setting

This investigation was a secondary analysis of data collected as part of two large, multi-center, prospective cohort studies of adult patients who presented to an ED within 24 hours of an MVC and were discharged to home after evaluation. The primary aim of the parent studies was to assess the association between epidemiologic and genetic characteristics and pain outcomes after a MVC. Data were collected at the ED visit via patient interview and self-administered surveys, and then at 6 weeks, 6 months, and 1 year after the ED visit. Participants in the non-Hispanic white cohort were enrolled at eight EDs in four states (Florida, Massachusetts, Michigan, and New York) between February 2009 and October 2011. Participants in the African-American cohort were enrolled at seven EDs across the U.S. (Alabama, Florida, Massachusetts, Michigan, New Jersey, Pennsylvania and the District of Columbia) between September 2012 and September 2016. The studies were approved by the Institutional Review Boards (IRB) at each of the study sites and all participants provided written informed consent. Further details of study methodology are described elsewhere.[30; 35; 38]

2.2 Study Population

Patients aged 18 to 65 years old who presented to a participating ED within 24 hours of an MVC and were unlikely to require hospitalization were screened for study eligibility. Patients who were admitted to the hospital, had any fractures other than phalangeal fractures, had more than 4 lacerations requiring sutures or a single laceration more than 20 cm in length, or had intracranial or spinal injuries were excluded. Spinal injury was defined by the presence of a fracture, dislocation, or new neurologic deficit. Patients who were not alert and oriented also were excluded, as were pregnant patients, inmates, and patients unable to read and understand English. Enrollment in the first cohort was limited to non-Hispanic white subjects of European-American descent and the second cohort was limited to black subjects of African-American descent; race and ethnicity
was by self-identification. The parent studies enrolled participants of specific racial and ethnic makeup because the studies included genetic analyses and population stratification within the sample can introduce bias. [8]

2.3. Measures

Participants completed study assessments while in the ED. The assessments were administered via a combination of a structured RA-administered interview (e.g., characteristics) and self-administered web-based forms on a lap-top computer (e.g., Peri-traumatic Distress Inventory, Pain Catastrophizing Scale). All participants completed the interview in the same manner and sequence. The following characteristics were assessed: sociodemographics (e.g. age, sex, education level, income level, employment status, marital status); pre-MVC health status, medical history, medication history, and MVC characteristics (e.g. position in the vehicle, speed, seatbelt use); these have been detailed elsewhere along with complete study methodology. [30] Data regarding participants’ injuries were abstracted from the ED medical record.

2.3.2. Pain: severity, location, and presence of widespread pain

All participants were assessed by a trained RA after evaluation by their treating provider, but before analgesic treatment was received in the ED; analgesics taken prior to arrival in the ED were recorded. Pain assessments included locations of pain, severity of pain, and the presence of widespread pain. Pain was assessed in 19 discrete body regions evaluated in the Regional Pain Scale [55] using verbal patient self-reports. In each body region in which the participant reported pain, average pain severity was assessed using a verbal 0-to-10 numerical rating scale (NRS). Verbal scores have advantages over written responses (e.g. visual analogue scales) in acute care settings because of limitations imposed by pain and injury. In addition, verbally administered NRSs have been validated as highly correlated with visual analogue scale scores. [23] Widespread pain was defined according to the American College of Rheumatology definition: axial pain plus pain in an upper and lower segment plus pain on the left- and right-side. Moderate to severe axial pain was defined as a pain score of ≥ 4 out of 10 on the NRS in either the back, neck or shoulders (axial). [27] Moderate to severe axial pain was evaluated because it is associated with risk for chronic pain development[34; 47] and because it
is correlated with other patient-centered outcome measures, such as pain interference with function.[45]

2.3.3. Pain related symptoms: distress, optimism, and somatic symptoms

Since pain is a multi-dimensional experience, other pain-related characteristics were assessed: somatic symptoms, peri-traumatic distress, pain catastrophizing, and generalize optimism about recovery.

Somatic symptom burden was assessed by asking participants to rate the severity (0 to 10 NRS) of 10 common posttraumatic symptoms.[26] The total number of symptoms (0 to 21 pre-MVC, 0 to 10 in the ED) was calculated as the number of somatic symptoms with a response of ≥1). Pre-MVC somatic symptom burden was also assessed in the ED by asking participants to rate the severity (0 to 10 NRS) of 21 common somatic symptoms (eg., nausea, dizziness, fatigue, ringing in ears, constipation, or diarrhea) during the week prior to the MVC.

Peri-traumatic distress was assessed using the Peritraumatic Distress Inventory. A Peritraumatic Distress Inventory cut-off score of ≥23 was used to define marked distress symptoms (‘‘distress’’).[37] The Peritraumatic Distress Inventory has demonstrated good internal reliability among MVC patients (Cronbach’s alpha = 0.8) and good test-retest reliability (r=0.77).[6]

Pain catastrophizing was assessed using the Pain Catastrophizing Scale (PCS). This 13-item scale (combined score range 0 to 52) assesses an individual’s tendency to experience fear, anxiety, and helplessness in response to pain. Higher scores indicate higher levels of catastrophizing and the test has demonstrated good internal reliability (Cronbach’s alpha = 0.87).[51] The PCS has been validated to assess pain catastrophizing following an MVC.[50]

Generalized optimism about recovery was assessed using the10-item Life Orientation Test–Revised survey (LOTR).[16] Only items 1, 2, 3, 4, 7, 9, and 10 are scored. The Life Orientation Test-Revised has demonstrated good internal reliability (Cronbach’s alpha = 0.8) and good test-retest reliability (r=0.78). To assess confidence in recovery, participants were asked “‘How certain, or sure, are you that you will fully recover from this accident’” on a scale of 0 to 10 scale, where 0 denotes “‘certain you will not recover’” and 10 denotes “‘certain you will recover fully.”’ This measure has been previously used to assess confidence in recovery in
MVC patients. Lastly, participants were asked to estimate how many days they expected it would take for them to recover physically and how many days they expected it would take for them to recover emotionally from the accident.

2.4 Overview of Analyses, Matched Cohort

An evaluation of racial differences necessitates the use of observational data. In order to control for confounding of the relationship between race and pain, we performed our analysis in a cohort of African-Americans and non-Hispanic white MVC patients matched by propensity scores. A propensity-matched analysis was conducted for two main reasons. First, we desired to obtain a marginal effect estimate of race on pain characteristics. Standard regression would produce an effect estimate conditional on specific covariates (e.g. socioeconomic status). Propensity scoring has been advocated as a means to examine racial differences and disparities for this reason.[57] While a marginal effect estimate may be produced with procedures such as standardization,[41] propensity scoring is a semi-parametric approach that may be more robust to model misspecification.[57] Second, there are likely many variables that confound the association between race and pain after a MVC, and propensity scoring is capable of dealing with high-dimensional confounding.[42-44]

In order to ensure that we had chosen the matching method that would result in the least amount of bias, we also conducted trials of two additional matching algorithms: Mahalanobis distance matching and coarsened exact matching. Both Mahalanobis distance matching and coarsened exact matching resulted in larger standardized mean differences and greater reduction in the final sample size, a finding demonstrated elsewhere.[17; 48] Descriptive and inferential statistics were performed using Stata MP 13.0 statistical software (StataCorp, 2013. College Station, Texas).

2.4.2 Multiple Imputation of Missing Covariate Data

Predictors of the outcome were recorded with minimal missingness; over 95% of the baseline covariates had no more than 1% missing values, and the maximum proportion of missing for any baseline covariate was 6.4%. Prior to calculation of the propensity score, multiple imputation of missing data was performed using chained
equations; this method specifies the conditional models for all of the variables with missing values.[52; 54] Multiple imputation is the preferred method to deal with missing data when propensity scores are being used as calculation of the propensity score using only complete cases can lead to a more biased point estimate.[29; 31] Covariates were imputed using only indicators for race (African-Americans or non-Hispanic white) and other covariates including: sociodemographics, pre-MVC health status, medical history, medication history and use, and MVC characteristics. Imputation did not include any outcome variables (pain and pain-related characteristics) to avoid introducing a spurious association with the outcome. Imputation was performed using logistic regression for binary variables, ordinal logistic regression for ordinal variables, linear regression for continuous variables and predictive mean matching for semi-continuous variables. Twenty ($m=20$) complete imputed datasets were generated and used for calculation of the propensity score and further analyses.

2.4.3 Propensity Score Modeling
The conditional probability of being African-American ($W_i=1$) versus non-Hispanic white ($W_i=0$), given the observed covariates, was estimated using logistic regression in a stepwise iterative fashion.[22] In brief, initial covariate selection for the propensity score model was based on substantive knowledge of factors that might predict a person’s race (age, gender, education, income, employment and partner status). Next, other covariates (e.g. medical history), as well as quadratic and interaction terms were selected based on likelihood ratio testing. The final propensity score model included variables (n=27) comprising socio-demographic data, MVC characteristics, past medical history, and study site. Individuals with propensity scores outside the region of common support (area with overlapping propensity scores between groups) were truncated. Socio-demographic characteristics were compared between those included and excluded (truncated) from analyses.

The propensity scores were then used to assemble two cohorts, African-Americans and non-Hispanic white MVC patients, matched by propensity scores. We performed 1:1 nearest neighbor matching using greedy matching with replacement. Matching with replacement has been shown to result in decreased bias.[11] We imposed a caliper of 0.25 standard deviations of the linear propensity score (propensity score on the logit scale) in order to reduce bias by eliminating poor matches at the extremes.
We then assessed whether covariates were balanced between groups: Student’s t-test for continuous variables and z-test for binary variables to assess differences between groups by testing the null hypothesis that average difference in average covariate values is equal to zero for each covariate. We also determined the standardized mean difference between groups before and after propensity score adjustment. A standardized mean difference $\leq 0.1$ is considered to be optimal.[40; 42] Imbalance of covariates was re-addressed by re-specification of the initial propensity score model and then repeating the procedures above until the distribution of covariates was similar between the two groups, African-Americans and non-Hispanic white participants. Although a matched sample was generated, we did not conduct a paired analysis (e.g. paired t-test) because we did not utilize exact matching methods.[49] Although the covariate distribution will be the same between the two cohorts on average, unlike exact matching methods, a pair matched by the propensity score can still differ with respect to various covariates (e.g., age). Despite this, matching based on propensity scores can result in more narrow confidence intervals, therefore standard errors were corrected to account for this.[1; 2]

2.4.4 Racial differences in pain characteristics immediately following MVC

Within the matched cohort, we examined racial differences in acute pain, a greater number of body regions with acute pain, as well as pain-related symptoms (such as somatic symptoms and peri-traumatic distress) and analgesics (opioids and NSAIDs) administered in the ED and prescribed at discharge. Pain was assessed prior to analgesic administration in the ED. To examine differences in pain and pain-related symptoms between the two groups, within the matched cohort, we used Student’s t-test or Mann-Whitney U test for continuous outcomes and the Chi-square test for categorical outcomes. Given that matching was performed with replacement, estimates were made using frequency weights in order to reflect the number of times cases are selected as a match. We determined the relationships between race and pain severity, and race and analgesic administration, using multivariable logistic regressions within the matched sample. The final model included race, as well as other covariates thought to confound the relationship between race and pain or analgesic use. This “doubly robust” method of adjusting for covariates even after matching, attempts to reduce residual confounding that may occur after propensity score matching.[5] Covariate inclusion was based on substantive knowledge and
included: socio-demographic characteristics, past medical history (including medication use), and crash characteristics.[19; 47; 53; 56] The Hosmer-Lemeshow test was used to examine the goodness of fit of the multivariable logistic model.[28]

2.4.3. Racial differences in predictors of acute pain.

We hypothesized that African-American and non-Hispanic White MVC patients will have differences in pain characteristics, and specifically the primary outcome of pain severity (i.e. the proportion with moderate to severe axial pain). After comparisons were made, we attempted to further understand any observed differences by exploring whether or not predictors of pain severity (pre-MVC and MVC characteristics) following MVC are modified by race. First, we used regression and internal validation techniques to develop a model that predicts the likelihood that a patient will have moderate to severe acute axial pain in the ED. Next, we examined whether there was statistical interaction between race and the covariates in the model in order to posit racial differences in factors that predict pain.

Candidate predictor variables were selected based on substantive knowledge. We then performed forward stepwise predictor selection (logistic regression) starting with age and sex. Age and sex were included in the model because we had a strong \textit{a priori} belief that these factors are associated with pain. Stepwise selection was chosen because it performs automated variable selection and is easy to implement, interpret, and tends to produce more parsimonious models compared to other selection heuristics (e.g., backwards selection, forward-backwards selection, all-subsets regression).

Forward stepwise selection adds predictors in a sequential fashion and keeps them in the model if adding them improves the fit of the model. Specifically, we kept the predictor if the likelihood ratio p-value is $<0.10$ and the Akaike information criterion (AIC) value is lower for the model \textit{with} versus \textit{without} the considered predictor. After all linear terms were considered, we then examined interaction terms (cross-products), followed higher order univariate terms (e.g. $\text{age}^2$). We considered examined alternative modeling strategies, such as Least Absolute Shrinkage and Selection Operator (LASSO), but this did not improve model performance.
We performed the step-wise selection (logistic regression) utilizing 10-fold cross-validation in order to limit model over-fitting. In brief, the data was split into 10 random deciles. Nine of the deciles were used to develop a model (training set) and then predictions were made on the remaining decile (test set). This was repeated 10 times until each decile of the data has served as a test portion.

Model performance was assessed in each iteration of the cross-validation procedure using the area under the Receiver Operating Characteristic curve (AUC): a plot of the detection rate (true positives) versus the false positive rate in the test versus training sets. An ideal model (no false positives with 100% detection rate) has an AUC of 100%, and a model that approximates chance has an AUC of 50%. We also measured calibration by constructing calibration plots – a comparison of predicted and observed probabilities of outcomes by decile of predicted risk. The final model was chosen based on maximizing the AUC and calibration. After the final model was selected, we introduced interaction terms with race and each of the predictors, and interaction terms were retained based on likelihood ratio testing. The predictors in the model are presented in the results section. We do not report on the coefficients or odds ratios associated with model because the goal of model was not to develop a predictive tool, but rather to provide insights in mechanisms and differences in pain. A tool that predicts acute pain in the ED would have little clinical value as such a instrument would not be used to guide analgesia, nor would it supplant assessing a patient’s pain directly.

3. Results

3.1 Cohort characteristics

A total of 931 participants completed the ED interview in the African-American cohort; 948 participants completed the ED interview in the non-Hispanic white cohort. In both cohorts, slightly more than 60% of enrolled study participants were female (Table 1). The median age in both cohorts was 35 years (range 18 to 65). The vast majority of participants in both cohorts (>95%) had musculoskeletal strain only, the remainder had minor associated injuries such as abrasions. Prior to matching, education, income, partner status, and employment were significantly different between the African-American and non-Hispanic white cohorts. Socio-demographic characteristics of the two cohorts, before and after matching are displayed in table 1. After
matching, socio-demographic differences were modest and no longer statistically significant (Table 1). In crude
(unmatched, unadjusted) analyses, African-American participants experienced more severe pain in the early
aftermath of MVC (NRS 7.5 (IQR 6.9) vs. 6 (4,7); p<0.001) and more body regions with pain (4 (IQR 2, 6) vs.
3 (IQR 1, 5), p<0.001).

3.2. Racial differences in acute pain outcomes: matched bivariate analyses

3.2.1. Pain severity and extent

Within the matched cohort, African Americans had more severe pain and more body regions with pain (Table 2)
in the early aftermath of MVC. A higher proportion of African-American participants had moderate to severe
pain (94.0% vs. 82.3%). However, differences between African American and Non-Hispanic white participants
in widespread pain were no longer significant (26.6% vs. 20.1%).

3.2.2 Pain location

Figure 1 displays the distribution of moderate or severe pain in African American and Non-Hispanic white
participants within the matched cohort. In both groups, the neck was the body region that had the highest
proportion of participants reporting moderate to severe pain. Racial differences in acute pain were most
pronounced in the axial region: a significantly higher proportion of African-Americans reported moderate to
severe pain in three out of four of the axial areas: neck, shoulders, and lower back.

3.2.3. Other symptoms: somatic symptoms, psychologic symptoms, optimism, and recovery expectations

In the matched bivariate analyses, there were no significant differences in peritraumatic distress or pain
catastrophizing between African American and Non-Hispanic white participants. African Americans reported
significantly fewer somatic symptoms (e.g., nausea), but had less overall optimism and perceived that it would
take longer to recover both physically and emotionally from their accident (Table 3).
3.4 Racial differences in analgesic treatment: matched univariable analyses

In matched univariable analyses, African American participants were less likely than non-Hispanic white participants to receive an opioid analgesic in the ED, and were more likely to receive an NSAID (Table 2). Similarly, African Americans were also less likely than Non-Hispanic white participants to receive a prescription for an opioid analgesic at discharge, and were more likely than Non-Hispanic white participants to receive an NSAID at discharge (Table 2).

3.5. Racial differences in pain and analgesic treatment: matched multivariable analyses

In the multivariable models conducted in the matched sample, African Americans were more likely to have moderate to severe axial pain (OR 3.2; 95% CI 2.1, 5.0, p<0.001) and higher NRS scores on average (1.3; 95% CI: 1.1, 1.6; p=<0.001). After adjustment for pain and other covariates, Non-Hispanic white patients were more likely to receive an opioid analgesic in the ED (OR 2.0; 95% CI 1.4, 3.0, p<0.001) and at discharge (OR 4.9; 95% CI 3.4, 7.1, p<0.001); non-Hispanic white patients were also less likely to receive an NSAID in the ED (OR 0.54; 95% CI 0.38, 0.78; p=0.001) or at discharge (0.31; 95% CI 0.43, 0.84).

Within the matched cohort, the following variables were predictive of acute moderate to severe pain in the ED: race, history of depression, gender, history of chronic opioid use, BMI, having less than a high school education, being in a stopped vehicle and smoking (AUC = 0.71; 95%CI 0.68, 0.74). Based on likelihood ratio testing, there was no significant interaction between race and any of the predictors. When the final model was applied to the entire unmatched cohort, this model still showed fair performance (AUC = 0.76; 95%CI 0.74, 0.78).

4. Discussion

In a cohort of MVC patients matched on patient and crash characteristics, African-Americans had more severe pain and more body locations with pain than non-Hispanic White participants. Our work adds to the growing body of evidence that suggest that African-Americans experience more intense pain as has been demonstrated in studies of other acutely painful conditions (e.g. acute pancreatitis).[7; 10; 33; 39] While
observed differences in the unmatched sample may be confounded by factors like socio-economic status (SES), this is very unlikely to be the case in the matched analyses. In other words, differences in pain intensity cannot be explained by SES, crash characteristics or clinical history. It may be tempting to argue that differences in the severity of acute pain may be due to differences in the reporting of pain, rather than actual differences in pain perception or sensitivity. However, African-Americans reported fewer somatic symptoms and similar levels of peri-traumatic distress and pain catastrophizing, making response bias less likely. In addition, our previous work has shown that African-Americans have high rates of transitioning to chronic pain after experiencing acute pain following an MVC, and the severity of acute pain is likely the strongest predictor of chronic pain development.[33-35] It is therefore likely the acute pain experience is influenced by other factors and not just due to bias in the participants’ reports of pain as this would be unlikely to be sustained over time and across studies.

To understand factors influencing acute pain in the ED, we performed predictive modeling and examined whether there was interaction between race and the predictors in the final model. In the final model, a history of depression, female gender, history of chronic opioid use, higher body mass index (BMI), having less than a high school education, being in a stopped vehicle, being a cigarette smoker, and African-American race were significant predictors of having higher pain severity in the ED. The fact that race is a significant predictor is congruent with our findings that there are racial differences in acute pain. Several factors that we identified as predictors of acute pain (depression, gender, education and being in stopped vehicle) have been shown in other studies to be predictive of ongoing pain and non-recovery after an MVC.[35; 46] This consistency implies generalizability of our study findings. Other predictors have a strong biologic plausibility – both chronic opioid use and tobacco smoking have been demonstrated to result in alterations to pain sensitivity and perception.[4; 15] While we identified a subset of variables that had satisfactory predictive ability in determining which patients would have moderate to severe pain, we did not identify any statistically significant interactions. This suggests that there are other unmeasured factors influencing observed racial differences in pain severity (e.g. biological).
It is also worth noting that on average, compared to non-Hispanic white patients, African-Americans reported less certainty about their recovery following the accident and estimated that the twice as long was needed to recover both physically and emotionally. Early expectations for recovery have been demonstrated to be an important prognostic factor for pain and functional improvement after an MVC and are viewed as potentially modifiable.[9] Future work might examine the role that both pain severity and recovery expectations have in explaining racial differences in chronic pain development.

The proportion of African-Americans receiving any analgesics in the ED (both opioids and NSAIDs) was similar to non-Hispanic white patients (about 83% in both cohorts). However, after adjusting for pain severity and other covariates, non-Hispanic white patients were twice as likely to receive an opioid analgesic than African-Americans. This indicates possibly disparities in analgesic treatment, a finding that has been noted previously.[13; 36] However, it is not known which participants may have been offered (and declined) opioid pain medications as our data only reflect medications given and not the desire for pain medication. We also did not capture potential contraindications to one treatment or the other. Future work should examine whether disparities in analgesic prescribing impact outcomes such as pain after discharge and transition to chronic pain.

4.1. Limitations

As with any observational study, there exists the possibility of unmeasured confounding (due to factors that were either not measured or measurable) leading to bias in the results of the matched analysis. Given our research question is only amenable to studies of observational data, we have attempted to minimize confounding through use of propensity score matching and further adjustment for residual confounding in our regression analyses. One potential source of residual confounding is that our study sites were not the same for the two cohorts. Discrepant analgesic prescribing practices between study sites could account for these differences and the role of the site and also the individual provider should be examined in further work on this topic. Our predictive model is only meant to give insight into potential biologic or socio-cultural mechanisms of acute pain pathogenesis and cannot be interpreted as causal. Although the scales we use have good reliability,
the remaining measurement error in them may influence our coefficient estimates. Lastly, our study results may not generalize to other populations or clinical conditions causing acute pain, further study is needed.

4.3. Conclusion

Racial differences in the severity of acute post-traumatic pain following a motor vehicle collision are not explained by factors such as socio-economic status or crash characteristics. Despite a higher burden of acute pain (more intense pain and more body areas with pain), African-Americans were less likely to receive opioid analgesics and more likely to receive NSAIDs. Further work is needed to understand the relationship between pain severity, disparities in analgesic treatment and longer-term outcomes, such as the development of post-MVC chronic pain.

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References


Figure 1. Proportion of participants in the matched cohort experiencing moderate to severe pain, by cohort and body region
Table 1. Socio-demographic characteristics of participants in both cohorts of motor vehicle collision patients, before and after propensity-score matching.

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<td>33.8 (30.6 – 37.3)</td>
<td>36.7 (29.2 – 44.8)</td>
<td>0.228</td>
</tr>
<tr>
<td>$20,000 – $80,000</td>
<td>57.1 (53.4 – 60.6)</td>
<td>53.7 (50.4 – 57.1)</td>
<td></td>
<td>56.7 (43.1 – 60.1)</td>
<td>54.9 (47.1 – 62.4)</td>
<td></td>
</tr>
<tr>
<td>&gt; $80,000</td>
<td>8.2 (6.4 – 10.5)</td>
<td>32.4 (29.3 – 35.6)</td>
<td></td>
<td>9.5 (7.6 – 11.8)</td>
<td>8.4 (6.0 – 11.6)</td>
<td></td>
</tr>
<tr>
<td>Education complete</td>
<td></td>
<td></td>
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<tr>
<td>Less than high school</td>
<td>8.7 (7.0 – 10.7)</td>
<td>4.4 (3.3 – 6.0)</td>
<td>&lt;0.001</td>
<td>7.9 (6.2 – 10.0)</td>
<td>6.2 (4.1 – 9.4)</td>
<td>0.170</td>
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<tr>
<td>High school graduate</td>
<td>31.4 (28.5 – 34.5)</td>
<td>19.5 (17.0 – 22.1)</td>
<td></td>
<td>31.8 (28.6 – 35.1)</td>
<td>27.2 (22.7 – 32.2)</td>
<td></td>
</tr>
<tr>
<td>College graduate</td>
<td>41.2 (38.0 – 44.4)</td>
<td>39.0 (35.9 – 42.2)</td>
<td></td>
<td>40.3 (37.0 – 43.8)</td>
<td>42.9 (37.7 – 48.3)</td>
<td></td>
</tr>
<tr>
<td>Post-graduate level</td>
<td>18.7 (16.4 – 21.4)</td>
<td>37.1 (34.1 – 40.2)</td>
<td></td>
<td>20.0 (17.3 – 22.9)</td>
<td>23.7 (19.4 – 28.5)</td>
<td></td>
</tr>
<tr>
<td>Lives with partner</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>37.1 (34.1 – 40.3)</td>
<td>51.9 (48.7 – 55.1)</td>
<td>&lt;0.001</td>
<td>38.7 (35.4 – 42.1)</td>
<td>42.7 (35.4 – 50.3)</td>
<td>0.103</td>
</tr>
<tr>
<td>Employment status</td>
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<td></td>
</tr>
<tr>
<td>Work full time</td>
<td>54.0 (50.8 – 57.2)</td>
<td>58.3 (55.2 – 61.4)</td>
<td>0.061</td>
<td>55.3 (51.8 – 58.7)</td>
<td>55.9 (50.5 – 61.1)</td>
<td>0.843</td>
</tr>
<tr>
<td>Disabled</td>
<td>6.0 (4.7 – 7.8)</td>
<td>3.2 (2.2 – 4.5)</td>
<td>0.003</td>
<td>5.7 (4.2 – 7.5)</td>
<td>5.0 (3.1 – 8.0)</td>
<td>0.673</td>
</tr>
</tbody>
</table>

Continuous data are presented as mean (SD). Categorical data are displayed as proportions (%) and 95% confidence intervals unless otherwise specified.
Table 2. Pain and analgesic treatment following motor vehicle collision in a African-American vs. Non-Hispanic white participants (after propensity matching)

<table>
<thead>
<tr>
<th></th>
<th>African-American (n=796)</th>
<th>Non-Hispanic white (n=796)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain intensity (NRS)</td>
<td>7.1 (2.2)</td>
<td>5.7 (2.5)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Moderate to severe pain (%)</td>
<td>94.0 (92.1 – 95.4)</td>
<td>82.3 (77.8 – 86.0)</td>
<td>0.003</td>
</tr>
<tr>
<td>Number of body regions with moderate to severe pain</td>
<td>4.4 (3.6)</td>
<td>3.5 (3.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Widespread pain (%)</td>
<td>26.6 (23.7 – 29.8)</td>
<td>20.1 (16.2 – 24.8)</td>
<td>0.266</td>
</tr>
<tr>
<td>Analgesic Treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>In the ED:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opioids</td>
<td>26.0 (22.3 – 27.7)</td>
<td>38.4 (28.8 – 48.9)</td>
<td>0.001</td>
</tr>
<tr>
<td>NSAIDS</td>
<td>57.8 (53.2 – 62.2)</td>
<td>44.1 (34.5 – 54.2)</td>
<td>0.003</td>
</tr>
<tr>
<td>At discharge:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Opioids</td>
<td>30.9 (26.9 – 35.2)</td>
<td>69.6 (60.8 – 77.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>66.7 (62.3 – 70.9)</td>
<td>42.4 (33.1 – 52.4)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Continuous data are presented as mean (SD). Categorical data are displayed as proportions (%) and 95% confidence intervals unless otherwise specified. NRS= numerical rating score.

*Proportion of participants with NRS ≥ 4 in any location.
Table 3. Differences in somatic, symptoms, psychological symptoms, optimism and recovery expectations following motor vehicle collision in African-American vs. Non-Hispanic white participants (after propensity matching)

<table>
<thead>
<tr>
<th></th>
<th>African-American</th>
<th>Non-Hispanic white</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=796)</td>
<td>(n=796)</td>
<td></td>
</tr>
<tr>
<td>Peri-traumatic somatic symptoms</td>
<td>3.6 (2.7)</td>
<td>4.0 (2.9)</td>
<td>0.016</td>
</tr>
<tr>
<td>Peri-traumatic Distress (PDI)</td>
<td>21.9 (11.4)</td>
<td>21.4 (10.3)</td>
<td>0.591</td>
</tr>
<tr>
<td>Pain catastrophizing (PCS)</td>
<td>13.2 (12.6)</td>
<td>11.8 (11.9)</td>
<td>0.156</td>
</tr>
<tr>
<td>Optimism (LOTR)</td>
<td>8.5 (2.3)</td>
<td>9.1 (1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Physical recovery (days)</td>
<td>30.2 (73.5)</td>
<td>14.5 (42.6)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Emotional recovery (days)</td>
<td>56.3 (117.5)</td>
<td>23.4 (64.5)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as mean (SD). PDI = peri-traumatic distress inventory. PCS = pain catastrophizing scale. LOTR = Life orientation test-revised. *Participant estimate of time need to recover.
Categorical data are displayed as proportions (%) and 95% confidence intervals unless otherwise specified.* Represents significant difference in the proportion of participants reporting moderate to severe pain in that body region between cohorts (P<0.05).