Investigating Key Predictors of Persistent Low Back Pain: A Focus on Psychological Stress

Katherine M. Bernier Carney, PhD, RN[a,*,1], **Jessica W. Guite, PhD**[b], **Erin E. Young, PhD**[a,2], **Angela R. Starkweather, PhD, ACNP-BC, FAAN**[a,b]

[a]School of Nursing, University of Connecticut, 231 Glenbrook Road, Unit 4026, Storrs, Connecticut 06269-4026

[b]Center for Advancement in Managing Pain, School of Nursing, University of Connecticut, 231 Glenbrook Road, Unit 4026, School of Nursing, University of Connecticut, Storrs, Connecticut 06269-4026

Abstract

Aim: To investigate the degree to which psychological stress, self-reported pain scores, and pain sensitivity during an acute state of low back pain (LBP) predict the development of persistent LBP trajectories.

Background: Identifying which factors influence LBP trajectories is critical to understand why some individuals experience persistent LBP and to illuminate areas for nursing intervention.

Methods: A secondary data analysis of a prospective study examining trajectories of LBP was conducted. The sample was comprised of 217 adults with acute-onset LBP recruited from the community and followed over 24 weeks. Variables of interest included demographic data, perceived stress scores, self-reported pain scores, and somatosensory characteristics collected within the first 4 weeks of LBP onset. The data were analyzed using non-parametric bivariate comparisons and a semi-parametric Cox proportional hazards model with interval-censoring.

Results: Individuals with higher psychological stress scores were less likely to experience pain resolution (Hazard ratio [HR] = 0.555, 95% confidence interval [CI] = 0.36–0.85, p = 0.02). After

*Corresponding author: Katherine M. Bernier Carney, University of Utah, College of Nursing, 10 South 2000 East, Salt Lake City, Utah 84112; katherine.carney@utah.edu.

adjustment for covariates in the final model, analysis revealed household income (HR = 2.79, 95% CI [1.63–4.67], p < 0.001) to be the dominant predictor of LBP persistence in this sample.

Conclusion: Heightened psychological stress and pain severity as well as decreased pressure pain thresholds were indicated as influential factors of LBP trajectories, although household income was identified as the dominant predictor. Strategies which integrate assessment of stress, self-reported pain scores, pain sensitivity, and social determinants for patients experiencing pain are needed to advance nursing care.

Keywords

Low back pain; chronic pain; psychological stress; social determinants of health

Background

Non-specific persistent low back pain (pLBP) has garnered frequent attention in the last few decades as a top cause of mobility limitation in adults worldwide (GBD 2017 Disease and Injury Incidence and Prevalence Collaborators, 2018). In the United States (US), pLBP is estimated to effect over 13% of the population at any given time, with an estimated annual financial burden of $30.3 billion as of 2010 (Gaskin & Richard, 2012; Shmagel, Foley, & Ibrahim, 2016). pLBP, defined as idiopathic low back pain lasting more than three months, is included in the recently designated "primary pain syndrome" category of the International Classification of Diseases Version 11 (Treede et al., 2019). Primary pain syndromes are conditions in which pain is the disease rather than a symptom of another ongoing disease process.

Various biopsychosocial processes contribute to underlying mechanisms which induce pain persistence (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Previous research has pointed to stress, depression, and maladaptive coping behaviors as contributing factors to the persistence of chronic pain (Allegri et al., 2016; Lunde & Seiberg, 2020; Mun et al., 2019; Meints & Edwards, 2018). These cognitive and emotional processes may help to drive and maintain ongoing pain states by mediating pain responses and outcomes, including pain severity and sensitivity (Edwards, Dworkin, Sullivan, Turk, & Wasan, 2016; Meints & Edwards, 2018).

Deconstructing the relationship between psychological stress and the development of pain persistence may shed light on the mechanisms that are engaged in difficult to treat primary pain syndromes. It has been speculated that chronic stress is a mediating factor in persistent pain states which contributes to pathophysiological modifications in nociceptive pathways leading to a heightened pain sensitivity and response to noxious stimuli (i.e. hyperalgesia) (Jennings, Okine, Roche, & Finn, 2014). Moreover, the size and direction of stress effects on chronic pain has been proposed to be heavily influenced by individual differences in the physiological stress response (Vachon-Presseau, 2018). Changes in emotional regulation can promote increases in sensitivity of the central and peripheral neurons, resulting in localized pain at the site of the noxious stimulus as well as at distal sites (Egloff, Hirschi, & von Kanel, 2013; Imbe, Iwai-Liao, & Senba, 2006; Lunde & Seiberg, 2020; Jennings et al., 2014). Widespread changes in peripheral and central pain pathways have been documented

through the use of quantitative sensory testing (QST) and functional magnetic resonance imaging, showing that individuals with pLBP experience increased pain sensitivity and hyperactivity of neuronal signaling (Starkweather et al., 2016b; Starkweather et al., 2016c; Marcuzzi, Wrigley, Dean, Graham, & Hush, 2018). However, the direct influence of stress, pain severity, and pain sensitivity as mechanistic factors in developing pain trajectories prior to a chronic pain diagnosis is not well established (Lunde & Seiberg, 2020). A recent review suggested that stressful events in childhood may contribute to the development of pLBP in adulthood, although limitations related to the rigor of the reviewed primary studies contributed to challenges in identifying definitive associations (Buscemi, Chang, Liston, McAuley, & Schabrun, 2019). Further investigations outlining the relationship between psychological stress and LBP trajectories are warranted.

Stress is particularly relevant to nursing as it represents a potentially modifiable factor that can be assessed throughout all levels of clinical care and for which interventions could happen in clinical settings or the community. Understanding how stress influences an individual's pain trajectory will assist nurses to identify key risk factors at pain onset and develop appropriately targeted interventions. Thus, the objectives of this study were twofold: (1) to explore the degree to which increased psychological stress, self-reported pain scores, and pain sensitivity (primary risk factors) during a phase of acute LBP are independently predictive of pain trajectories over time; and (2) to identify if these primary risk factors, taken together at baseline, present a model predicting the trajectory of low back pain. Our hypothesis was that individuals with heightened psychological stress scores, self-reported pain scores, and pain sensitivity at baseline would be less likely to experience pain resolution.

Methods

Design and Sample

This study presents a secondary analysis of an existing data set which measured prospective changes in pain characteristics, somatosensory data, and genetic factors of individuals during the transition from acute to chronic LBP over time (Starkweather et al., 2016b; Starkweather et al., 2016c). Briefly, 220 English-speaking adults aged 18–50 with acute onset low back pain (>24 hours and <4 weeks, preceded by >1 pain-free month) were recruited through informational flyers placed around three university campuses, primary care clinics, and in the general public, and consented to enroll. Data were collected every six weeks for up to 24 weeks after the baseline visit if the subjects continued to meet study criteria for ongoing LBP (>2 out of 10 on numeric rating scale [NRS]). Subjects were considered to have resolved pain if they rated their LBP as < 2 at any of the six week follow-up visits, which would have triggered the end of study participation based on the study protocol (n=79). Subjects were considered to have persistent unresolved pain if they continued to experience pLBP at the 24-week visit (n=55). Subjects who continued to have pain, but did not attend follow-up appointments or were not able to be contacted for study continuation (i.e., attrition), were considered undefined unresolved (n=83). After the consenting procedure, three subjects were identified to be ineligible based on study parameters and were removed from further study participation, leaving 217 subjects for this

analysis. Institutional Review Board approval was attained for the primary study as well as this secondary, ongoing analysis.

Measures

After enrollment in the primary study, all participants completed an initial visit in which demographics, LBP history, self-report measures (pain characteristics; mood state; coping strategies; reactivity; psychological stress; disability status; work history), and somatosensory data were collected. To meet this study's objectives, the self-report measures included in this secondary data analysis were the Brief Pain Inventory-Short Form (BPI; worst pain, least pain, average pain, pain now, and pain interference) and the Perceived Stress Scale (PSS). The five BPI items assess pain severity and activity interference using 11-point, Likert-type scales with lower ratings indicating less pain severity or interference, and have been previously validated in chronic pain populations (Cleeland & Ryan, 1994; Keller et al., 2004). The PSS is a 10-item, self-report questionnaire that measures psychological stress (Cohen, Kamarck, & Mermelstein, 1983) with an observed Cronbach's a = .92 reported in the parent study (Starkweather et al., 2016b). Scores for the PSS range from 0 to 40, with lower PSS scores indicating lower levels of psychological stress. Average values of the PSS in the general population of the US from 2009 have been reported as a mean of 15.52 for men and mean of 16.14 for women (Cohen & Janicki-Deverts, 2012).

Quantitative sensory testing (QST) was used to measure peripheral and central somatosensory changes over time, which assists in characterizing functional changes in sensory nerves (Belfer, & Dai, 2010). Understanding these changes during the course of developing pLBP informs an objective assessment of pain trajectories. In the parent study, a standardized protocol collected QST data on the dorsum of the non-dominant hand (control site) and three lumbar sites (painful site) (Starkweather et al., 2016a). In this secondary analysis, we included the QST measures of mechanical pain sensitivity (MPS), dynamic mechanical allodynia (ALL), cold pain threshold (CPT), heat pain threshold (HPT), and pressure pain threshold (PPT), as these measures have previously been shown to differentiate pain sensitization in subjects with pLBP (Starkweather et al., 2016c; Marcuzzi et al., 2018).

Analytic Strategy

Statistical analyses were all conducted using SPSS v25 and RStudio v1.1.453 software. We used descriptive statistics for categorical demographic variables. We computed the median and interquartile range for continuous demographic characteristics and primary study variables. A level of significance (a) of 0.05 was established *a priori* for all statistical testing.

In order to address the first aim of identifying if higher levels of psychological stress, self-reported pain scores, and pain sensitivity were independently predictive factors of pain trajectories, we created survival plots using a non-parametric maximum likelihood estimator (NPMLE) developed for use with interval censored data (Anderson-Bergman, 2017). The technique of interval censoring was implemented because the exact day of pain resolution (<2 on NRS) was not collected in the primary study that would allow for the creation of a continuous pain duration variable (Zhang & Sun, 2009). We established the visit number at which pain resolution was identified as the event, or hazard, in this scenario. Subjects that

were lost to follow-up were also included in the analysis using the technique of right censoring, with the second time-point established as ∞ (Anderson-Bergman, 2017; Zhang & Sun, 2009).

Confounding demographic variables included in the analyses were age, sex, race, ethnicity, body mass index, income, and smoking (De Palma, Ketchum, & Saullo, 2012; Maly, & Vallerand, 2018; Shi, Weingarten, Mantilla, Hooten, & Warner, 2010; Shmagel et al., 2016). We divided all continuous variables into "high" and "low" groups based on the median. We collapsed race into 3 groups (White/Caucasian, Black/African American, and other/more than one race) based on frequencies reported in the parent study. All other demographic variables were categorically split into two groups based on the measure (e.g., smoker vs. non-smoker). The NPMLE plots allowed us to visualize if the groups differed in the probability of pain resolution over the 24-week study.

To meet the first aim, we also examined the statistical differences between groups visualized in the NPMLE plots by conducting bivariate analyses using a semi-parametric regression modeling procedure for interval-censored data, as established by Anderson-Bergman (2017) for R software. We used bootstrap sampling (n = 100) to adjust for bias. All variables were evaluated to ensure they met the proportional hazards assumption prior to fitting a proportional hazards model. If this assumption was not met, then a proportional odds ratio was used instead (Anderson-Bergman, 2017). A ratio less than one indicates that the risk factor had a decreased likelihood of pain resolution, while a ratio greater than one denotes that the risk factor had an increased likelihood of pain resolution (Bradburn, Clark, Love, & Altman, 2003).

To address the second aim of this study - identifying if perceived stress, pain score, and pain sensitivity together predicted LBP trajectories - we created a Cox proportional hazards regression model using a multistep selection process. First, all variables, including confounding factors, which had significant findings in the initial NPMLE bivariate analyses were included in order to build a preliminary model using multivariate analysis. Once the dominant predictor variable was identified in this model, we completed further analysis using the proportional hazards procedure to determine possible covariates to include in the final model. A variable was considered to be a covariate if it changed the hazard ratio of the dominant variable by greater than 25% in comparison to the model without the target variable included. This process was repeated until all potential covariates (i.e., age, race, smoking, PSS, BPI, QST variables) were considered and the final model was set. Lastly, correlation coefficients were calculated to examine associations between the dominant predictor variable and alternative factors.

Results

Participants Characteristics

In the total sample, the majority (51.2%) of subjects identified as male with a median age of 26 years old. Most participants had completed some post-secondary education and 71% of subjects had a household income of less than $60,000 per year. Although the majority of subjects (73.7%) of subjects had a previous history of low back pain, 74.2% expected their

current pain to resolve after the baseline visit. Overall, most participants (76.5%) considered themselves to be in good or excellent health. Table 1 displays descriptive statistics of the demographics and pain related characteristics at the initial study visit.

The Influence of Individual Risk Factors on Pain Trajectories

The results from the bivariate and multivariate analyses are shown in Table 2. In the bivariate analyses, psychological stress was found to be a significant risk factor as individuals with a self-reported PSS score of greater than 17 were 44.5% less likely (Hazard ratio [HR] = 0.555, 95% Confidence Interval [CI] [0.36 – 0.85], p = 0.02) to experience pain resolution. Additionally, as reported in Table 2, all of the pain scores reported in the BPI were significantly associated with longer pain trajectories, each indicating that reporting higher pain at the initial visit was associated with decreased likelihood of pain resolution. PPT was the only QST variable with a significant finding between groups, with higher pain thresholds at the painful (low back) site (HR = 1.885, 95% CI [1.14 – 3.11], p = 0.01) and at the control (dominant arm) site (HR = 2.159, 95% CI [1.31 – 3.35], p = 0.001) demonstrating a greater likelihood of pain resolution. Figure 1 displays examples of the NPLME survival plots created for PSS, the current pain severity score taken at the baseline study visit (BPI now), and PPT for the painful LBP site.

Multiple confounding factors were also found to influence pain trajectories. The results indicate that being older than 26 (HR = 0.448, 95% CI [0.26 – 0.74], p = 0.006), having a BMI greater than 26.5 (HR = 0.584, 95% CI [0.34 – 0.99], p = 0.02), or identifying as a current smoker (HR = 0.301, 95% CI [0.14 – 0.64], p = 0.001) were each associated with decreased likelihood of pain resolution. In contrast, identifying as White/Caucasian (HR = 5.413, 95% CI [2.59 – 11.27], p <0.001) or another race (HR = 4.577, 95% CI [2.01 – 10.41], p <0.001), compared to identifying as Black/African American, significantly increased the likelihood of pain resolution. Additionally, subjects who reported having a household income greater than $60K per year were over 3 times more likely (p <0.001) to experience pain resolution.

The Influence of Multiple Primary Risk Factors on Pain Trajectories

When all variables with significant findings from the bivariate analysis were included in a Cox proportional hazards regression model, neither psychological stress, individual self-reported pain scores, nor pain sensitivity factors were indicated as dominant predictors of LBP trajectories. Alternatively, the preliminary model revealed household income to be the dominant predictor variable in this sample, as noted in Table 2. The covariates that changed the HR of household income greater than 25% included psychological stress, current pain severity score at baseline visit (BPI now), PPT at the control site, and age. After adjusting for covariates in the final model, the hazard ratio for household income was 2.79 (95% CI [1.63–4.67], p < 0.001).

Correlation results show that household income was significantly and negatively associated with worst pain score (BPI worst) (ϕ = -0.018, p = 0.007) and pain interference (BPI interference) (ϕ = -0.181, p = 0.01). Household income also demonstrated a weak positive correlation with PPT at the painful site (ϕ = 0.19, p < 0.01) and PPT at the control site (ϕ =

0.123, p < 0.01). Additionally, two confounding factors were correlated with household income, African American race (r = -0.422, p < 0.001) and smoking status (ϕ = -0.297, p < 0.001).

Discussion

The purpose of this study was to investigate if increased levels of psychological stress, self-reported pain, and pain sensitivity during an acute state of LBP were predictive of pLBP. Our findings demonstrate that, although these factors influenced the pain trajectories, household income was the dominant predictor in this sample. The final model suggests that subjects who had an annual household income greater than or equal to $60,000 and whose age was under 26 years with lower stress scores, lower reported pain severity, and higher pressure pain thresholds at the time of the baseline visit were most likely to experience resolution of their pain. These findings align with previous studies of individuals experiencing chronic pain conditions, including pLBP (Janevic, McLaughlin, Heapy, Thacker, & Piette, 2017; Mun et al., 2019; Shmagel et al., 2016).

The primary risk factors interest, baseline psychological stress and self-reported pain along with PPT, were individually indicated in the bivariate analyses as influencing factors on the LBP trajectories. Additionally, in the multivariate analysis, these three factors were also found to be key to characterizing LBP trajectories in this sample. However, the hypothesized grouping of these risk factors was not substantiated when accounting for the confounding factors in the final model. The results from this analysis indicated that external and non-modifiable factors primarily influenced the development of pLBP. These findings support the biopsychosocial model of pain (Gatchel et al., 2007), demonstrating the importance of examining biological, psychological, and social factors when accounting for mechanisms contributing to pLBP.

As psychological stress was identified as a covariate in the final model, the present findings suggest that household income is closely associated with perceived stress and may explain the relationship between perceived stress and pain persistence in the bivariate model. Poor adaptation to chronic stress can increase allostatic load, the impact of which is seen can be seen in associations between low socioeconomic status and increased rates of chronic disease, including persistent pain conditions (Buscemi et al., 2019; Fliesser, De Witt Huberts, & Wippert, 2017; McEwen, & Gianaros, 2010; Maly & Vallerand, 2018; Vachon-Presseau et al., 2013). It is also possible the PSS measure captured a sense of financial insecurity in the framework of general or chronic stress. This finding is in agreement with prior evidence using the PSS, describing increased levels of reported stress for individuals with lower income (Cohen & Janicki-Deverts, 2012). Investigations of psychological stress as a mechanism in developing pain trajectories continues to be an important area of research (Lunde & Seiberg, 2020).

Subjective pain severity and interference scores and one measure of pain sensitivity (PPT) were also included in the final model as covariates. It is interesting that the pain severity captured at the time of the baseline visit (BPI now) was the strongest covariate out of all subjective pain scores in this analysis. Bias should be considered since subjects were

participating in a study that pulls attention to the experience of pain when considering these findings. Contrary to our study, a large systematic review found that baseline pain was not strongly predictive of chronic pain trajectories (Chou & Shekelle, 2010).

Differential baseline PPTs were also found in subjects who experienced pain persistence and confirms previous findings (Starkweather et al., 2016c; Marcuzzi et al., 2018; Yildiz et al., 2017). An enhanced pressure pain sensitivity at the control (LBP) site could indicate early central sensitization in pain pathways (Woolf, 2011). As the biological mechanisms of persistent stress are closely related to the biological mechanisms of pain, examining the mechanisms of stress as they relate to those of chronic pain continues to be an important consideration for future studies and nursing practice (Abdallah, & Geha, 2017; McEwen, 2013; Vachon-Presseau et al., 2013; Vachon-Presseau, 2018).

Taken together, these results suggest that psychological stress, pain severity, and pain sensitivity during acute LBP are influential factors in pLBP trajectories, supporting previous work on stress and pain trajectories (Allegri et al., 2016; Jennings et al., 2014; Mun et al., 2019; Vachon-Presseau, 2018). Moreover, the findings from this analysis add to the ongoing evidence that social factors affect pain outcomes and persistence (Janevic et al., 2017; Maly & Vallerand, 2018). These results reinforce the critical need for an improved recognition of personal and social factors in pain-related assessments. Building nursing practice standards which include evaluations of these factors will help bring light to how situational contexts and illness experiences can influence the biopsychosocial patterns of disease, including pLBP. Assessing an individual's perception of stress will further help nurses to advance integration of social contexts into illness and pain care.

Study Limitations

As described in the methods, over 30% of the subjects included in the analysis did not complete follow-up visits and, therefore, their low back pain trajectory remains undetermined. Although we attempted to account for this by applying the methods of right censoring and a bootstrapping technique, bias still may have been introduced. Additionally, we only included variables in the model that were found to be significant in the bivariate analysis. Due to this approach, the final model did not include sex or BMI, which have both been shown to impact pLBP outcomes (De Palma et al., 2012; Larsson, Björk, Börsbo, & Gerdle, 2012). Examining perceived stress as a baseline predictive factor does not account for changes in levels of stress as pain continues or distinguish if the level of stress measured correlates with ongoing pain changes, which limits our ability to make conclusions regarding long-term associations between psychological stress and chronic pain. Lastly, alternative variables were included in the parent study that should be considered in subsequent investigations, including coping strategies, pain-related disability, and mood state.

Future Directions

Our findings support prior evidence that multiple factors play a major role in the development of persistent pain states. This study points to the need for the thorough evaluation of social and clinical factors during pain care consultations and the increased

recognition of how pain-related health care disparities may evolve. Alone, nurses cannot identify and address every factor which impacts a painful experience or limits access to holistic pain care. Therefore, the focus should concentrate on collaborating with other team members that have an active role in caring for a patient with pain, including physicians, psychologists, social workers, and family members. Through their relationships with patients, nurses can be leaders in helping to drive these teams and the identification of key factors that may contribute to persistent pain conditions. Interdisciplinary team collaboration and the use of social support strategies that integrate assessment of pain, stress and social determinants for patients experiencing pain will help to advance nursing practice. Future research that focuses on the impact that nurse led social support strategies have on changing pain trajectories can provide further evidence of the value of advancing this practice model.

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Highlights

- Baseline stress, self-reported pain, and pain sensitivity impact LBP trajectories

- Higher household income increased the likelihood of pain resolution over 24 weeks

- Income's relation to LBP course influenced by baseline stress, pain and sensitivity

- Consistent evaluation of biopsychosocial factors of pain could advance nursing care
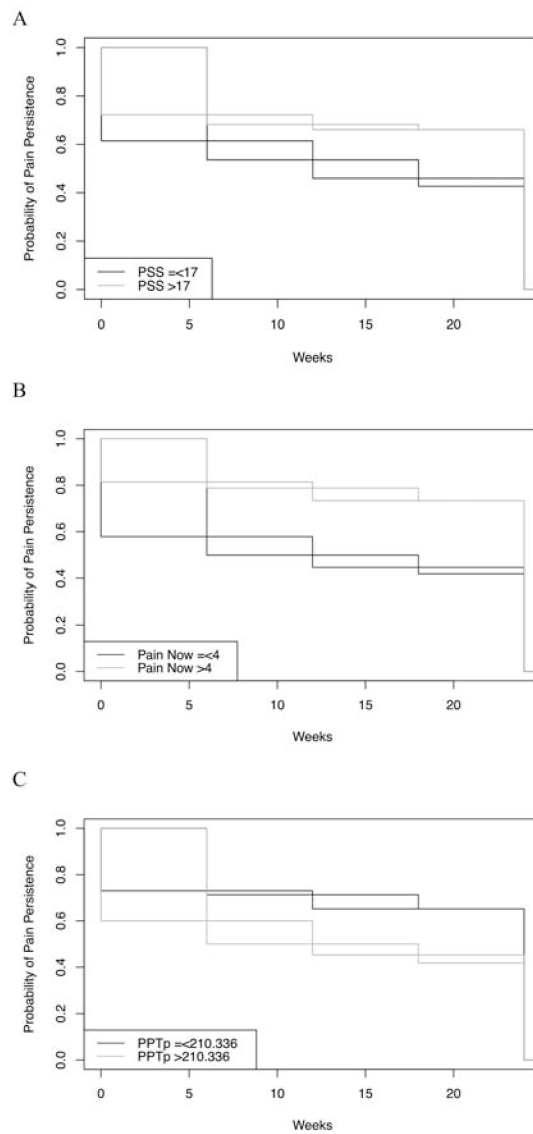
Figure 1 – .
Example Plots of Survival Analysis for Three Individual Risk Factors. *Note*. Panel A: Non-parametric maximum likelihood estimator (NPMLE) plot for Perceived Stress Scale (PSS) scores, p = 0.02. Panel B: NPMLE plot for Brief Pain Inventory (BPI) score at time of baseline study visit, p < 0.001. Panel C: NPMLE plot for pressure pain threshold (PPT) at painful back site, p = 0.01.

Table 1

Participant Demographic and Pain Characteristics (n = 217)

Characteristic	n	%	Med	IQR
Age			26	19
Sex, Male	111	51.2		
Race				
Caucasian	88	40.6		
Black/African American	90	41.5		
More than one race, Other	39	17.9		
Ethnicity				
Hispanic/Latino	16	7.4		
Household Income				
<$60K per year	154	71		
$60K per year	63	29		
BMI			26.5	7.94
Current smoker	63	29		
Prior history of LBP	160	73.7		
Expected pain to resolve	161	74.2		
Perceived Stress Scale (0–40)			17	7.39
Brief Pain Inventory, (NRS 0–10)				
Worst			6	3
Least			2	3
Average			4	3
Now			4	4
Interference			3.14	3.9
QST, Painful site				
MPS, rating 0–10			2.66	3.67
ALL, rating 0–10			0.33	1.83
CPT, °C			21.86	13.55
HPT, °C			39.53	5.30
PPT, kPa			210.36	211.03
QST, Control site				
MPS, rating 0–10			1.33	3
ALL, rating 0–10			0	0.92
CPT, °C			19.83	14.93
HPT, °C			40.63	5.78
PPT, kPa			213.63	147.33

Note. n = Number, count; Med = Median; IQR = Interquartile Range; BMI =Body Mass Index; LBP = Low Back Pain; NRS = Numeric Rating Scale; QST = Quantitative Sensory Testing; MPS = Mechanical Pain Sensitivity; ALL = Dynamic Mechanical Allodynia; CPT = Cold Pain Threshold; HPT = Heat Pain Threshold; PPT = Pressure Pain Threshold; C = Celsius; kPa = Kilopascal.

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Table 2

Hazard Ratios to Pain Resolution

Variable	Bivariate Analysis			Multivariate Analysis[a]		
	PE	HR [95% CI]	P-value	PE	HR [95% CI]	P-value
Primary Risk Factor						
PSS (0–40)						
>17	−0.5878	0.555 [0.36 – 0.85]	0.02*	−0.2658	0.766 [0.41 – 1.42]	0.39
BPI (NRS 0–10)						
Worst, >6	−1.068	0.343 [0.19 – 0.6]	<0.001*	−0.5426	0.581 [0.25 – 1.33]	0.2
Least, >2	−1.018	0.361 [0.21 – 0.61]	<0.001*	−0.3307	0.718 [0.27 – 1.85]	0.49
Average, >4	−0.5217	0.593 [0.36 – 0.96]	0.01*	0.4726	1.604 [0.68 – 3.74]	0.27
Now, >4	−1.015	0.362 [0.19 – 0.65]	<0.001*	−0.5512	0.576 [0.2 – 1.58]	0.28
Interference, >3.14	−0.6886	0.502 [0.3 – 0.83]	0.007*	0.16120	1.175 [0.63 – 2.16]	0.6
QST, painful site						
MPS, NRS >2.66	−0.4382	0.645 [0.4 – 1.01]	0.06			
ALL, NRS 0–10	−0.3315	0.717 [0.43 – 1.17]	0.18			
CPT, >21.86°C	−0.3976	0.671 [0.42 – 1.05]	0.08			
HPT, >39.53°C	0.2009	1.222 [0.76 – 1.95]	0.41			
PPT, >210.336 kPa	0.6337	1.885 [1.14 – 3.11]	0.01*	−0.07417	0.384 [0.43 – 1.97]	0.84
QST, control site						
MPS, NRS >1.33	0.1122[a]	1.119 [0.59 – 2.09]	0.72			
ALL, NRS >0.1	−0.2598	0.771 [0.5 – 1.18]	0.23			
CPT, >19.83°C	−0.2101	0.810 [0.53 – 1.23]	0.32			
HPT, >40.63 °C	0.4021	1.495 [0.93 – 2.39]	0.09			
PPT, >213.63 kPa	0.7695	2.159 [1.31 – 3.35]	0.001*	0.4545	1.575 [0.65 – 3.77]	0.307
Confounding Factor						
Sex[b]						
Male	−0.166	0.846 [0.45 – 1.58]	0.56			
Age						

Variable	Bivariate Analysis			Multivariate Analysis[a]		
	PE	HR [95% CI]	P-value	PE	HR [95% CI]	P-value
>26 years old	−0.802	0.448 [0.26 − 0.74]	0.006[*]	−0.19560	0.822 [0.36 − 1.87]	0.64
Race[c]						
More than one race,	1.521	4.577 [2.01 − 10.41]	<0.001[*]	0.7074	2.029 [0.48 − 8.42]	0.33
White/Caucasian Other	1.689	5.413 [2.59 − 11.27]	<0.001[*]	0.9005	2.461 [0.76 − 7.93]	0.13
Ethnicity[b]						
Non-Hispanic/Latino	−0.02409	0.976 [0.27 − 3.46]	0.97			
Household Income						
$60K per year	1.101	3.008 [1.88 − 4.79]	< 0.001[*]	0.7532	2.124 [1.06 − 4.24]	0.03[*]
BMI						
>26.5	−0.5367	0.584 [0.34 − 0.99]	0.02[*]			
Smoking						
Current smoker	−1.198	0.301 [0.14 − 0.64]	0.001[*]	−0.1194	0.887 [0.31 − 2.51]	0.82

Note. Variable values split categorically or by median value in the sample. PE = Parameter estimate; HR = Hazard ratio; CI = Confidence interval; PSS = Perceived Stress Scale; BPI = Brief Pain Inventory; NRS = Numeric rating scale; QST = Quantitative sensory testing; MPS = Mechanical pain sensitivity; ALL = Dynamic mechanical allodynia; CPT = Cold pain threshold; C = Celsius; HPT = Heat pain threshold; PPT = Pressure pain threshold; kPa = Kilopascal; BMI = Body mass index.

[*] Level of significance set at α = 0.05

[a] Hazard ratios shown for preliminary Cox regression model, prior to identifying covariates.

[b] Proportional odds model used.

[c] Compared to identifying as Black/African American.