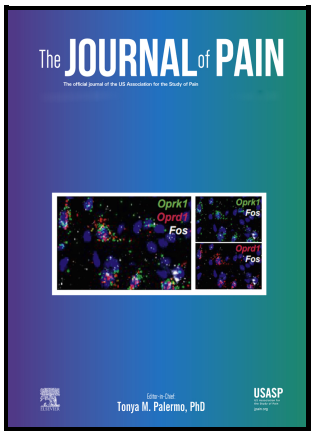

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Adverse Childhood Experiences and Chronic Low Back Pain In Adulthood: The Role of Emotion Regulation

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Abstract

Chronic low back pain (cLBP) is characterized by biopsychosocial determinants that collectively result in substantial burden at the individual, community, and healthcare system levels. A growing body of literature suggests that childhood adversity is longitudinally associated with the development and maintenance of various chronic pain conditions in adulthood. Little research has investigated the psychological processes that might underlie the association between adverse

childhood experiences (ACEs) and cLBP. Emotion regulation comprises a substantive part of the subjective experience of pain and may be a potential mechanism through which ACEs contribute to cLBP etiology and maintenance. Thus, the current study examined the extent to which emotion dysregulation mediated the relationship between ACEs and pain severity (pain at rest and movement-evoked pain) in adults with cLBP. Participants included 183 adults (53.0% female, 62.5% non-Hispanic Black) between the ages of 18 and 85 with cLBP. Participants self-reported on ACEs, pain, difficulties in emotion regulation, depression, and completed brief physical function tasks. In data analytic models, sociodemographic variables were included as covariates. Mediation analyses revealed that emotion regulation mediated the relationship between ACEs and cLBP severity at rest (indirect effect = 0.15 (95% CI [0.06 to 0.25]) and with movement (indirect effect = 1.50 (95% CI [0.69 to 2.57]). Findings suggest ACEs are linked to cLBP severity in adulthood though difficulties in emotion regulation. This aligns with research demonstrating that childhood maltreatment can lead to difficulties in emotion regulation which perpetuate over the lifespan to impact adult health outcomes.

Trial Registration: This study utilized baseline data collected as part of a parent trial titled "Examining Racial and SocioEconomic Disparities in Chronic Low Back Pain" (ERASED - ClinicalTrials.gov ID: NCT03338192).

Perspective: This study presents emotion dysregulation as a psychological pathway through which childhood adversity may contribute to chronic low back pain in adulthood. This work may bolster our understanding of social experiences as risk factors for chronic pain, while identifying targets for clinical intervention.

Keywords: emotion regulation; adverse childhood experiences; chronic low back pain; social experiences; lifespan

Introduction

Chronic low back pain (cLBP) is characterized by physiological, psychological, and social dimensions that together result in substantial burden at the individual, community, and healthcare system levels [47]. cLBP contributes to limited physical activity, social isolation, psychopathology,

reduced work productivity, and heavy healthcare utilization [23; 50]. In fact, cLBP is the leading cause of physician visits in the United States and the second leading cause of disability worldwide [27; 84]. Though previous studies have shown social indicators of health (SDH) to shape the distribution of behavioral risk factors, comorbid conditions, and exposure to environmental risks, this research remains understudied in non-specific cLBP [13; 34; 39; 58; 61]. The majority of cLBP is classified as "non-specific", due to an absence of discernible pathological abnormalities in the spine or nearby tissues [68]. Furthermore, in cases where pathophysiological changes are observed, there are frequent discrepancies between the radiographic images and the severity or manifestation of symptoms [8]. These observations underscore the necessity for research exploring factors, beyond pathobiology, that contribute to cLBP.

Adverse childhood experiences (ACEs) are SDH consisting of both direct and indirect adverse events such as emotional, physical, sexual abuse, parental psychopathology, substance abuse, early parental loss due to death/abandonment, parental incarceration, or conflict [12]. Though findings from several studies suggest there is an association between ACEs and chronic pain, the evidence regarding the magnitude and direction of this relationship has been mixed [16; 22]. Moreover, there has been little to no research investigating the psychological processes, such as emotion regulation, that might underlie this relationship.

Emotion regulation refers to the cognitive and behavioral strategies used to modulate the expression, frequency, and nature of emotions during stressful and non-stressful circumstances [32]. Childhood maltreatment is known to hamper development and acquisition of critical emotion regulation skills [33; 45; 51; 52]. Emotion regulation comprises a major part of the subjective experience of pain, and may be a route through which ACEs contribute to cLBP [1; 42; 72]. Furthermore, difficulties in emotion regulation have been established as a transdiagnostic risk and maintenance

factor underlying many health conditions, including cLBP [42]. Finally, understanding the relationship between emotion regulation and chronic pain underscores the importance of comprehensive assessment of the pain experience.

Clinical studies examining psychological features (e.g., emotional regulation and ACEs) in the context of pain have focused almost exclusively on pain severity at rest (PAR). There is growing recognition of movement-evoked pain (MEP) as an alternative assessment model, which encompasses the investigation of sensory, motor, and psychological factors influencing pain[19]. Importantly, the fear-avoidance model highlights how maladaptive cognitive frameworks often lead to pain-related fear and, ultimately, movement avoidance. Over time, the avoidance of movement leads to increased pain due to atrophy which exacerbates this cycle [71]. Thus, the assessment of MEP may capture more psychological distress relevant for emotion regulation compared to PAR in individuals with cLBP [44].

Taken together, childhood adversity may increase the risk of developing and worsening cLBP by hampering the development and acquisition of critical emotion regulation skills. It is also likely that MEP as opposed to PAR is more efficient at evoking psycho-emotional mechanisms that contribute to the pain experience for this clinical population. Thus, the primary objective of this study was to examine the extent to which emotion regulation mediates the relationship between ACEs and cLBP severity (PAR and MEP). In our investigation, we sought to assess the significance of the hypothesized mediation while adjusting for potentially confounding factors known to influence both cLBP and emotion regulation including age, sex, race, body mass index (BMI), cLBP duration, medication use, and depressive symptoms.

Methods

Study Overview

The present study is part of an ongoing parent project that employs a biopsychosocial model to investigate factors that contribute to racial and socioeconomic differences in cLBP severity and disability (NCT03338192 - Examining Racial and SocioEconomic Disparities in cLBP [ERASED]). The participants described in the current analysis were recruited between November 2017 and December 2021. Findings from the present study were presented in poster format at the 2022 Annual Scientific Meeting of the United States Association for the Study of Pain [79] . Figure 1 depicts a flow diagram illustrating matriculation through the current study. All potential participants underwent a telephone and electronic medical record screening to determine eligibility. Health and cLBP history were reviewed to verify continued participation. Eligible individuals underwent the informed consent processes prior to participating in the study. Enrolled participants completed two experimental study sessions (separated by one week). During the first session, they provided demographic information and completed psychosocial and clinical pain assessments. Participants then returned for their second study session one week later and completed measures of MEP and physical function. Study procedures followed the cLBP research standards outlined by the Research Task Force of the National Institutes of Health Pain Consortium[23] The data collected as part of this proposal and the larger ERASED study were approved and conducted in compliance with the University of Alabama at Birmingham (UAB) Institutional Review Board (IRB). In alignment with efforts to enhance quality, transparency, and consistency of patient and public involvement (PPI) in research design, we acknowledge that there was no PPI in the conceptualization, design, conduct, or dissemination of this secondary data analysis [74].

[FIGURE 1 HERE]

Participants

Participant data were collected in accordance with the research standards outlined for chronic low back pain [23]. Participants were recruited using study flyers posted within a UAB pain treatment clinic and the surrounding Birmingham community. The study eligibility criteria required participants to (1) have persistent, non-specific chronic low back pain (cLBP) for at least 3 months and pain occurring on at least half the days in the past 6 months. (2) Participants had to be between 19 and 85 years old (this range was chosen to capture young adults with cLBP and excluded individuals increasingly likely to meet one or more exclusion criteria). (3) Participants also had to identify as either Non-Hispanic Black or Non-Hispanic White. A stratified sampling approach (incorporating racialized identity and reported household income) was used to ensure balanced representation and recruitment of equivalent groups based on race and SES. In this study, 186 individuals were consented and 183 participants with nonspecific cLBP were included in the final sample.

Initial Screening and Review of Medical Records

All participants completed a telephone-based screening consisting of a health history and medical record review to confirm cLBP diagnosis and eligibility for this research. Individuals were excluded if they endorsed cLBP attributable to specific factors (e.g. ankylosing spondylitis, infection, malignancy, compression fracture or other trauma), systemic rheumatic disease, or any chronic pain condition reported as more prominent or severe than the cLBP. Additionally, exclusion criteria included a history of significant low back surgery in the past year, current cancer diagnosis or history of cancer involving chemotherapy/radiation, uncontrolled hypertension (SBP/DBP of > 150/95), cardiovascular or peripheral arterial disease, poorly controlled diabetes

(HbA1c > 7%), history of stroke or seizures, circulatory disorders, human immunodeficiency virus, neurological disease, and serious psychiatric disorder requiring hospitalization in the past 12 months. Pregnancy was also considered as an exclusion criterion.**These conditions affect both participant safety and potentially alter pain perception.*

Measures

Sociodemographic Variables

Data was collected through self-report questionnaires. Demographic and clinical variables of interest included: (1) age, (2) race/ethnicity, (3) sex, (4) marital status, (5) body mass index (BMI, (6), duration of pain, and (7) current pain treatments, (8) annual household income level, and (9) highest education level.

Psychosocial Measures.

Adverse childhood experiences (ACEs). The ACEs questionnaire assesses childhood exposure to physical, emotional, sexual abuse, neglect, parental divorce, maternal domestic violence, presence of substance use, psychopathology, or incarceration in the household. Higher scores indicate more experiences of childhood adversity.

Difficulties in emotion regulation (DER). The DER scale assesses modulation of arousal, awareness, understanding, and acceptance of emotions, and the ability to act in desired ways regardless of emotional state [30]. The 36-item questionnaire results in six subscales including: nonacceptance of emotional responses (e.g. "*when I'm upset, I become embarrassed for feeling that way*"), difficulty engaging in goal-directed behavior (e.g. "*when I'm upset, I have difficulty getting work done*"), impulse control difficulties (e.g. "*I experience my emotions as overwhelming and out of control*"), lack of emotional awareness (e.g. reverse-coded item: "*I am attentive to my feelings*"), limited access to emotion regulation strategies (e.g. "*when I'm upset, my emotions feel*

overwhelming"), and lack of emotional clarity (e.g. "*I am confused about how I feel*"). Higher scores indicate greater difficulty for subscales and the total score. Internal consistency for the DERs overall scale (α = .93) and subscales (αs = .82 - .89) has been found to not differ across Asian American, African American, and Caucasian demographic groups [30; 67]. This measure has previously been applied to cLBP populations [42].

Pain Measures

Pain at rest (PAR). The four-item Brief Pain Inventory – Short Form (BPI-SF) assesses pain severity, impact on daily function, pain location, and medication use. We assessed PAR by using the severity subscale which consists of four items (worst pain in the past 24 hours, least pain in the past 24 hours, average pain severity, and pain right now). Each item was scored from 0 ('no pain or does not interfere') to 10 ('worst imaginable pain or completely interferes'). Higher scores indicate greater pain severity. Previous research has established the Cronbach α coefficient to be .85 for the BPI intensity subscale using samples of chronic, non-malignant pain conditions, including cLBP [49; 73; 77].

Movement-evoked pain (MEP). Bed Task Assessment: Participants were asked to rate their pain in their lower back getting into and getting out of a bed that was 0.9 m in height. Box Lift Assessment; participants were asked to rate the pain in their lower back while lifting a weighted box (4 kg for females, and 6.3 kg for males) from the floor onto the bed, and then back to the floor. A numeric rating scale was utilized to rate pain intensity, where 0 = "no pain" and 100 = "most intense pain imaginable." The numeric average of these pain scores for the box lift and bed task was calculated to create the MEP variable. These functional tasks have been examined in previous research with cLBP populations [75].

Covariates

The following covariates were included in all the multiple regression models to examine our effects of interest more precisely. *Age.* Given epidemiological evidence indicating that the incidence and prevalence of severe and cLBP increase over the lifespan, we asked participants to self-report on their current age [83]. *Sex.* Women often report a higher prevalence of chronic pain conditions, including cLBP, as well as greater pain severity compared to men [17; 78]. We asked participants to self-report on their biological sex to account for these disparities. *Race.* Existing literature highlights racial disparities in cLBP pain severity such that Non-Hispanic Black patients experience more severe, and disabling cLBP compared to Non-Hispanic White patients. These data underscore the importance of accounting for differences in the pain experience of various racialized groups [7; 31]. Participants self-identified their own racial group as either Black or African American or White or Caucasian European. *Body mass index (BMI).* Findings indicate that higher BMI is associated with an increased risk of cLBP, possibly by contributing to the pathogenesis of the condition via excessive load bearing, increased mechanical demands, and changes to metabolic factors [69; 81]. Height and weight measurements were used to calculate BMI for each participant (weight in kilograms divided by the square of height in meters). Clinical cutoff scores for BMI were taken from the Centers for Disease Control and Prevention (Underweight: less than or equal to 18.4, Normal weight: 18.5 - 24.9, Overweight: 25.0 - 39.9, Obese: greater than or equal to 40.0) [88]. *cLBP duration.* Participants were asked "How long have you had low back pain?" and responded using the following categories: (1) Between 3 months and 6 months, (2) Between 6 months and 1 year, (3) Between 1 year and 3 years, (4) Between 3 years and 5 years, (5) Between 5 years and 10 years, (6) Between 10 years and 20 years, and (7) Greater than 20 years. *Medication use.* Participants were asked "Are you currently taking any medications (prescription or over the counter) for chronic pain or any other reason?" They responded by indicating 1 'Yes' or 0 'No".

Depressive symptoms. Depression is strongly associated with both ACEs, as well as pain intensity and degree of disability in patients with cLBP [54; 59]. However, we sought to determine if DERs would mediate associations between ACEs and cLBP severity above and beyond the contribution of depression. The Center for Epidemiological Studies-Depression (CESD) scale is a 20-item questionnaire which allows for self-report of depressive symptoms [29]. Items are rated from 0 to 3 (0 = Rarely/None of the Time, 1 = Some/Little of the Time, 2 = Moderately/Much of the time, 3 = Most/Almost All the Time). Higher scores indicating a higher number of depressive symptoms and range from 0 to 60. The CESD has a Cronbach α coefficient of 0.8 to 0.9 within community samples [24; 80].

Data Analysis

A suitable sample size for this study was determined *a priori* using G*Power, version 3.1.9.6 [26]. Parameter specifications included a desired statistical power of 0.80 or greater and a two-tailed alpha level of 0.05. Effect sizes were derived from previous literature addressing the association between ACEs and DERs ($r = 0.24$, $p < .0.001$) and the association between ACEs and back pain severity ($r = 0.22$, $p < .0.001$) [14; 60]. According to these parameters, a total sample size of 160 people with cLBP or more was appropriate to achieve sufficient (i.e., >0.80) statistical power in support of our planned analyses.

All data analysis was completed using SPSS (Statistical Package for the Social Sciences) version 29.0 [38]. Data was collected from 186 participants with cLBP. Notably, the ACEs questionnaire was added to ERASED after data collection was initiated. Individuals who were never administered the ACEs questionnaire were deleted listwise, which resulted in a final sample size of n = 183 cLBP participants. Data missingness on the ACEs variable was addressed by manual imputation of '0' for individual missing items (n = 8; mean number of missing items

= 1). This was determined to be a conservative method of handling missing data given that most items on the questionnaire have a greater than 75% of being endorsed 'No' except for separated/divorce which is 50%. Finally, hot deck data imputation was used to ensure complete study data on all remaining variables of interest (PAR, n = 3; MEP, n = 3; CPM, n = 3; TS, n = 3 [53].

All mediation analyses were conducted using PROCESS version 4.0 developed by Hayes [35]. PROCESS parameters were set such that models would be robust to violations in assumptions of normality and homoscedasticity. Bootstrapping was set to 10,000 samples with a 95% confidence interval (CI) to address distributional assumptions and achieve greater specificity of CI upper and lower limits. A heteroscedasticity-consistent standard error estimator, HC4, was used to address possible bias in the covariance matrix which could lead to erroneous significance tests and confidence intervals [36]. Continuous independent variables that define products were mean-centered to address the possible concern of multicollinearity between independent variables and the constructed cross-product term.

Descriptive statistics (mean, standard deviations, and frequencies) were obtained for primary variables and sociodemographic characteristics. Correlations of primary study variables were also estimated. Simple mediation multiple regression models were used to determine the associations between variables of interest in people with cLBP (PROCESS Model 4). **Figure 2A** depicts the conceptual diagram for our analyses. In all models, DERs was set as the mediator (M) and ACEs was set as the independent variable (X). The dependent variables (Y) were PAR (Model A) and MEP (Model) in individuals with cLBP. To examine our effects of interest more precisely, we included race, age, sex, BMI, cLBP duration, and medication use as covariates in our statistical models. Once designated as a covariate, PROCESS includes these variables in all aspects of the

mediation model (i.e. controls for the influence of each covariate in relation to the mediator and the dependent variables).

[FIGURE 2 HERE]

Results

Demographic Characteristics of the Sample

The average age of the sample was 44.05 years (*SD* = 13.87) with a range of 18 to 80 years. Much of the sample identified themselves as Non-Hispanic Black (62.8%), female (53.0%), and married (37.2%). The largest proportion of the sample (16.4%) reported their income to be between $0 and $9,999. The most common work status was "fulltime" (47%), followed by permanent disability (13.1%), and working "part-time" (10.9%). The most endorsed highest level of education was 'some college' (32.2%). Sample characteristics are displayed in **Table 1.**

[TABLE 1 HERE]

Clinical Characteristics of the Sample

The average number of ACEs was 1.91 (*SD* = 1.90), with a range of 0 to 9. Table 2 included details about the distribution of ACEs across the sample. The average score for DERs was 69.72 (*SD* = 20.28) on the DERs, with a range of 38 to 144. The average score for depressive symptoms was 17.21 (*SD* = 11.22) on the CES-D, with a range of 0 to 60; approximately 49.18% of the sample reported scores above the clinical depression cut-off (CES-D score of 16) [62]. Average MEP severity was 28.11 (*SD* = 25.91) with a range of 0 to 95. Average PAR on the BPI-SF was 4.24 (*SD* = 2.43), with a range of 0 to 10. Duration of cLBP ranged from 3 months to over 20 years, with the largest proportion of the sample having experienced pain for 5-10 years (23.5%). Additionally, over 65% of the sample endorsed taking some form of pain medication for their

cLBP. The average BMI was 31.16 (*SD* = 6.90) across the study sample with a range of 18.88 to 53.49. Descriptive characteristics for primary study variables are displayed in **Table 3.**

[TABLE 2 HERE]

[TABLE 3 HERE]

Zero-Order Correlations among Primary Study Variables

Individuals with higher ACEs scores exhibited greater difficulties with emotion regulation. ($r = .28$; $p < .01$). Similarly, individuals with higher ACEs scores presented with greater levels of self-reported depression via CESD ($r = 0.25$; $p < .01$). Notably, ACEs were not associated with PAR nor MEP. However, greater emotion dysregulation was associated with greater PAR ($r = 0.34$; $p < .01$) and greater MEP ($r = 0.29$; $p < .01$). All bivariate correlations between primary study variables are presented in **Table 4**.

[TABLE 3 HERE]

Mediating Effects of Emotion Regulation

Pain at Rest (PAR): A mediation model was performed to determine whether DERs mediated the association between ACEs and PAR, (Model A; **Figure 2B**). The overall model, adjusted for covariates, accounted for a significant 34% of the total variance in PAR ($F(9, 173) = 11.80$, $p < 0.01$). The coefficients for race (*coeff* = 1.85, $p < 0.01$), age (*coeff* = 0.03, $p = 0.03$) and depression ($t = 2.46$, $p = 0.02$) indicate that greater severity of PAR is reported by individuals identifying as older, Non-Hispanic Black, and those reporting higher levels of depressive symptoms. The mediation effect (c') of ACEs on PAR through DER had a point estimate of 0.05 and a 95% confidence interval of 0.004 to 0.118. This confidence interval suggests that, even after statistically controlling for covariates, the mediated effect was statistically significant at $p < 0.05$. The directions of paths a (*coeff = 1.57*, $p = 0.022$) and b (*coeff = 0.03*, $p = 0.0056$) are consistent

with the interpretation that greater ACEs are associated with greater DERs, which in turn, is associated with greater PAR.

Movement-Evoked Pain (MEP): A second simple mediation model was conducted to determine the extent to which DERS would mediate the association between ACEs and MEP (Model B; **Figure 2C**). The overall model, adjusted for covariates, accounted for a significant 27% of the total variance in MEP ($F(9, 173) = 8.49$, $p < 0.01$). The coefficients for race (*coeff* = 16.15, $p < 0.01$) and age (*coeff* = .340, $p < 0.01$) indicate that greater severity of MEP is reported by individuals identifying as older and Non-Hispanic Black. The mediation effect (c') of ACEs on MEP through DER had a point estimate of 0.544 and a 95% confidence interval of 0.044 to 1.21. This confidence interval suggests that, after statistically controlling for covariates, the mediated effect remains statistically significant at $p < 0.05$. The directions of paths a (*coeff* = 1.57, $p = 0.022$) and b (*coeff* = 0.34, $p = 0.01$) are consistent with the interpretation that greater ACEs is associated with greater DERs, which in turn, is associated with greater MEP.

Discussion

The primary objective of the current study was to determine the extent to which the association between childhood adversity and adult cLBP was mediated by emotion dysregulation. Emotion dysregulation is a known transdiagnostic risk factor connecting ACEs and adult psychopathology; our study extends this research to cLBP. Consistent with our hypothesis, greater difficulties with emotion regulation indeed mediated the relationship between ACEs and cLBP severity at rest and with movement. No significant direct associations were observed between ACEs and measures of cLBP severity (MEP, PAR) in our sample.

A wealth of literature demonstrates that ACEs can interrupt the development of emotional processes and lead to deficits in emotion regulation that last a lifetime [45]. Socioemotional development begins when primary attachments are formed with a child and their parents/caregivers [32]. Healthy attachment is characterized by feelings of security and safety. Child-caregiver relationships play a significant role in the development of emotion regulation through attunement, responsivity, understanding, and guidance [52]. Child emotion regulation relies heavily on social cues provided by caregivers that ultimately shape a child's self-esteem and concept. Normative development is characterized by more effective emotion regulation strategies as age increases.

The context in which development occurs can help or hinder this process depending on the stage of psychological, neurological, and biological maturation [18]. Individuals with history of childhood adversity have been found to rely more heavily on maladaptive emotion regulation strategies such as suppression, and demonstrate higher emotional lability, greater emotional intensity, and report greater negative affect later in life [51]. Emotion dysregulation has been identified as a mediator of associations between childhood adversity and conditions such as: depression, self-harm, posttraumatic stress disorder, substance use, and eating disorders [15; 56; 57].

Importantly, emotion regulation contributes to the subjective experience of pain by modulating perceptions of intensity, appraisals, interpretations of pain, and how individuals cope [41]. Chronic pain patients demonstrate increased difficulty identifying, differentiating, and regulating emotions, as well as selecting emotion regulation strategies[1]. A study by Le Borgne, Boudoukha, Petit and Roquelaure [42] found that lack of emotion regulation and awareness were associated with worse pain outcomes in a sample of individuals with cLBP. A more recent systematic review identified emotion dysregulation as a key risk factor in the development and

experience of chronic pain [41]. A study by Garland, Reese, Bedford and Baker [28] found female patients with chronic pain who reported exposure to ACEs were at especially high risk for becoming ensnared in the downward spiral of emotion dysregulation and subsequent opioid use disorder. The authors propose that impairment to emotion regulation may contribute to ineffective coping, and a shift to opioids as a means of coping with affective distress. A more recent study examined associations between childhood adversity, emotion regulation, and pain in individuals with alcohol use disorder [87]. The authors demonstrated a positive association between childhood emotional abuse severity and anxiety which in turn was negatively associated with pain tolerance. Furthermore, emotional dysregulation and anxiety acted as serial mediators in the association between childhood emotional abuse and pain tolerance. In line with previous research, the current study demonstrates that ACEs can increase adult cLBP severity through impaired emotion regulation, and highlights an important target for treatment.

Though no significant direct associations were observed between ACEs and cLBP severity in our sample, this finding is not entirely inconsistent with existing literature. A study by Craner, Lake, Barr, Kirby and O'Neill [20] found significantly greater pain ratings in participants reporting ≥ 4 ACEs compared to participants with 0-1 ACE. The participants within our sample reported an average of two ACEs, suggesting they may not have experienced a level of ACEs sufficient to support a direct association with cLBP severity. Moreover, participants reported on PAR and acute pain that arose during movement tasks. Our choice of assessment methods may partly explain why we were unsuccessful in finding a relationship between ACEs and cLBP severity. Specifically, it may be that the 24-hour version of the BPI and functional MEP tasks used in this study do not share the same relationship with ACEs as other measures that capture a longer duration of pain, such as the 7-day version of the BPI ("average pain severity for the past seven days") or measures

that capture multidimensional aspects of pain. In fact, a recent study by Leisner, Gerhardt, Tesarz, Janke, Seidler and Eich [43] utilized pain drawing, the McGill pain questionnaire, and the pain experience scale to examine ACEs and cLBP. They demonstrated that experience of childhood abuse was associated with higher pain intensity, spatial extent of pain, affective and sensory pain sensation, and disability compared to cLBP patients who had not experienced abuse. Finally, the lower number of reported ACEs in our sample may be influenced by participant-level factors such as recall bias, social desirability, or mistrust, particularly among racialized groups [9; 21; 37; 65]. Historical mistreatment and systemic disparities in healthcare and research settings may contribute to heightened levels of mistrust and skepticism toward disclosing sensitive information[6]. Therefore, addressing these nuances is essential for interpreting ACE data accurately within diverse populations. Future research should include pain assessments that capture varying levels of chronicity, pain type, behavior, and functional outcomes to elucidate the association more fully with ACEs.

Clinical Implications and Future Directions

Considering that ACEs are risk factors for the development, maintenance, severity, and exacerbation of chronic, it is imperative that pain providers not only assess and account for childhood adversity in their patients, and adopt a trauma-informed care approach when interacting with and treating their patients. [3-5; 10; 64; 70; 86] Indeed, experiences of ACEs and other trauma can negatively influence the way patients interact with healthcare providers which may result in poorer patient satisfaction, treatment adherence and health outcomes [11; 14; 25; 40; 63; 82]. Adopting a trauma-informed care approach may result in improved patient-provider relationships and, as a result, better pain outcomes.

Moreover, due to the role of DER among patients with cLBP and a history of ACEs, these patients may benefit from engaging in therapies and strategies aimed at improving emotion regulation skills [46]. For example, Dialectical Behavioral Therapy has been shown to improve emotional regulation by naming and understanding emotions, decreasing the frequency of unpleasant emotions, decreasing vulnerability to emotions, and decreasing emotional suffering [55]. It is possible that these strategies may be beneficial for people with cLBP and a history of ACEs to better regulate their emotions and decrease the impact of emotion regulation on pain. Notably, recent research focused on emotion regulation and pain has elucidated associations among various types of emotion regulation, pain and trauma [2; 48]. This growing body of literature not only provides avenues for tailoring emotion regulation interventions to pain patients who have experienced trauma, but also underscores the importance of investigating specific aspects of emotion regulation in relation to pain experience. Future studies should aim to delineate the unique relationships between cLBP pain severity and specific DERS subscales, such as nonacceptance of emotional responses, difficulty engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarit

Limitations

The present study focuses primarily on a psychological pathway through which ACEs may contribute to cLBP pain. Yet ACE-pain relationships remain above and beyond the influence of psychological consequences [76; 85]. Moreover studying these relationships within chronic pain populations can be complex given the effect of ACEs upon pain processing which may be masked by disease processes [66]. While our research provides valuable insights, it's important to acknowledge limitations in our statistical design and methodology. Though more commonly utilized in longitudinal research designs, there is evidence that mediation analysis with cross-

sectional data may be conducted provided there is a well-defined theoretical model. We acknowledge that cross-sectional data lack the temporal sequencing of longitudinal designs. However, we believe this research still offers insights into associations between variables. There are limitations regarding the assessment of ACEs in this study. For example, analyses relied on retrospective self-reporting, but research indicates that these often disagree with prospective reports [9]. Furthermore, the use of a dichotomous response set to assess presence of ACEs fails to capture important factors such as severity, frequency, timing, chronicity, and discontinuity of experiences. Finally, our sample comprised of community-dwelling, Non-Hispanic Black and White individuals with cLBP, which limits the generalizability of our study findings. Future research should incorporate longitudinal designs with multiple time-points to address temporal precedence, more diverse samples, and replicate these findings in other chronic pain conditions.

Conclusion

Chronic low back pain (cLBP) is a debilitating condition affecting millions of people internationally. Research has shown that exposure to ACEs can increase the risk of developing chronic pain later in life. The present study extends these findings by demonstrating that impaired emotional regulation, which can arise due to childhood adversity, may act as a psychological pathway through which ACEs contributes to cLBP in adulthood. These findings underscore the need for a biopsychosocial approach to chronic pain management that accounts for not only the physical contributors but also psychological and social factors.

Disclosures

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Conflicts of Interest: The authors of this manuscript certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Availability of data and materials

The data that supports the findings from this study are available from the corresponding author upon reasonable request.

Figure Legends

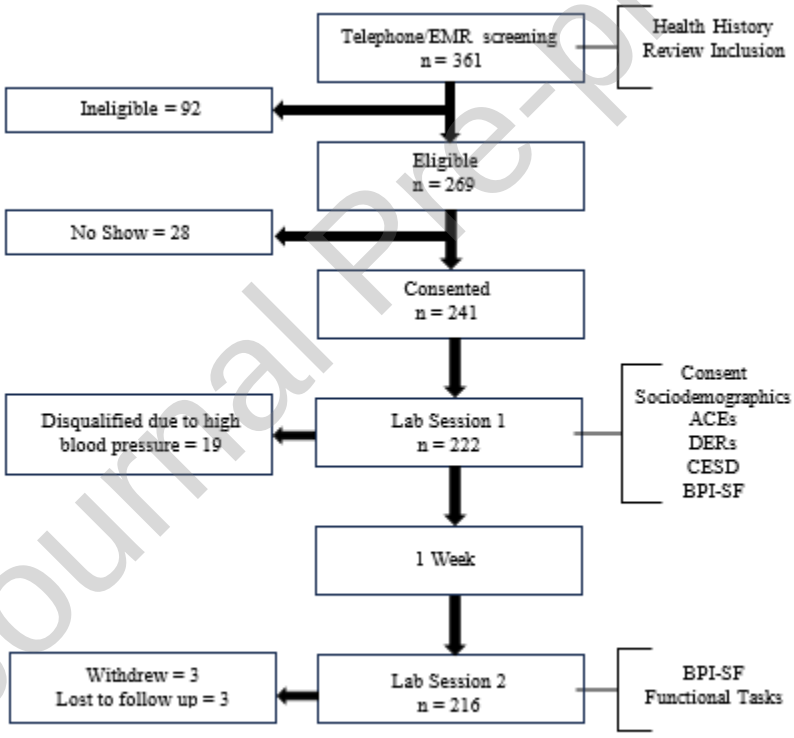

Figure 1. Flow diagram depicting matriculation through the present study. Note: EMR = electronic medical record; ACEs = adverse childhood experiences; DERs = Difficulties with emotion regulation scale; CESD = center for epidemiological studies depression scale; BPI-SF = brief pain inventory – short form.

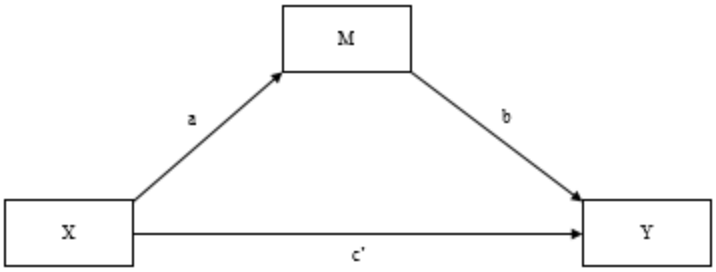

Figure 2A. Simple mediation depicting the estimated total and direct effect of the independent variable (X) on the dependent variable (Y) as well as the indirect effect of the independent variable on the dependent variable through a mediator variable (M).

Figure 2B.

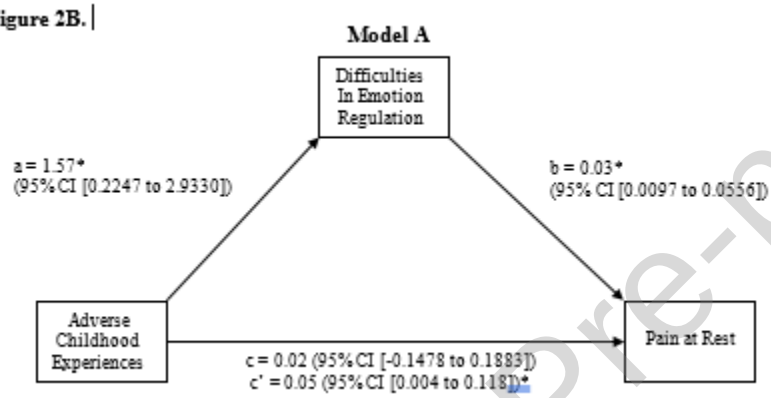

Figure 2B. The regression coefficients for the direct relationship between Adverse Childhood Experiences (X) upon Pain at Rest (Y) as well as the indirect effect through the mediator variable, Difficulties in Emotion Regulation (M). * indicates the coefficient is statistically significant at the p = 0.01 level.

Figure 2C.

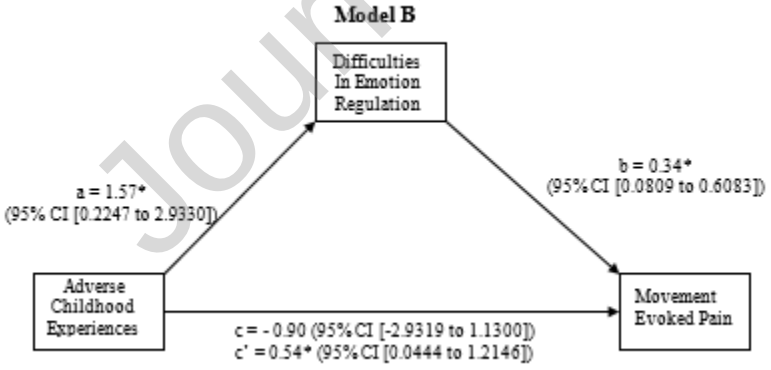

Figure 2C. The regression coefficients for the direct relationship between Adverse Childhood Experiences (X) upon Movement-Evoked Pain (Y) as well as the indirect effect through the mediator variable, Difficulties in Emotion Regulation (M). * indicates the coefficient is statistically significant at the p = 0.01 level.

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Table 1. Sample Characteristics. *LTR = long term relationship.

Variable		***M***	***SD***
Age		44.05	13.87
BMI		31.16	6.90
		n	***%***
Sex	Female	97	53.0
	Male	86	47.0
Race	Non-Hispanic Black	115	62.5
	Non-Hispanic White	69	37.5
Income	$0-9,999	30	16.8
	$10,000-24,999	39	21.2
	$25,000-49,999	38	20.7
	$50,000-74,999	29	15.8
	$75,000-$99,999	21	11.4
	$100,000 and greater	24	13.0
Work Status	Full-time	86	47.0
	Part-time	20	10.9
	Unemployed	20	11
	Retired	10	5.5
	Disability	25	13.6
	Student	10	5.4
	Homemaker	5	2.7
	Other	6	3.2
Highest Education	Partial high school	10	5.5
	High school graduate	33	18.0

	Partial college	59	32.2
	College graduate	38	20.8
	Grad/professional training	43	23.5
Marital Status	Married	68	37.2
	Widowed	5	2.7
	Divorced/Separated	25	13.6
	Never Married	50	27.3
	Living with Partner	20	10.9
	Other	15	8.2

Table 2. Distribution of ACEs Across the Sample.

Number of ACEs	**n**	**%**
.00	48	26.2
1.00	49	26.8
2.00	30	16.4
3.00	22	12.0
4.00	15	8.2
5.00	7	3.8
6.00	7	3.8
7.00	3	1.6
8.00	1	.5
9.00	1	.5
Total	183	100.0

Table 3. Primary Study Variables. *denotes a covariate. ACEs = adverse childhood experiences; DERs = difficulties in emotion regulation scale; CES-D = center for epidemiological studies depression scale; PAR = pain at rest; MEP = movement-evoked pain; cLBP = chronic low back pain.

Variables	***M***	***SD***
ACEs	1.91	1.90
	n	***%***
Emotional/psychological abuse	42	23.0
Physical abuse	33	18.0
Sexual abuse	31	16.9
No love in household	39	21.3
Neglect	13	7.1
Parent divorce/separation	94	51.4
Witnessed domestic violence	22	12.0
Household problematic drinking	45	24.6
Household member was mentally ill	46	25.1
Household member went to prison	25	13.7

		M	*SD*
DERs		69.72	20.28
CES-D		17.21	11.21
Pain Variables			
PAR		4.24	2.43
MEP		28.11	25.91
		n	***%***
cLBP duration *	3 to 6 months	8	4.4
	6 months to 1year	12	6.6
	1 to 3 years	31	16.9
	3 to 5 years	34	18.6
	5 to 10 years	43	23.5
	10 to 20 years	42	13.0
	Over 20 years	13	7.1
cLBP medication*	Yes	120	65.6
	No	63	34.4

Table 4. Bivariate Correlations of Primary Study variables.

	1 ACES	**2** DERS	**3** CES-D	**4** PAR	**5** MEP
1	-				
2	0.28**	-			
3	0.25**	0.66**	-		
4	0.11	0.34**	0.41**	-	
5	0.05	0.29**	0.29**	0.74**	-

Highlights

- ACEs interrupt the development of emotional processes, with lifelong effects.
- ACEs are associated with greater difficulties with emotion regulation in adulthood.
- Adults with poor emotion dysregulation report greater cLBP severity.
- Emotional dysregulation may explain how ACEs contribute chronic pain over time.