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Adverse Childhood Experiences and Chronic Low Back Pain in Adulthood: Roles of Emotion Regulation and Socioeconomic Status

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ADVERSE CHILDHOOD EXPERIENCES AND CHRONIC LOW BACK PAIN IN ADULTHOOD: ROLES OF EMOTION REGULATION AND SOCIOECONOMIC STATUS

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ABSTRACT

Adverse childhood experiences (ACEs) can have profound deleterious effects on physical health and psychological functioning in adulthood. ACEs may be a social determinant of chronic pain development and severity in adulthood; however, little research to date has investigated the psychological processes that might underlie this association. Emotion regulation, an established transdiagnostic risk factor underlying psychopathological conditions, may be a potential mediator of the relationship between ACEs and pain. Lower socioeconomic status is associated with greater risk of ACEs and poor chronic pain outcomes, yet research has not clarified the role of subjective versus objective determinants of socioeconomic factors within these associations. Finally, pain research has prioritized the assessment of pain-at-rest, yet there is a need for research which examines pain with movement and endogenous pain modulation. The primary objective of this study was to examine the relationships among ACEs, emotion regulation, and adult cLBP, as well as to determine whether associations vary by SES.

Study participants included 183 adults (53% female, 62.8% non-Hispanic Black) with chronic low back pain (cLBP). Participants reported on ACEs, emotion regulation, pain-at-rest, movement-evoked pain, area deprivation, and subjective social status. All participants completed experimental assessment of endogenous pain modulation using quantitative sensory testing. Sociodemographic data were also collected.

No significant bivariate associations were observed between ACEs and cLBP severity measures. Greater ACEs were significantly associated with less efficient pain inhibition, but only for those living in less socioeconomic disadvantage. Greater ACEs were associated with greater difficulties in emotion regulation; though this association was not moderated by indicators of SES. Emotion dysregulation and pain severity measures were significantly associated, however the same was not found for endogenous pain modulation responses. The data revealed that the relationship between ACEs and movement-evoked pain was significantly mediated by emotional dysregulation. Unexpectedly, this mediation effect did not differ in strength or magnitude according to indicators of SES. The present study reinforces the link between ACEs and chronic pain, underscoring the importance of childhood maltreatment, impaired emotional regulation, and socioeconomic disadvantage as a risk factors cLBP. Implications of these findings are discussed.

Keywords: social determinants, chronic pain, psychology

TABLE OF CONTENTS

Page

LIST OF TABLES

LIST OF FIGURES

Figure *Page*

BACKGROUND AND SIGNIFICANCE

Chronic Low Back Pain

Low back pain is characterized by physiological, psychological, and social dimensions that together result in substantial burden at the individual, community, healthcare system, and government level. Notable impacts include limited physical activity, pain, social isolation, psychopathology, reduced work productivity, financial burden, and heavy healthcare utilization (Deyo et al., 2015; Meucci et al., 2015). Low back pain is a considered a symptom that can result from several different known or unknown abnormalities or diseases. For most individuals with low back pain, it is not possible to identify an exact cause. This condition often presents with pain that radiates down one or both lower limbs, and can include neurological symptoms in the lower extremities (Dionne et al., 2008). Furthermore, a large percentage of individuals with low back pain also report pain in other regions of their body, resulting in increased disability, and poorer treatment response (Hartvigsen et al., 2013). Chronic low back pain (cLBP) is defined as pain that continues for 12 weeks or longer, even after an initial injury or underlying cause of acute low back pain has been treated. In 2016, 13% of adults in the United States experienced cLBP and it remains a leading cause of disability in the United States (Maher et al., 2017).

Social Determinants of Chronic Low Back Pain

Though the biopsychosocial model is the dominant framework for conceptualizing cLBP, there remains a dearth of research on the social determinants operating within the condition (Pincus et al., 2013). Social determinants of health (SDH) characterize the

economic, educational, physical, and interpersonal environments where people live, learn, and work (Braveman et al., 2011) (Hasbrouck, 2021; Pronk et al., 2021). These factors act at the individual, community, and system level to create the context within which health behaviors develop and contribute substantially to a variety of health conditions – including cLBP.

Studies demonstrates that SDH shape the distribution of behavioral risk factors, comorbid conditions, and exposure to environmental risks. Relationships between the SDH and disease are demonstrated by the social gradient in health whereby lower socioeconomic status is associated with worse health. Research suggests that these relationships exist between cLBP and SDH as well. Certain social risk factors (such as education and occupation) are repeatedly associated with negative outcomes (Karran et al., 2020). However, much of the current cLBP literature focuses on individual-level social determinants rather than examining the greater social conditions that may impact outcomes. There is a need for greater recognition that SDH are clearly associated with disparities in cLBP outcomes and further research examining potential explanatory mechanisms.

Adverse Childhood Experiences

ACEs are SDH consisting of both direct and indirect traumatic events that act as substantial sources of social threat (Felitti et al., 1998). ACES can include emotional, physical, sexual abuse, parental psychopathology, substance abuse, early parental loss due to death/abandonment, parental incarceration, or conflict (Boullier & Blair, 2018). Data from the 2016 and 2018 Health Resources and Services Administration's National Survey of Children's Health revealed 30% to 45% of children have experienced at least one ACE

in their life (Groenewald et al., 2020; Lebrun-Harris et al., 2022). Across the nation, the most prevalent ACEs surround economic hardship (25%) and parental/guardian divorce (25%). Furthermore, the risk of ACEs is affected by racial and ethnic minority group belonging; 60% of non-Hispanic White children reported no ACEs compared to 49% of Hispanic and 39% of non-Hispanic Black children (Sacks & Murphey, 2018). Finally, studies consistently demonstrate the profound deleterious effect of ACEs upon subsequent physical health and psychological functioning in adulthood (Hughes et al., 2017; Petruccelli et al., 2019).

Though previous research substantiates an association between ACEs and chronic pain, the evidence regarding the magnitude and direction of this relationship has been mixed (Bussières et al., 2020). A 2005 metanalysis found that individuals with ACEs report a greater number of pain symptoms. Results indicated chronic pain patients to be more likely to report ACEs compared to non-patients with chronic pain or healthy controls. In addition, individuals reporting pain were also more likely to also report ACEs (Davis et al., 2005). However, though studies have established the presence of ACEs to increase the likelihood of developing adult back pain, there has been little to no research investigating the psychological processes that might underlie this relationship.

Emotion Regulation

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 (Gross, 2015). Emotion regulation comprises a major part of the subjective experience of pain, and may be a potential route through which ACES contribute to cLBP etiology and maintenance (Solberg Nes et al., 2009). A confluence of social factors present

during early life (e.g., sociocultural influences, temperament, family environment) dynamically interact to influence the development of emotion regulation (Morris et al., 2007). Research demonstrates that childhood experiences become biologically, psychologically, and emotionally embedded to ultimately impact adult functioning (Gunnar & Quevedo, 2007). A large body of research demonstrates that childhood maltreatment hampers the development and acquisition of critical emotion regulation skills (Loman & Gunnar, 2010; Milojevich et al., 2020). Finally, difficulty in emotion regulation (DER) has been established as a transdiagnostic process; DER is a risk or maintenance factor underlying many health conditions, including cLBP (Le Borgne et al., 2017).

Emotion regulation related to chronic pain is characterized by deficits in emotional identification, regulation of negative affect, and emotional regulation strategy selection (Aaron et al., 2020). *Emotional Identification:* Persistent stressors, such as chronic pain, can weaken the ability to identify and differentiate between positive and negative emotional states, and hinder adaptive regulation. In cLBP populations, decreased emotional awareness has been associated with increased pain, disability, and psychological distress (Le Borgne et al., 2017). These findings are substantiated by high rates of alexithymia – inability to describe and label emotions, and limited introspective thinking – in individuals with cLBP (Kapadi et al., 2021). Emotion labeling comprises a form of implicit emotion regulation and has been found to reduce negative affect (Torre & Lieberman, 2018). However, given that individuals with chronic pain often describe their emotions in physical terms, they may be biased toward explicit emotion regulation strategies, and fail to engage in other effective options. *Regulation of Negative Affect:* Much research has focused on pain catastrophizing - the tendency to magnify pain,

ruminate upon pain-related cognitions, and feel helpless when faced with pain. The maladaptive response is associated with diminished pain inhibition, longer recovery times, pain severity, and disability (Quartana et al., 2009). Ineffective down-regulation of negative affect has been shown to contribute to the Fear-Avoidance model of chronic pain. According to the model, when pain is perceived as excessively threatening, then maladaptive responses, such as catastrophizing, can lead to activity avoidance. Ultimately, individuals become stuck in a vicious cycle whereby their avoidance of activity leads to physical deconditioning. *Emotional Regulation Strategy Selection:* A past systematic review suggests that use of maladaptive emotion regulation strategies is directly associated with poor outcomes such as increased pain intensity, disability, and depressive symptoms (Koechlin et al., 2018).

Social-Safety Theory

Social-Safety Theory (SST) provides a framework for understanding how social factors such as ACEs may contribute to the development and perpetuation of cLBP, by influencing psychological risk and resilience (Slavich, 2020). The theory conceptualizes situations of social threat (the opposite of social safety) as instances of adversity that influence neurocognition and immune mechanisms to affect health. According to SST, evolution has preserved a bias for social bonds in humans as these connections offered increased safety in the face of physical threat (Dunbar & Shultz, 2007). Over millions of years, this primary drive has led to the development of the "social brain" – extensive, intricately connected brain regions (amygdala, mentalizing network, empathy network, and mirror neuron system) responsible for mediating social cognition and behavior (Atzil et al., 2018; Kennedy & Adolphs, 2012). Bidirectional interactions between social factors and

pain have been well established (Sturgeon & Zautra, 2016). Research demonstrates that social threat (i.e., exclusion, rejection, discrimination, or isolation) can lead to the development and exacerbation of chronic pain (Riva et al., 2014). Similarly, pain has been found to negatively impact social relationships (Riva et al., 2011). Furthermore, studies have identified overlapping neurochemical and neuroanatomical underpinnings of physical pain and social threat, indicating that the processing of social rejection may annex established pain pathways (Eisenberger et al., 2003).

The bias toward social bonds is further represented within the immune system; humans have maintained an adaptive, anticipatory inflammatory response in the face of social threat (Glaser & Kiecolt-Glaser, 2005). However, higher-order cognitive abilities (i.e., evaluation of past and imagination of future instances of social threat) can hijack the immune response (Dantzer, 2018; Denson et al., 2009; Irwin & Cole, 2011). Chronic activation can result in an accumulation of "wear and tear" upon the immune regulatory system, and lead to a plethora of adverse health outcomes, including pain (Furman et al., 2019; Nees et al., 2019). Taken together, SST suggests that social threat may place individuals at an increased risk of developing chronic physical health conditions such as cLBP. Infancy to adolescence represents a period likely to be particularly sensitive to social threat (such as maltreatment) considering the substantial physical, psychological, and behavioral development that occurs at this time. More specifically, childhood is a crucial period when individuals learn how to communicate, interact, and understand others. It is well established that childhood maltreatment and neglect can result in the development of maladaptive cognitive frameworks surrounding social safety and threat. Research demonstrates that the experience of ACEs is related to greater impairment in social functioning including greater

perceived social isolation, greater family conflict, and decreased perceptions of social support.

Socioeconomic Disadvantage

Socioeconomic disadvantage can be reconceptualized as circumstances of social threat which contribute to the development, maintenance, and exacerbation of chronic pain (Craig et al., 2020). A substantial body of research describes how individual-level socioeconomic characteristics impact physical and mental health outcomes (Chen & Miller, 2013). Notably, low childhood SES is associated with greater risk of maltreatment, indicating its potential influence upon the association between ACEs and adult cLBP (Karran et al., 2022). Overall, higher rates of cLBP are seen in individuals reporting low SES, lower educational achievement, and physically demanding jobs (Fliesser et al., 2018; Ikeda et al., 2019; Vos & et., 2016). However, the impact of socioeconomic disadvantage upon health cannot be fully assessed without considering the community context since socioeconomic and environmental factors (e.g., access to healthcare, exposure to crime and violence, crowding, municipal services, recreational resources, and food insecurity) cluster at the neighborhood level to effect disease burden, violence, injury, and healthcare opportunity (Gaskin et al., 2019; Ross & Mirowsky, 2001).

Composite measures such as the ADI accurately capture neighborhood socioeconomic context (Chamberlain et al., 2020; Durfey et al., 2019). The ADI assesses neighborhood deprivation using socioeconomic variables within the following categories: education, employment, income, and housing quality (Kind & Buckingham, 2018). A recent study by Jackson et al. (2021) found the ADI to be an effective measure of neighborhood disadvantage and a better predictor of cLBP severity than individual-level SES measures.

Studies examining the ADI in cLBP samples demonstrate that vulnerability to this condition may be greater for those in disadvantaged communities. Rumble et al. (2021) found a positive relationship between pain intensity and neighborhood disadvantage, and that influence of pain status (cLBP versus pain-free) upon sleep disturbance was greater for individuals living in more disadvantaged neighborhoods.

Much existing research solely focuses on objective social status measures of SES such as educational level, income, occupational status, and the ADI among others. However, the inclusion of SSS may provide a more complete picture of an individual's social standing (Cundiff & Matthews, 2017). This is because SSS represents an individual's perception of their social standing that is independent from SES alone, and is sensitive to the real-world implications of one's resources (Singh-Manoux et al., 2005). SSS can act as an important indicator of how individuals internalize the reality of their socioeconomic condition by capturing feelings of relative deprivation, financial (in)opportunity, and social mobility. Low SSS has been associated with poor physical, mental, and health behavior such as hypertension, depression, diabetes (Adler & Stewart, 2007; Cené et al., 2016; Zell et al., 2018). A recent study by Mu et al. (2022) examining community-dwelling older men (≥65 years) found that SSS moderated the effect between back pain and mental health, such that more severe back pain was associated with worse mental health outcomes for individuals with low SSS. Given that SSS appears to impact pain and mental health both directly and indirectly (above and beyond traditional SES indicators), it appears warranted to consider how SSS may influence the relationship between ACEs and cLBP.

Pain Experience and Processing

Given the highly subjective experience of pain, self-report has been the gold standard for assessing experimental pain. Numerical rating scales (NRS) for pain are one of the most common tools used in clinical settings. Historically, clinical and experimental pain research has prioritized the assessment of the sensory and affective qualities of *pain at rest (PAR).* However, this limited focus has hampered research on the mechanisms underlying pain. There is now a growing consensus that research should include a variety of pain assessment methods to expand our understanding of somatosensory function in musculoskeletal pain conditions.

Movement contributes substantially to pain and disability for cLBP, however *movement-evoked pain (MEP)* remains understudied due to a traditional focus on PAR assessments (Litcher-Kelly et al., 2007). A growing body of literature indicates that PAR fails to capture pain related to movement, and documented differences in treatment efficacy for MEP vs PAR suggest distinct underlying mechanisms (Landmark et al., 2011; Rakel & Frantz, 2003). MEP is an emerging model of pain assessment which integrates the study of sensory, motor, and psychological determinants of pain (Corbett et al., 2019). By incorporating MEP, we may be able to clarify how pain and movement interact bidirectionally within a chronic pain population.

Though the etiology of the vast majority of cLBP is nonspecific and poorly understood, there is consensus that sensitization of central and peripheral pain processing pathways plays an important role (Giesecke et al., 2004; Pavlaković & Petzke, 2010). *Endogenous pain modulation* describes the complex descending inhibitory and facilitatory processes within the central nervous system that regulate pain perception (Ossipov et al., 2014). The assessment of endogenous pain modulation provides information regarding the

pain pathways involved in central sensitization (Corrêa et al., 2015; Staud, 2012). A large body of research indicates that the balance of pain facilitatory and inhibitory processes is affected by emotional, cognitive, behavioral, and physiological factors. Quantitative sensory testing (QST) involves non-invasive, standardized experimental procedures that can be used to understand endogenous pain modulation, with the goal of clarifying the mechanisms underlying chronic musculoskeletal pain. QST allows for systematic assessment of nociception using multiple stimulus modalities which engage different nerve fibers, components of afferent somatosensory transmission, and central nervous system pain pathways (Cruz-Almeida & Fillingim, 2014; Fillingim et al., 2016).

Thermal (heat, cold) and mechanical (pressure) stimulation of cutaneous tissue is most frequently used in QST research. Pressure stimuli are particularly useful for musculoskeletal pain conditions, such as cLBP, as increased pain sensitivity can demonstrate mechanical sensitization (Sangesland et al., 2017). QST results in both static and dynamic response measures, the latter being more reliable and validated for musculoskeletal pain (Arendt-Nielsen & Yarnitsky, 2009). response measures include pain threshold (the intensity at which a stimulus is first perceived to be painful) and tolerance (the maximum tolerated pain produced by a stimulus). Dynamic response measures include temporal summation (TS) and conditioned pain modulation (CPM). TS occurs when a noxious stimulus, applied repeatedly at the same intensity, leads to an increase in perceived pain. TS is the psychophysical manifestation of wind-up; a pain facilitatory processing. CPM describes the inhibition of a noxious stimulus through the application of another noxious stimulus elsewhere on the body. CPM reflects the manifestation of diffuse noxious inhibitory controls - ascending projections from adverse stimuli trigger supraspinal regions

which in turn activate descending inhibitory (opioidergic, serotonergic, noradrenergic) projections (Ramaswamy & Wodehouse, 2021).

A growing body of research indicates that individuals with chronic pain demonstrate pathophysiological pain processing compared to controls using QST methodology (LeResche et al., 2013). For individuals with cLBP, this dysfunction is characterized by diminished inhibition (shorter duration CPM) and increased facilitation of pain (greater TS summation of mechanical and heat pain), which has been associated with increased severity and disability (Corrêa et al., 2015; Mlekusch et al., 2016; Owens et al., 2016). Research continues to substantiate the use of QST for characterizing normal and pathophysiological pain perception, differentiating chronic pain conditions, generating pain modulation profiles (to predict risk and resilience), and as a treatment outcome marker.

Taken together, it is likely that social threat, such as socioeconomic disadvantage or childhood maltreatment, may increase the risk of developing cLBP by hampering the development and acquisition of critical emotion regulation skills. The primary objective of this study is to examine the relationships among ACEs, emotion regulation, and adult cLBP. We will further determine whether the associations are affected by neighborhood deprivation and perceived social standing. The findings from this proposed research may provide evidence for a psychological pathway through which social experiences may contribute to the development of cLBP.

SPECIFIC AIMS AND HYPOTHESES

Specific Aim 1: To investigate the extent to which ACEs are associated with pain at rest, movement-evoked pain, endogenous pain modulation profiles (TS and CPM), and both objective and subjective indicators of SES (SSS and ADI) in people with cLBP.

Hypothesis 1: Greater number of ACEs will be associated with greater severity of adult cLBP severity (↑PAR, ↑MEP) and a pro-nociceptive endogenous pain modulation profile (↑TS, ↓CPM), particularly for individuals with lower SES (↓SSS and ↓ADI).

Specific Aim 2: To examine associations among ACEs, difficulties with emotion regulation and objective, as well as subjective indicators of SES in people with cLBP.

Hypothesis 2: Greater number of ACEs will be associated with greater difficulties in emotion regulation, particularly for individuals with lower SES (↓SSS and ↓ADI).

Specific Aim 3: To determine if difficulties with emotion regulation help explain the relationship between ACEs and adult cLBP, and whether this is affected by SES.

Hypothesis 3: Greater difficulties with emotion regulation will help explain (i.e., mediate) the relationships between ACEs and cLBP severity (↑PAR, ↑MEP) as well as a pro-nociceptive endogenous pain modulation profile (↑TS, ↓CPM), particularly for individuals with lower SES (↓SSS and ↓ADI).

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 procedures and methods detailed below are limited to the proposed study. The data collected as part of this proposal and the larger ERASED study were approved and conducted in compliance with an Institutional Review Board (IRB).

All potential participants underwent a telephone and electronic medical record screening to determine eligibility. Health and cLBP history were reviewed by the principal investigator (Goodin) 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 which clinical, social, QST, psychological, and functional performance data is collected. **Figure 1** depicts a flow diagram illustrating matriculation through the current study.

Participants

The participants included in this study were selected using the screening criteria of the parent project (ERASED). Eligible participants (1) endorsed persistent, non-specific cLBP for at least 3 months and pain for at least half the days in the past 6 months (criteria

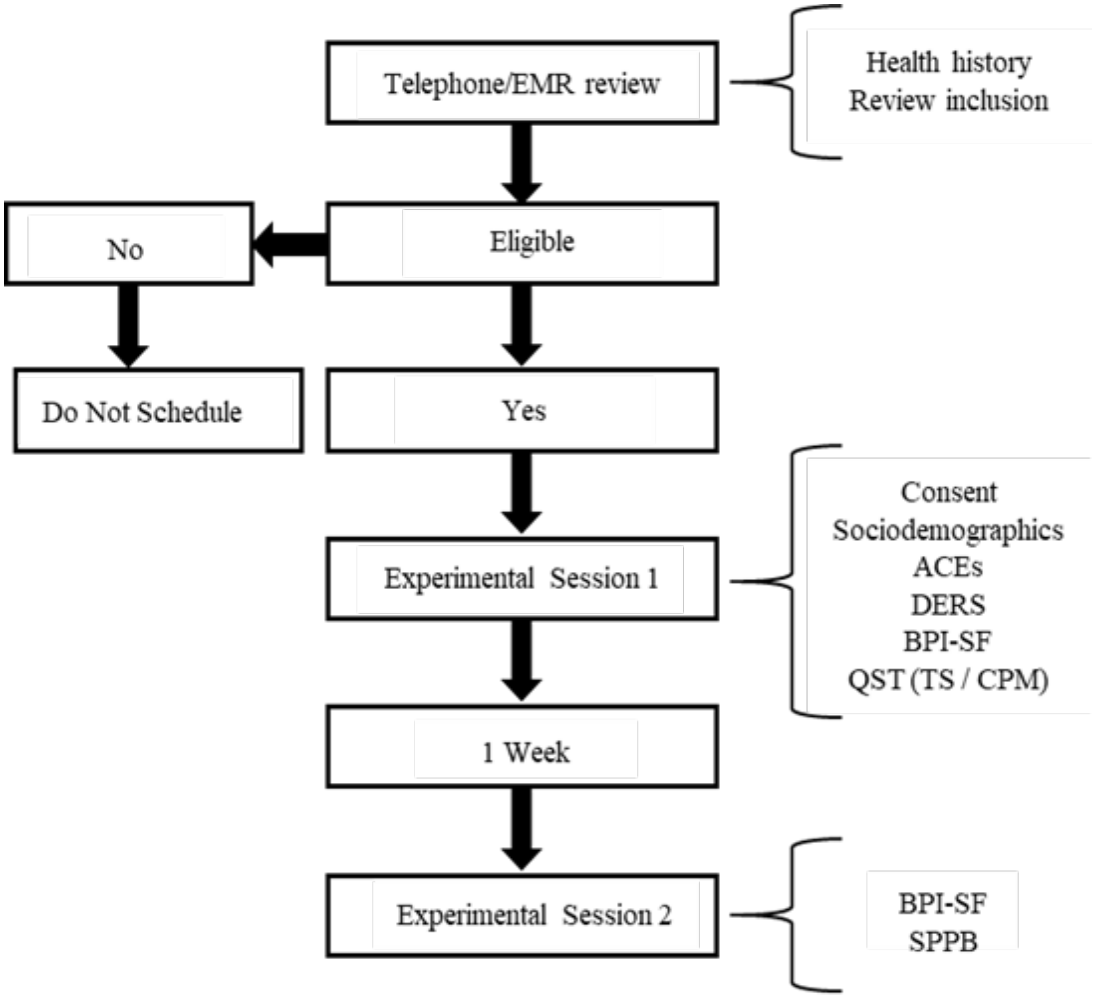

Figure 1. Flow diagram depicting matriculation through the parent study (NCT03338192). Note: EMR = electronic medical record; ACEs = Adverse Childhood Events; DERS = difficulties with emotion regulation scale; BPI = Brief Pain Inventory – Short Form; QST = Quantitative Sensory Testing; TS = temporal summation; CPM = conditioned pain modulation; SPPB = Short Physical Performance Battery

by the American College of Physicians and the American Pain Society), (2) were between 19 and 85 years of age (this range was chosen to capture young adults with cLBP and excluded individuals increasingly likely to meet one or more exclusion criteria), and (3) identified as either non-Hispanic Black or non-Hispanic White.

Participants with medical conditions that could interfere with study procedures or interpretation of study results were excluded. Exclusionary conditions include low back

pain attributable to other factors (e.g. ankylosing spondylitis, infection, malignancy, compression fracture of other trauma); systemic rheumatic disease; any chronic pain condition reported as more prominent or severe than the cLBP; 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; serious psychiatric disorder requiring hospitalization in the past 12 months; and pregnancy. *These conditions affect both participant safety and potentially alter pain perception.

Procedure

Recruitment and Screening

Participants were recruited primarily using study flyers posted within a UAB pain treatment clinic and the surrounding Birmingham community. A stratified sampling approach (using reported household income and number of home occupants) was incorporated to ensure recruitment of similar numbers of non-Hispanic Blacks and non-Hispanic Whites with high and low SES.

A telephone screening determined participants' initial eligibility, after which an electronic medical record review was completed (where possible) for all potential participants to confirm study eligibility. The review confirmed cLBP status, and assessed the reported duration of cLBP, current and past treatments for cLBP, comorbid conditions, and current medication use for all participants. This cLBP screening protocol was based on

research standards developed by the Research Task Force of the NIH Pain Consortium (Deyo et al., 2015).

Measures

Demographic and Clinical Measures.

Data was collected through self-report questionnaires. Demographic and clinical variables of interest included: (i) *age*, (ii) *race/ethnicity*, (iii) *gender*, (iv) *marital status,* (v) *body mass index (BMI*, (vi), *duration of pain, and (vii) current pain treatments*.

Socioeconomic Measures.

Income. Annual household income was collected as an individual-levels SES characteristic and it was assessed as an ordinal variable: (1) "Less than $10,000," (2) "$10,000 to $19,000," (3) "20,000 to 29,999," (4) "30,000 to 39,000," (5) "40,000 to $49,000," (6) "50,000 to 59,999," (7) "60,000 to 69,999," (8) "70,000 to $79,999," (9) "80,000 to 89,999," (10) "$90,000 to $99,999," (11) "100,000 to $149,999," and (12) "150,000 or more."

Education. Educational attainment is another individual-level SES characteristic that was assessed as an ordinal variable: (0) "N/A," (1) "No Schooling Completed," (2) "Nursery School to 8th Grade," (3) "9th, 10th, or 11th grade," (4) "12th grade, no diploma," (5) "High School Graduate – High School Diploma or Equivalent (e.g., GED)," (6) "Some College Credit, but Less Than One Year," (7) "One or More Years of College, No Degree," (8) "Associate's Degree," (9) "Bachelor's Degree," (10) "Master's Degree," (11) "Professional Degree (e.g., MD)," and (12) "Doctorate Degree (e.g., PhD)."

Area deprivation index (ADI). We used the 2019 ADI v3.0 to assign each participant a State Area Deprivation Index (sADI) value according to their census block

group at the time of injury and follow-up (Kind & Buckingham, 2018). The sADI served as a neighborhood-level indicator of economic (dis)advantage. sADI values range from 1 to 10 with higher scores indicating greater neighborhood deprivation. "Neighborhood" is defined as a Census Block Group. The measure has previously been utilized within cLBP research (Rumble et al., 2021).

Subjective social status (SSS). The MacArthur scale of SSS assesses an individual's perceived social status in comparison to their local community (Adler & Stewart, 2007). In this instance, community SSS (cSSS) best represents an individual-level SES indicator that is shaped by neighborhood SES. Participants placed an 'X' on the rung which they feel best captures their own cSSS, using a diagram of a ladder (where the top rung represents people perceived to possess the highest cSSS and the bottom rung represents those perceived to have the lowest). Studies confirm application of this measure in cLBP research (Mu et al., 2022; Shaked et al., 2016).

Psychosocial Measures.

Adverse childhood experiences (ACEs). The ACEs questionnaire assesses childhood exposure to physical, emotional, sexual abuse, neglect, parental divorce, domestic violence, substance use, psychopathology, and incarceration. Higher scores indicate more experiences of childhood trauma.

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 (Gratz & Roemer, 2004). The 36-item questionnaire results in six subscales: nonacceptance of emotional responses (items 11, 12, 21, 23, 25, 29), difficulty engaging in goal-directed behavior (13, 18, 20R, 26, 33), impulse

control difficulties (3, 14, 19, 24R, 27, 32), lack of emotional awareness (2R, 6R, 8R, 10R, 17R, 34R), limited access to emotion regulation strategies (15, 16, 22R, 28, 30, 31, 35, 36), and lack of emotional clarity (1R, 4, 5, 7R, 9). Higher scores indicate greater difficulty for subscales and the total score. This measure has previously been applied to cLBP populations (Le Borgne et al., 2017).

Pain Measures

Pain at rest. The nine-item Brief Pain Inventory – Short Form (BPI-SF) assesses pain severity, impact on daily function, pain location, and medication use. For this study, we examined a single item assessing the severity of participants' pain experienced at the time of the visit ("pain right now"). The item was scored from 0 ('*no pain or does not interfere'*) to 10 ('*worst imaginable pain or completely interferes'*). Higher scores indicate worse pain severity. The BPI-SF has previously been used in samples with cLBP (Mendoza et al., 2006; Song et al., 2016).

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 then 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 (Strath et al., 2022).

Dynamic QST Modalities. Quantitative Sensory Testing (QST) procedures were utilized to assess endogenous pain facilitatory processes (temporal summation (TS) of mechanical pain) and inhibitory processes (conditioned pain modulation or CPM).

Temporal Summation (TS). TS of mechanical pain was assessed using a weighted (512 mN) pinprick stimulator. The location of the stimulation was the erector spinae muscles of the lumbar spine. At an angle perpendicular to the contact point, the stimulator was lowered gently until the weighted probe retracted fully within the cylinder. The pin prick was administered once, and then participants were prompted to provide a pain intensity rating using a NRS whereby 0 is 'no pain' as 100 is 'most intense pain imaginable.' The pin prick was then repeated 10 consecutive times at a rate of one contact per second. Next, participants provided a single rating reflecting the greatest pain intensity experienced during the 10 contacts. The procedure was repeated twice on the same body area (lumbar spine). Pain ratings for the single and multiple contacts were averaged over the two trials. Prior to data analysis, TS effects (Δ change) were calculated by subtracting the pain intensity ratings following the first contact from the ratings following the series of 10 contacts.

Conditioned pain modulation (CPM). CPM was tested at the same location (erector spinae muscles of the lumbar spine) by simultaneously applying algometry (test stimulus) during immersion of the hand into a cold pressor (conditioning stimulus). Three applications of a handheld algometer were used to determine each participants' baseline pressure pain thresholds (PPTs). As pressure gradually increased (30 kilopascals/second), participants were asked to indicate when the stimulation is first perceived to be painful. After the baseline PPT is determined, the participants underwent two trials of cold pressor immersion. Participants were asked to place their entire hand, up to the wrist, into 12°C

water for 60 seconds. Immediately following the removal of the hand from the cold pressor, the algometer was used to elicit noxious stimulation at the lumbar region. Participants indicated when they first perceived the pressure as painful (conditioned PPT). The trial was repeated for a second time following a two-minute rest period. The three baseline PPTs were averaged, as were the two conditioned PPTs. CPM effects were calculated as a percent change from baseline ((Conditioned PPT – Baseline PPT) / Baseline PPT) * 100).

Data Analysis

SPSS (Statistical Package for the Social Sciences) version 29.0 was utilized to analyze data at a statistical significance level of $p < 0.05$ (IBMCorp, 2021). All hypotheses were assessed using PROCESS version 4.0 developed by Hayes (2012) . Variables of interest were examined to identify missing values, assumption violations and statistical outliers. In regard to external validity, 5 to 10% of cases with missing data were recognized as acceptable to consider for listwise deletion (Tabachnick et al., 2007). As a contingency, a simple data imputation method may be conducted to ensure complete study data for all participants. Descriptive statistics (mean, standard deviations, and frequencies) were obtained for primary variables and sociodemographic. Correlations of primary study variables were also estimated.

Statistical Power

Recommended sample size was determined using G*Power, version 3.1.9.6 (Faul et al., 2007). A priori power calculations were conducted using two bivariate normal models. Parameter specifications included power of 0.80 and an alpha level of 0.05. Effect sizes were based in previous literature; the first model focused on ACEs and DERs ($r = 0.24$, $p < .0.001$), and the second examined ACEs and back pain ($r = 0.22$, $p < .0.001$) (Brown et

al., 2018; Poole et al., 2018). According to these parameters set as two-tailed, a total sample size ranging from 106 to 159 individuals would be appropriate for our planned analyses. The recommended sample size for the current study was determined to provide sufficient power for the detection of statistical significance.

Inferential Statistics

Covariates

Covariates were included in all the multiple regression models to examine our effects of interest more precisely. Age. Subjects self-reported on their current age. Sex. Subjects self-reported on their biological sex.

Depression. The Center for Epidemiological Studies-Depression scale is a 20-item questionnaire which allows for self-report of depressive symptoms. 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. Race. Subjects identified their own racial group by selecting from the following categories: (1) Black or African American, (2) White, Caucasian, or European, (3) Asian or Asian American, (4) American Indian/Alaskan Native, (5) Native Hawaiian or Other Pacific Islander, (6) Multiracial, or (7) Other.

Body mass index (BMI). Height and weight measurements were collected as part of the baseline visit. These measurements were then used to calculate BMI for each participant (weight in kilograms divided by the square of height in meters).

LBP 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. Subjects 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".

Hypothesis 1

Moderated multiple regression models were used to determine the associations between ACEs, pain at rest, movement-evoked pain, endogenous pain modulation profiles (TS and CPM) and indicators of SES in people with cLBP. We ran two models for each pain assessment method. In the first model, we used an objective indicator of SES (ADI) to moderate the association. In the second model, we used a subjective indicator of SES (cSSS) to moderate the association.

Hypothesis 2

Moderated multiple regression models were used to determine the associations between ACEs, DER, and indicators of SES in people with cLBP. ACEs was set as the independent variable and DER as the dependent variable. In the first model, we used an objective indicator of SES (ADI) to moderate the association. In the second model, we used a subjective indicator of SES (cSSS) to moderate the association.

Hypothesis 3

Moderated-mediation multiple regression analyses involve the integration of three models in the form of a conditional process model. The first model involved a simple mediation which estimated the total and direct effect of the independent variable (X; ACEs)

on the dependent variable (Y; pain severity), as well as the indirect effect of the independent variable on the dependent variable through a mediator variable (M; DERs). The second model was a simple moderation analysis assessing whether the relationship between X and Y is moderated by a new variable (W; ADI/cSSS). The third analysis examined the extent to which X differentially influences Y as determined by W, while holding M constant. Finally, the three basic models were integrated into a coherent conditional process model (**Figure 2**).

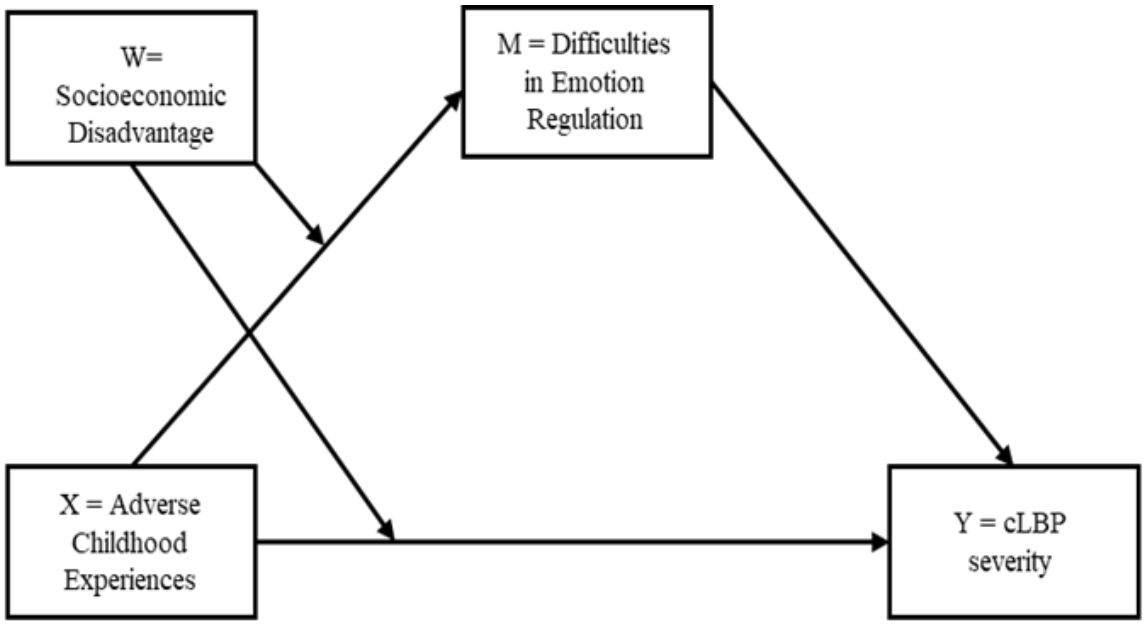

Figure 2. Conceptual diagram of the integrated conditional process model (Aim 3).

RESULTS

Data Inspection and Missing Data

Data was collected from 186 participants with cLBP. Outliers (n = 1) and individuals who were never administered the ACEs questionnaire were deleted listwise, which resulted in a final sample size of n = 183 cLBP participants. Missingness on ACE variable was addressed by imputation of '0' for individual missing items. 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%.

Hot deck data imputation method was used to ensure complete study data for participants on all remaining variables of interest (i.e. DERs, CESD, cSSS, ADI, and pain dependent variables). The imputation involved replacing missing values with the values from a "donor" participant that matched the "donee" in researcher-selected categories called "deck variables". Hot deck imputation has been found to be advantageous as it allows for retention of the whole sample and statistical power. Furthermore, the imputed values are typically realistic and do not fall outside of the range of possible values (Myers, 2011). The deck used for imputation in this study comprised of three dichotomous variables: race (NH Black or NH White), sex (male or female), and high-school degree (yes or no).

Assumption Testing

Assumption testing was carried out by recreating PROCESS moderation models in SPSS. The CPM variable contained one outlier positioned 3 standard deviations above the mean which contributed to a kurtotic distribution; this case was deleted listwise prior to

data analyses. PROCESS parameters were set such that models would be robust to violations in assumptions of normality and homoscedasticity, respectively. 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 (Hayes & Cai, 2007). 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.

Demographics and Clinical Characteristics

Sample characteristics are displayed in **Table 1**. The average age for 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 reported their annual household income to be between $0 and $9,999 (16.4%). 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%).

Table 1

Sample Characteristics

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

	$10,000-14,999	15	8.2
	$15,000-19,999	17	9.2
	$20,000-24,999	7	3.8
	$25,000-29,999	9	4.9
	$30,000-34,999	12	6.5
	$35,000-39,999	6	3.3
	$40,000-44,999	4	2.2
	$45,000-49,999	7	3.8
	$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
	Temporarily off work	2	1.1
	Unemployed, looking for work	10	5.5
	Unemployed, not looking for work	8	4.4
	Retired	10	5.5
	Permanent disability	24	13.1
	Temporary disability	1	0.5
	Full-time student	9	4.9
	Part-time student	1	0.5
	Homemaker	5	2.7
	Never worked	1	0.5
	Other	5	2.7
Highest Education	Partial high school	10	5.5
	High school graduate	33	18.0
	Partial college	59	32.2
	College/university graduate	38	20.8
	Grad/professional training	43	23.5
Marital Status	Married	68	37.2
	Widowed	5	2.7
	Divorced	22	12.0
	Separated	3	1.6
	Never Married	50	27.3
	Living with Partner	20	10.9
	*LTR not co-habiting	5	2.7
	Other	10	5.5

Notes: *LTR = long term relationship.

Descriptive characteristics for primary study variables are displayed in **Table 2**. The average BMI was 31.16 (*SD* = 6.90) for participants, with a range of 18.88 to 53.49. The average number of ACEs was 1.91 (*SD* = 1.90), with a range of 0 to 9. The most endorsed type of ACE was 'parent divorce/separation' (51.4%) followed by 'household member was mentally ill' (25.1%), 'household member had problematic drinking' (24.6%), 'emotional/psychological abuse' (23.0%), 'no love in the household' (21.3%), 'physical abuse' (18.0%), 'sexual abuse' (16.9%), 'household member went to prison' (13.7%), 'witnessed domestic violence toward mother' (12.0%), and 'neglect' (7.1%). The average score for difficulties in emotion regulation 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 50. The average state-level ADI score was 5.17 (*SD* = 3.26), with a range of 0 to 10. The average cSSS score was 6.05 (*SD* = 2.07), with a range of 0 to 10. Average movement-evoked pain severity was 28.11 (*SD* = 25.91) with a range of 0 to 95. Average pain severity at rest on the BPI-SF was 4.24 (*SD* = 2.43), with a range of 0 to 10. Average temporal summation Δ change was 18.54 (*SD* = 18.74) with a range of -10.0 to 95.0. Average conditioned pain modulation percent change was 9.78 (*SD* = 29.91), with a range of -45.0 to 131.30. 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.

Bivariate correlations between primary study variables are presented in **Table 3.** There was a weak, positive correlation between ACEs and DERs (*r* = .28) as well as between ACEs and CES-D (*r* = 0.25); both associations were statistically significant (*p* = .01). Notably, ACEs were not associated with any pain outcomes (PAR, MEP, TS, or CPM). DERs demonstrated moderate associations with cSSS (*r* = - 0.32) and PAR (*r* = 0.34), and a weak association with MEP (*r* = 0.29); all relationships were statistically significant (*p* = 0.01).

Table 2

Primary Study Variables

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
Socioeconomic Variables			
State-level ADI		5.17	3.26
Community SSS		6.05	2.07
Pain Variables			
PAR		4.24	2.43
MEP		28.11	25.91
TS effect		18.54	18.74
CPM effect		9.78	29.91
		n	***%***
Duration cLBP*	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
Pain medication*	Yes	120	65.6
	No	63	34.4

Notes: *denotes a covariate. ACEs = adverse childhood experiences; DERs = difficulties in emotion regulation scale; CES-D = center for epidemiological studies depression scale; ADI = area deprivation index; SSS = subjective social status; PAR = pain at rest; MEP = movement-evoked pain; TS = temporal summation; CPM = conditioned pain modulation; cLBP = chronic low back pain.

Table 3

Bivariate Correlations of Primary Study variables.

	1 ACES	**2** DERS	**3** CES-D	**4** sADI	**5** cSSS	**6** PAR	**7** MEP	**8** TS	**9** CPM
1	-								
2	0.28**	-							
3	0.25**	0.66**	-						
4	0.06	0.11	0.20**	-					
5	0.01	-0.32**	-0.32**	0.02	-				
6	0.11	0.34**	0.41**	0.31**	-0.16*	-			
7	0.05	0.29**	0.29**	0.27**	-0.15*	0.74**	-		
8	-0.01	-0.01	0.01	-0.04	-0.90	0.12	0.18*	-	
9	0.01	0.11	0.18*	0.04	-0.15	0.11	0.12	0.19**	-

Notes: *Correlation is significant at the 0.05 level (2-tailed). **Correlation is significant at the 0.01 level (2-tailed). Bivariate correlations between variables reflect Pearson's R. ACEs = adverse childhood experiences; DERs = difficulties in emotion regulation scale; CES-D = center for epidemiological studies depression scale; sADI =

state-level area deprivation index; cSSS = community-level subjective social status; PAR = pain at rest; MEP = movement-evoked pain; TS = temporal summation; CPM = conditioned pain modulation.

Inferential Statistics

Specific Aim 1

Hypothesis 1: Greater number of ACEs will be associated with greater severity of cLBP in adulthood (↑PAR, ↑MEP) and a pro-nociceptive endogenous pain modulation profile (↑TS, ↓CPM), particularly for individuals with lower SES (↓SSS and ↓ADI).

Pain At Rest. A moderation test was run with ACEs as the independent variable, PAR as the dependent variable, and sADI as a moderator. There were no significant main effects of sADI or ACEs, and no significant interaction of these variables upon PAR (B = -0.02, CI [-0.08, 0.04], p = 0.59). A second moderation test was run cSSS as a moderator. There were no significant main effects of cSSS or ACEs nor interaction effects upon PAR (B = 0.00, CI [-0.08, 0.08] p = 0.92).

MEP. A moderation test was run with ACEs as the independent variable, MEP as the dependent variable, and sADI as a moderator. There were no significant main effects of sADI or ACEs, and no significant interaction of these variables upon MEP (B = -0.45, CI [-1.12, 0.223], p = 0.19). A second moderation test was run cSSS as a moderator. There was a significant main effect of cSSS upon MEP (B = -2.22, CI [-4.31, -0.13], p = 0.04) and no main effect of ACEs. There was no significant interaction of these variables upon MEP (B = -0.30, CI [-0.82, 1.41], p = 0.60).

TS. A moderation test was run with ACEs as the independent variable, TS as the dependent variable, and sADI as a moderator. There were no significant main effects of sADI or ACEs, and no significant interaction of these variables upon TS (B = 0.15, CI [-

0.34, 0.64], $p = 0.55$). A second moderation test was run cSSS as a moderator. There were no significant main effects of cSSS or ACEs nor interaction effects upon TS ($B = -0.60$, CI [-1.42, 0.24], $p = 0.16$).

CPM. A moderation test was run with ACEs as the independent variable, CPM as the dependent variable, and sADI as a moderator. There were no significant main effects of sADI or ACEs on CPM. There was a significant interaction between ACEs and sADI in relation to CPM ($B = 1.00$, CI [0.30, 1.71] $p = 0.01$). Participants with low sADI experienced a significant effect of ACEs on CPM ($B = -5.21$, CI [-8.66, -1.76], $p < 0.01$), such that greater ACEs was associated with a less efficient CPM response for those individuals with low neighborhood deprivation. Conversely, ACEs were not significantly associated with CPM for those with average ($B = 1.20$, CI [-3.54, 1.14], $p = 0.31$) or high sADI ($B = 2.81$, CI [-1.04, 6.66], $p = 0.15$). **Figure 3** displays the interaction effect of ACEs and sADI upon CPM. A second moderation test was run using cSSS as a moderator. There were no significant main effects of cSSS or ACEs nor interaction effects upon CPM, $B = -0.06$, CI [-1.51, 1.39], $p = 0.94$.

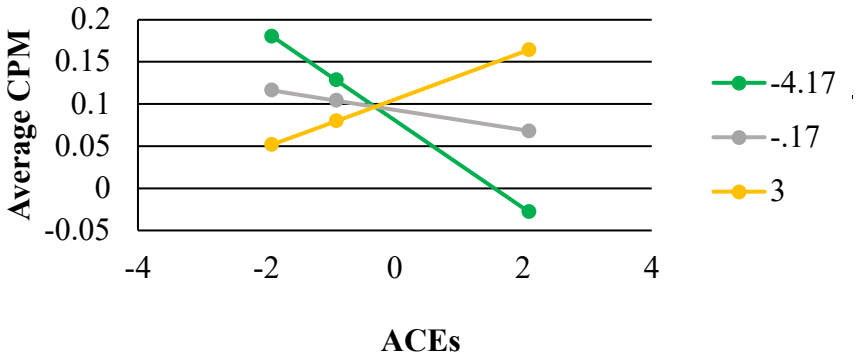

Figure 3. Interactional Effect of ACEs and sADI on CPM.
The following variables were mean centered prior to analysis and graphing: sADI and ACES. Moderator values (sADI) are the 16th, 50th, and 84th percentiles.

Specific Aim 2

Hypothesis 2: Greater number of ACEs will be associated with greater difficulties in emotion regulation, particularly for individuals with lower SES (↓SSS and ↓ADI).

DERS. A moderation test was run with ACEs as the independent variable, DERs as the dependent variable, and sADI as a moderator. Findings revealed that ACEs was significantly and positively associated with DERs (B = 1.60, t = 2.21, p = 0.03), suggesting that greater childhood adversity is related to greater difficulties with emotion regulation in adulhood for people with cLBP. The main effect of sADI (B = 0.12, t = 0.26, p = 0.80) as well as the effect of the interaction between ACEs and sADI (B = -0.08, t = -0.34, p = 0.74) on DERs were not significant. A second moderation test was run with cSSS as a moderator. The overall model fit analyzing the effect of ACEs on DERs remained significant (R^2 = 0.52, F(10, 172) = 14.11, p < 0.01), which was primarily driven by the significant association between ACEs and DERS. However the main effect of cSSS (B = -0.82, t = -1.31, p = 0.19) and effect of the interaction between ACEs and cSSS (B = -0.52, t = -1.18, p = 0.24) on DERs were not significant.

Specific Aim 3

Hypothesis 3: Greater difficulties with emotion regulation will help explain the relationships between ACEs and cLBP severity (↑PAR, ↑MEP) as well as a pro-nociceptive endogenous pain modulation profile (↑TS, ↓CPM), particularly for individuals with lower SES (↓SSS and ↓ADI).

Pain At Rest. A moderated-mediation model was conducted with ACEs as the independent variable, PAR as the dependent variable, DERs as the mediator, and sADI as a moderator. The overall model fit analyzing the effect of ACEs on PAR was significant (R^2 = 0.34, F(11, 171) = 8.60, p < 0.01). The main effect of ACEs (B = -0.02, t = -0.21, p

= 0.51) and sADI on PAR was not significant (B = 0.09, t = 1.67, p = 0.10). The effect of the interaction between ACEs and sADI on PAR was not significant (B = -0.15, t = -0.50, p = 0.62). The indirect effect of ACEs on PAR through DERs was not statistically significant (B = 0.02, t =1.77, p = .08). Finally, the index of moderated mediation had a value of -0.001 that was not statistically significant (95% CI [-0.01, 0.01]).

A second moderated-mediation model was conducted with cSSS as a moderator. The overall model fit analyzing the effect of ACEs on PAR was significant (R^2 = 0.33, F(11, 171) = 8.94, p < 0.00). The main effects of ACEs (B = -0.01, t = -0.75, p = 0.45) and cSSS on PAR were not significant (B = -0.12, t = -1.17, p = 0.25). The effect of the interaction between ACEs and cSSS on PAR was not significant (B = 0.12, t = 0.39, p = 0.70). The indirect effect of ACEs on PAR through DERs was not statistically significant (B = 0.02, t =1.73, p = .08). Finally, the index of moderated mediation had a value of -0.01 that was not statistically significant (95% CI [-0.04, 0.006]).

MEP. A moderated-mediation model was conducted with ACEs as the independent variable, MEP as the dependent variable, DERs as the mediator, and sADI as a moderator. The overall model fit analyzing the effect of ACEs on MEP was significant (R^2 = 0.29, F(11, 171) = 6.83, p < 0.01). The main effect of ACEs (B = -0.80, t = -0.72, p = 0.47) and sADI on MEP was not significant (B = 0.79, t = 1.36, p = 0.17). The effect of the interaction between ACEs and sADI on MEP was not significant (B = -0.42, t = -1.30, p = 0.19). The indirect effect of ACEs on MEP through DERs was statistically significant (B = 0.34, t =2.48, p = 0.01). Finally, the index of moderated mediation had a value of -0.03 that was not statistically significant (95% CI [-0.16, 0.13]).

A second moderated-mediation model was conducted with cSSS as a moderator. The overall model fit analyzing the effect of ACEs on MEP was significant (R^2 = 0.29, $F(11, 171) = 6.86$, $p < 0.01$). The main effect of ACEs (B = -0.65, t = -0.64, p = 0.52) and cSSS on MEP was not significant (B = -1.95, t = -1.85, p = 0.07). The effect of the interaction between ACEs and cSSS on MEP was not significant (B = 0.47, t = 0.89, p = 0.37). The indirect effect of ACEs on MEP through DERs was statistically significant (B = 0.33, t =2.47, p = 0.01). Finally, the index of moderated mediation had a value of -0.17 that was not statistically significant (95% CI [-0.51, 0.08]).

TS. A moderated-mediation model was conducted with ACES as the independent variable, TS as the dependent variable, DERs as the mediator, and sADI as a moderator. The overall model fit analyzing the effect of ACEs on TS was non-significant (R^2 = 0.29, F(11, 171) = 1.40, p = 0.18). The main effect of ACEs (B = 0.11, t = 0.13, p = 0.90) and sADI on TS was not significant (B = - 0.47, t = -0.86, p = 0.39. The effect of the interaction between ACEs and sADI on TS was not significant (B = 0.15, t = 0.59, p = 0.55). The indirect effect of ACEs on TS through DERs was not statistically significant (B = 0.00, t = 0.00, p = 1.00). Finally, the index of moderated mediation had a value of 0.00 that was not statistically significant (95% CI [-0.05, 0.05]).

A second moderated-mediation model was conducted with cSSS as a moderator. The overall model fit analyzing the effect of ACEs on TS was not significant (R^2 = 0.10, $F(11, 171) = 1.55$, p = 0.12). The main effect of ACEs (B = 0.22, t = 0.27, p = 0.78) and cSSS on TS was not significant (B = -1.01, t = -1.10, p = 0.27). The effect of the interaction between ACEs and cSSS on TS was not significant (B = -0.61, t = -1.39, p = 0.17. The indirect effect of ACEs on TS through DERs was not statistically significant (B = -0.04, t

=-0.38, p = 0.71). Finally, the index of moderated mediation had a value of 0.02 that was not statistically significant (95% CI [-0.13, 0.16]).

CPM. A moderated-mediation model was conducted with ACEs as the independent variable, CPM as the dependent variable, DERs as the mediator, and sADI as a moderator. The overall model fit analyzing the effect of ACEs on CPM significant (R^2 = 0.10, F(11, 171) = 2.17, p = 0.02). The main effect of ACEs (B = -1.02, t = -0.87, p = 0.38) and sADI on CPM was not significant (B = 0.31, t = 0.37, p = 0.71. The effect of the interaction between ACEs and sADI on CPM was significant (B = 1.00, t = 2.81, p = 0.01). The indirect effect of ACEs on CPM through DERs was not statistically significant (B = -0.00, t = -0.01, p = 0.99). Finally, the index of moderated mediation had a value of 0.00 that was not statistically significant (95% CI [-0.07, 0.07]).

A second moderated-mediation model was conducted with cSSS as a moderator. The overall model fit analyzing the effect of ACEs on CPM was not significant (R^2 = 0.06, F(11, 171) = 0.97, p < 0.47). The main effect of ACEs (B = -0.64, t = -0.47, p = 0.63) and cSSS on CPM was not significant (B = -1.19, t = -0.91, p = 0.36). The effect of the interaction between ACEs and cSSS on CPM was not significant (B = -0.08, t = -0.10, p = 0.92. The indirect effect of ACEs on CPM through DERs was not statistically significant (B = -0.04, t =-0.20, p = 0.84). Finally, the index of moderated mediation had a value of 0.02 that was not statistically significant (95% CI [-0.19, 0.25]).

DISCUSSION

The goal of the current study was to examine potential associations among ACEs, emotion regulation, and pain-related responses (i.e., pain-at-rest, movement-evoked pain, and endogenous pain modulation) in a sample of patients living with non-specific cLBP. The present study further sought to determine the extent to which SES moderated associations between (1) ACEs and pain, (2) ACEs and DERs, and (3) the mediation of the ACEs → DERS → pain. At present, there remains a paucity of studies aiming to elucidate the impact of social determinants of health (e.g., adverse interpersonal experiences, socioeconomic factors) on cLBP related outcomes (Karran et al., 2020; Yap et al., 2022). Individuals from minoritized racial/ethnic groups and lower socioeconomic position continue to experience inequities in cLBP outcomes. Addressing these disparities requires a comprehensive approach that examines the social context within which pain occurs and develops. Further, there remains a need for cLBP studies that incorporate movement-evoked pain assessments into their protocols. The preexisting literature would also benefit substantially from better understanding the complex descending inhibitory and facilitatory processes within the central nervous system that regulate pain perception in non-specific conditions such as cLBP. Thus, we hypothesized that there are significant associations among ACEs, emotion regulation, and adult cLBP, as well as to determine whether associations vary by SES.

Specific Aim 1

The primary objective of aim 1 was to examine the potential associations among ACES, cLBP severity (MEP, PAR) and endogenous pain modulation (TS, CPM). Additionally, aim one sought to examine whether socioeconomic status (ADI and SSS) mediated these associations. Findings did not support our hypotheses. Specifically, no significant associations were observed between ACEs and either of cLBP severity measures (MEP, PAR). However, the results revealed that greater ACEs is significantly associated with less efficient CPM, but only for those with low sADI.

Prior research found cLBP pain was greater among individuals from disadvantaged communities, indicating a positive relationship with ADI ((Rumble et al., 2021) We found the ADI to be positively associated with PAR and MEP, however associations with endogenous pain modulation appear more complicated. In our cLBP sample, ACEs were associated with decreased CPM for individuals residing in areas of low deprivation. In other words, the relationship between ACEs and impaired pain inhibition was strongest for those living in a more advantaged neighborhood. This finding is consistent with prior research by You and Megaher (2016) in a sample of healthy college students with history of childhood adversity. More specifically, the investigators identified a pain phenotype characterized by heightened central sensitization in the high adversity group. These individuals displayed increased pain facilitation via greater sensitization to temporal summation of second pain and a trend towards slower dissipation of this sensation. Our findings contribute to a growing body of literature that childhood adversity may shift inhibitory and facilitatory pain mechanisms towards central sensitization.

Notably, the effect of ACEs upon CPM was weakest for the portion of our sample residing in the most deprived neighborhoods. People from disadvantaged backgrounds may

have numerous psychosocial stressors that hinder CPM, making it difficult to assess the specific impact of ACES on CPM over and above other stressors. Being poor in the United States is associated with a plethora of stressors in addition to ACEs; poor sleep, inadequate housing, difficulty paying utilities, crime victimization and violence, manual labor, food deserts, and less education (Federman et al., 1996). In addition, individuals living in greater socioeconomic disadvantage have (1) limited control over their circumstances, (2) cannot predict how long the stressors will persist, and (3) have limited resources, like social support, to combat these stressors (Sapolsky, 2005). In contrast, individuals in more privileged neighborhoods may be less affected by psychosocial stressors, enabling a more tangible association between a history of ACEs and impaired CPM in adulthood among individuals with cLBP in this sample.

Research on healthy controls and animal models suggest that 'latent sensitization' provides an additional explanation (Rhudy & Hellman, 2022). Such studies demonstrate that spinal sensitization and impaired descending inhibition of spinal nociception is associated with exposure to adversity and number of adverse experiences. Under lower adversity exposure, there is little to no spinal sensitization and inhibitory mechanisms remain intact. Meanwhile, chronic exposure to adverse life experiences may enhance spinal sensitization, however compensatory pain inhibition pathways (supraspinal) keep this sensitization suppressed (lack of an effect on pain inhibition) (Kell et al., 2021). In our sample, it may be that living in socioeconomic disadvantage captures chronic exposure to adversity.

Finally, psychological resilience may also explain the weak association between ACEs and CPM for those subjected to greater neighborhood deprivation. Psychological

resilience is characterized by behavioral perseverance and cognitive/affective positivity. It has been found that many people living in poverty are able to accomplish their goals despite facing significant hardship. Enduring socioeconomic hardship may force individuals to become cognitively, affectively, and behaviorally flexible, as they must find creative ways to navigate their circumstances. Therefore, resilience may serve as an individual-level strength and buffer for these individuals, allowing them to adapt to their environment. Though few studies have examined pain resilience and pain in experimental settings, there appears to be a general pattern in which pain resilience is related to reduced pain when the stimulus is prolonged or repeated (Ankawi et al., 2020). Notably, research has demonstrated a greater effect of psychological resilience on experimental pain outcomes in participants characterized by high-stress (Friborg et al., 2006).

We were not able to find direct associations between ACEs and chronic pain using measures of PAR, MEP, TS, or CPM within our cLBP sample. This finding is not entirely inconsistent with those of previous studies as the literature has been mixed (Bussières et al., 2020). For example, a study by Craner et al. (2022) reported significant group differences in pain ratings based on the number of ACEs; more specifically the investigators found participants reporting ≥4 ACEs to endorse greater pain compared to participants with 0-1 ACE. On average, the participants within our cLBP sample reported experiencing two ACEs, which suggests that our sample may not have experienced enough ACEs to demonstrate a direct association with cLBP severity.

In the current study, 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 PAR and MEP do not share the same relationship with ACEs as measures that capture pain of longer duration ("average pain severity for the past seven days") or measures that capture multidimensional aspects of pain (unpleasantness). A recent study by Leisner et al. (2014) 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. 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.

Overall, the research relating ACEs to chronic pain remains mixed. A study examining endogenous pain modulation and ACEs in non-specific cLBP found that a history of childhood maltreatment was associated with altered pressure pain thresholds and increased TS in adult patients (Tesarz et al., 2016). The relationship between ACEs and spinal sensitization is also present in research focused on healthy controls. A recent study demonstrated history of sexual assault to be associated with lower pain thresholds, increased cool-detection sensitivity, and higher ischemia pain tolerance in healthy controls (Hellman et al., 2018; Hellman et al., 2019). Granot et al. (2011) identified paradoxical relationships between sexual abuse and experimental pain testing: abuse was associated with higher heat pain thresholds, though ratings of painful stimuli were higher and survivors were more likely to terminate painful stimuli. Fillingim and Edwards (2005) found no group differences in heat and ischemia pain threshold and tolerance or TS-pain

to heat in college students with and without a history of childhood abuse. Interestingly, participants with a history of abuse reported hypoalgesia during the TS-pain procedure.

Specific Aim 2

The primary objective of aim 2 was to examine associations among ACEs, difficulties with emotion regulation and objective, as well as subjective indicators of SES in people with cLBP. We hypothesized that a greater number of ACEs would be associated with greater difficulties in emotion regulation, particularly for individuals with lower SES (↓SSS and ↓ADI). Our hypotheses were partially supported. In our sample, greater number of ACEs were uniquely associated with greater difficulties in emotion regulation as an adult with cLBP; however, this association was not moderated by SES. Our findings align with a wealth of literature showing robust associations of ACEs and impairments in emotion regulation that are sustained into adulthood (McCrory et al., 2017).

Further, socioemotional development begins when primary attachments are formed with a child and its parents/caregivers (Gross, 2015). 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 (Morris et al., 2007). 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. Importantly, the context in which development occurs can help or hinder this process depending on the stage of psychological, neurological, and biological maturation (Cole, 2014).

A wealth of literature has demonstrated how ACEs can interrupt the development of emotional processes and lead to deficits in emotion regulation that last a lifetime (Loman & Gunnar, 2010). More specifically a history of childhood adversity is associated with greater use of suppression, rumination, higher emotional lability, greater emotional intensity, and greater negative affect later in life (Milojevich et al., 2020). Disruptions to emotion regulation comprise a transdiagnostic risk process for a plethora of psychopathological conditions (Cludius et al., 2020). Moreover, emotion dysregulation has been identified as a mediator of associations between childhood maltreatment and conditions such as: depression, self-harm, posttraumatic stress disorder, substance use, and eating disorders (Burns et al., 2010; O'Mahen et al., 2015; Peh et al., 2017). Importantly, emotion regulation contributes to the subjective experience of pain by modulating perceptions of pain intensity, appraisals and interpretations of pain, and how individuals cope with their condition (Koechlin et al., 2018). Chronic pain patients have been found to have increased difficulty identifying, differentiating, and regulating emotions, as well as selecting emotion regulation strategies(Aaron et al., 2020).

We did not find support for the hypothesis that the association between ACEs and emotion dysregulation would be strongest for individuals from low socioeconomic backgrounds, however a statistically significant negative association was present between DERs and SSS. Research has identified neurobiological mechanisms through which socioeconomic disadvantage may impact emotion regulation. Kim et al. (2016) found that perceived stress associated with socioeconomic disadvantages may contribute to reduced neural responses to infant cry - a proxy for understanding level of attunement towards the child. The study suggests that subjective perception of socioeconomic disadvantage can

explain reductions in caregiver sensitivity to infants which contribute to atypical attachment and, ultimately, result in disrupted development/acquisition of key abilities – including emotion regulation.

Notably, the association between DERs and SES was not evident using SADI. This further substantiates the necessity of research examining both subjective and objective measures of SES, and underscores a difference in their associations with psychological variables. Research examining SSS demonstrates its unique ability to speak to the internalized reality of an individual's socioeconomic condition by capturing feelings of relative deprivation, opportunity, and social mobility. Our findings suggest that the perceived implications of disadvantage may be more closely tied to how an individual regulates their emotions as opposed to objective indicators of their neighborhood socioeconomic context.

Specific Aim 3

The primary objective of aim 3 was to determine if difficulties with emotion regulation helped explain the relationship between ACEs and adult cLBP, and whether relationship is affected by SES. We hypothesized that greater difficulties with emotion regulation would mediate the relationship between ACEs and cLBP severity (↑PAR, ↑MEP) as well as a pro-nociceptive endogenous pain modulation profile (↑TS, ↓CPM), particularly for individuals with lower SES (↓SSS and ↓ADI). Our hypotheses were partially supported.

We found direct associations between DERs and pain severity (PAR and MEP), which aligns with previous literature examining pain intensity and emotion regulation. For example, a study by Le Borgne et al. (2017) found that lack of emotion regulation and

awareness were associated with worse pain outcomes in a sample of individuals with work-related cLBP. A more recent systematic review identified emotion dysregulation as a key risk factor in the development and experience of chronic pain (Koechlin et al., 2018). The specific direct association of DERs and MEP within our sample corroborates a wealth of preexisting studies grounded in the fear-avoidance model of musculoskeletal pain. This model highlights how poor emotion regulation and maladaptive cognitive frameworks contribute to pain-related fear and, ultimately, activity/movement avoidance. Over time, the avoidance of activity/movement leads to increased disability due to atrophy and exacerbates this cycle (Slepian et al., 2019).

We were unable to find direct associations between DERs and aspects of endogenous pain modulation measured via QST (TS and CPM). The previous literature examining the relationships between psychological factors and endogenous pain modulation is limited and mixed. A metaanalysis by Nahman-Averbuch et al. (2016) examining the CPM response found no associations with depression, anxiety, or pain catastrophizing in patients and healthy controls. One study examining temporal summation of second pain (TSSP) in 79 healthy individuals with varying degrees of borderline features found greater TSSP was accounted for by negative relationships rather than the emotion dysregulation (You & Meagher, 2017). It is possible that assessment methods requiring overt consideration of one's pain experience may capture emotional and psychological aspects of coping more easily than paradigms focused on endogenous processes. Further research is necessary to truly parse out the role of emotion regulation on psychophysiological aspects of cLBP.

Our data further revealed that the relationship between ACEs and MEP was significantly mediated by DERs. Contrary to our hypotheses, this mediation effect did not differ in strength or magnitude according to objective or subjective indicators of SES (i.e., lack of moderation). Emotion dysregulation has previously been established as a transdiagnostic risk factor connecting ACEs and later psychopathology; our study extends this research to chronic pain. Our findings align with similar research in chronic pain. A study by Garland et al. (2019) found female patients with chronic pain exposed 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 trauma, emotion regulation, and pain in individuals with alcohol use disorder (Zaorska et al., 2020). 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.

There has been less research examining the role of maladaptive psychological determinants in MEP; studies broadly report higher levels of negative affect and dysfunctional cognitive-affective coping with MEP (Palit et al., 2020). Our findings contribute to a growing body of evidence suggesting distinct underlying mechanisms for PAR and MEP (Landmark, et al., 2011; Rakel & Frantz, 2003). Given that movement contributes substantially to pain and disability for cLBP, it is possible that assessment of MEP captures more psychological distress relevant for emotion regulation compared to

pain measured at rest (Litcher-Kelly, et al., 2007). The results of the current study and others highlight the importance of incorporating pain assessment measures which consider the context of pain.

Implications

The present study provides further support for the link between ACEs and chronic pain, underscoring the importance of childhood maltreatment and impaired emotional regulation as a cross-cutting risk factor for psychopathological conditions, including cLBP (McLaughlin et al., 2020). Moreover, the results provide multiple targets for a more comprehensive approach to examining and treating cLBP.

ACEs. A topical review by Tidmarsh et al. (2022) found ACEs to be associated with greater pain complications (i.e. more pain symptoms, worse functional outcomes, higher prevalence of chronic pain conditions), pain catastrophizing and depression which, together, heighten the risk of patient attrition. Medical providers may be able to improve analgesia for these patients by incorporating a trauma-informed approach to their practice. Further, instituting ACE screenings as a part of routine practice may allow clinicians to identify and support at-risk patients. At current, cognitive behavioral therapy (CBT) is the most common evidence-based psychological treatment for chronic pain (Stewart et al., 2015). This is relevant because CBT for chronic pain involves helping individuals identify and modify negative thoughts and behaviors that may exacerbate their pain, while also teaching them coping skills and relaxation techniques. Directly addressing ACEs may increase the clinical benefit for CBT for individuals with cLBP. There is support for CBT, relaxation, and journaling focused on adverse life circumstances to reduce spinal

sensitization and increase inhibition of spinal nociception (Emery et al., 2006; Salomons et al., 2014; You et al., 2014).

Emotion Regulation. There is a substantial amount of evidence that suggests emotion-regulation skills are involved in the development, maintenance, and treatment of psychopathology (Le Borgne et al., 2017). Within psychotherapy, deficits in emotion regulation are addressed by cultivating skills in mindfulness, attention shifting/distraction, distress tolerance, and cognitive reframing. A review by Russell and Park (2018) found that mindfulness interventions showed the most consistent improvements, while evidence for attention shifting/distraction was less consistent. There was also evidence supporting the use of cognitive reframing from studies using randomized control group comparisons. Ultimately, interventions that combine multiple emotion regulation skills may most effectively improve pain outcomes (Zautra et al., 2008).

Socioeconomic Context. The current study demonstrates that greater neighborhood deprivation is associated with worse pain severity and can shape the association between early life adversity and endogenous pain modulation in adults with cLBP. Community level deprivation can be captured by economic instability, poor education, low social cohesion, high levels of crime/violence, lack of transportation, environmental risks, food deserts, and limited access to healthcare (Gaskin et al., 2019; Ross & Mirowsky, 2001). These issues span economic, social, and physical environments, thus efforts to address neighborhood and community socioeconomic deprivation requires the collaborative efforts of government, healthcare providers, community-based organizations, and stakeholders. In our cLBP sample, the average sADI for individuals overall was 5.17 (SD = 2.6), yet when this association is parsed by race, significant disparities emerge: the average sADI score

for individuals identifying as non-Hispanic white was 3.3 (SD = 2.5), while for those identifying as non-Hispanic Black, the average sADI score was 6.2 (SD = 3.1). These sADI disparities suggest that addressing the intersection of race and socioeconomic disadvantage is crucial for effectively managing and preventing chronic low back pain. Policymakers and healthcare professionals should prioritize interventions that target communities facing multiple forms of disadvantage, such as non-Hispanic Black individuals living in highly deprived neighborhoods.

Limitations

This study may have been affected by several limitations. The present study focuses primarily on a psychological pathway through which ACEs may contribute to cLBP pain, however this may not fully account for the ACEs-pain risk relationship. Studies demonstrate that ACE-pain relationships remain above and beyond the influence of psychological consequences (Sturycz et al., 2019; You et al., 2019). However, studying these relationships within chronic pain populations can be convoluted given the effect of ACEs upon pain processing which may be masked by disease processes (Rhudy & Hellman, 2022). Another limitation of the current study is the fact that the dependent and independent variables were assessed and examined at the same time. Moreover, participants were asked to report on whether they experienced ACEs in their childhood, a process that may have been affected by retrospective recall bias. Though our mediation model was theoretically based and may extend our understanding of chronic pain etiology, the subsequent conclusions remain bound by limitations of the statistical design. Cross-sectional designs are, at their core, snapshots of relationships in time. As a result, we cannot attribute causal relationships between variables of interest. There are limitations regarding

the assessment of ACEs in this study. For example, analyses relied on retrospective self-reporting, however research indicates these often disagree with prospective reports. 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, the study is limited regarding generalizability of findings; our sample focused exclusively on cLBP and only recruited non-Hispanic Black and White individuals. Future research should incorporate longitudinal designs with multiple timepoints to address temporal precedence, and more diverse samples to replicate these findings in other chronic pain conditions.

Conclusion

Chronic low back pain (cLBP) is a prevalent and 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 childhood maltreatment, may act as a psychological pathway through which ACEs contributes to cLBP in adulthood. Moreover, the study highlights the role of neighborhood-level deprivation in exacerbating pain severity among individuals with cLBP. Importantly, the study suggests that early life adversity and neighborhood deprivation can interact to affect pain modulation in individuals with cLBP. Taken together, these findings underscore the need for a biopsychosocial approach to chronic pain management that takes into account not only the physical contributors but also psychological and social factors.

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