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Recalled early life adversity and pain: the role of mood, sleep, optimism, and control

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Abstract

Early life adversity (ELA) has been associated with pain symptomatology in adulthood, but mechanisms and moderators of these associations are unclear. Using recall based and concurrently assessed self-report data, we examined associations between ELA, mood, sleep, and recent pain intensity and interference, and whether optimism and perceived control weakened these associations in a midlife community sample of diverse adults reporting some ELA. Controlling for demographic variables and BMI, higher levels of ELA were associated with more pain intensity and interference; greater sleep disturbance and negative mood accounted for these associations. When moderation was examined, only the path from sleep disturbance to pain interference was significantly attenuated for those with higher optimism and higher perceived control. These findings suggest that higher levels of ELA may link with pain in adulthood through poorer mood and sleep, and that resilience resources such as optimism and control may buffer some of these pathways.

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Compliance with ethical standards

Human and animal rights and Informed consent All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Conflict of interest The authors Ambika Mathur, Jennifer E. Graham-Engeland, Danica C. Slavish, Joshua M. Smyth, Richard B. Lipton, Mindy J. Katz, and Martin J. Sliwinski declare that they have no conflict of interest.

Keywords

Early life adversity; Mood disturbance; Sleep disturbance; Pain; Optimism; Perceived control and mastery; Structural equation modeling

Introduction

Physical pain is the most common symptom reported to health care providers and negatively affects the quality of life of millions of adults, even in the absence of chronic health conditions (Gatchel et al., 2007; Lumley et al., 2011; Sturgeon & Zautra, 2010). Evidence suggests that early life adversity, such as abuse or neglect in childhood, has been implicated in the incidence of pain in adulthood (Davis et al., 2004; Sachs-Ericsson et al., 2009), yet the mechanisms and moderators of this association are not well understood. Vulnerability and resilience factors may help explain connections between early adversity and future pain symptomatology. We posit that early adversity is particularly likely to contribute to pain when it leads to mood and sleep disturbances, both of which are associated with reported pain (Chapman et al., 2011; Fernandez, 2002; Lautenbacher et al., 2006; Sachs-Ericsson et al., 2009; Sachs-Ericsson et al., 2017). Further, research on resilience to stress and pain suggests that psychosocial resources related to optimism and perceived control may help some individuals maintain health and manage pain (Dunkel-Schetter & Dolbier, 2011; Sturgeon & Zautra, 2010). Our goal in the present research was to examine whether the amount of reported early adversity was linked to greater pain intensity and interference from pain among a diverse sample of adults who reported at least some degree of early adversity, and whether the vulnerability factors of mood and sleep disturbances helped explain these associations. An additional goal was to examine whether optimism and perceived control weakened the pathways between early adversity, mood, sleep, and pain.

Early life adversity and pain

Early adversity is a broad construct that can include major illnesses, accidents, parental substance abuse and/or divorce, financial issues, trauma, neglect, and physical and/or sexual abuse occurring before a child or teenager leaves home (Turner et al., 1995). Results vary regarding the contribution of early adversity to pain symptomatology in adulthood. Two reviews found that retrospective reports of childhood abuse and neglect were associated with an increase in co-occurring adversities and painful symptoms in adulthood (Davis et al., 2004; Sachs-Ericsson et al., 2009). Seemingly in contrast, an experimental study found that healthy individuals with a history of childhood abuse had decreased sensitivity to induced pain, yet increased pain complaints (Fillingim & Edwards, 2005). Two longitudinal studies have had diverse findings with regard to a connection between early adversity and pain. Jones et al. (2009) found that adversities such as having experienced institutional care, maternal death, hospitalization due to an accident, or familial financial hardship prior to age seven were associated prospectively with an increase in risk for chronic pain in adulthood. Conversely, Raphael et al. (2001) found no relationship among court-documented child abuse and neglect with later pain complaints; it could be, however, that reliance on court-documented abuse avoids issues with reporting bias but obscures the effects of unreported abuse (Davis et al., 2004). Despite inconsistencies, the literature suggests that early adversity

may render some individuals more vulnerable to pain. Thus, examination of factors that may help explain connections between early adversity and pain complaints in adulthood is warranted.

Mood, sleep, and pain

Psychological and behavioral factors, such as mood and sleep disturbances, may help explain linkages between early adversity and pain. Early adversity is strongly associated with greater negative affect and the development of mood disorders (Anda et al., 2006), both of which are related to pain symptomatology (Fernandez, 2002; Graham-Engeland et al., 2015; Lumley et al., 2011; Sachs-Ericsson et al., 2009, 2017). A greater number of adverse childhood experiences also has been associated with more sleep disturbance (Chapman et al., 2011; Kajeepeta et al., 2015), and poor sleep quality can exacerbate reported pain intensity (Lautenbacher et al., 2006; Smith & Haythorn-thwaite, 2004). Further, insomnia is often comorbid with depressive and anxiety disorders (for review, see Benca et al., 1997), and mood and sleep disturbances likely worsen pain synergistically (Strine & Chapman, 2005). Neuroendocrine alterations in stress responsive systems due to early trauma can contribute to greater pain sensitivity, as well as risk for depression, anxiety, and sleep disorders (Heim & Nemeroff, 2001; Kajeepeta et al., 2015; Sachs-Ericsson et al., 2009). Therefore, it is important to examine the role of mood and sleep disturbances concurrently in explaining associations between early adversity and pain in adulthood.

Resilience to early life adversity and pain through optimism and perceived control

A growing body of research has examined factors associated with resilience to early adversity (Cicchetti, 2013; Logan-Greene et al., 2014; Masten et al., 2004). Resilience is typically characterized as positive adaptation and maintained healthy functioning in the face of stress (for reviews, see Dunkel-Schetter & Dolbier, 2011; Southwick et al., 2014; Zautra et al., 2008). Personality and ego-related resources such as dispositional optimism and personal control have been implicated in resilience to adversity (see Chen & Miller, 2013; Dunkel-Schetter & Dolbier, 2011). Indeed, a positive future orientation, involving planning for the future and demonstrating optimism and flexibility, is considered a resilience trait (see Reich et al., 2010, p. 115). Furthermore, reframing the meaning of a stressor and adjusting oneself to uncontrollable situations (e.g., engaging in secondary control coping) has been associated with enhanced well-being (see Chen & Miller, 2013). Individuals who demonstrate resilience to pain by showing minimal behavioral impairment, effective coping strategies, and better physical functioning also tend to possess a more positive outlook on their lives and are more likely to believe that they can control their pain (Sturgeon & Zautra, 2010). Although the evidence suggests that optimism and perceived control may buffer negative psychological and physical health outcomes following early adversity, whether they specifically mitigate disturbances due to mood, sleep, and pain is not well characterized.

The current study

The present research aims to advance understanding of specific vulnerability and resilience factors implicated in the association between early adversity and pain in adulthood. We utilized a socioeconomically and ethnically diverse sample of adults with considerable variation in pain intensity and interference as well as in the frequency of early adversity. We

were interested in whether the amount of recalled early adversity would relate to the amount of mood, sleep, and pain disturbance. Thus, we limited the sample to those who reported at least some early adversity. We expected that higher levels of recalled early adversity would predict greater mood disturbance (as indexed by a latent construct composed of depressed mood, and anxiety and anger symptoms) and greater recent sleep disturbance, and that mood and sleep disturbances would predict greater pain intensity and interference. Further, we expected that mood and sleep disturbances would help explain associations between the degree of early adversity and pain. Another goal was to investigate whether optimism and perceived control would moderate the associations between early adversity, mood and sleep disturbances, and pain intensity and interference, with the expectation that these linkages would be weaker for individuals with greater optimism and perceived control.

Methods

Overview and participants

The data used for the present research were drawn from a baseline survey of a larger ongoing study that used systematic probability sampling to recruit participants from a housing cooperative in the Bronx, New York (for information on the larger study, see Scott et al., 2015). Participants in the larger study ($N = 337$) were between 25 and 65 years of age, ambulatory, fluent in English, and free of visual impairment. The baseline survey assessed demographic information and standard measures of stress, life experiences, health behaviors, and physical and mental health.

For the present research, the Childhood Traumas Scale (CTS; Turner et al., 1995) was used to assess early adversity. An item on sexual abuse and molestation replaced an item on repeating a year of school in this study. The present sample was not recruited on the basis of having experienced or reported early adversity. We restricted the sample to individuals who reported at least one adversity to increase generalizability to populations that have experienced some early adversity and to enable comparisons to past studies that have recruited on the basis of experienced or reported adversity, such as those reviewed above. The CTS asks about adverse experiences in eight different categories; 77% of the larger sample reported at least one adversity, with considerable variation: 31% reported one adversity, 29% reported two adversities, 19% reported three adversities, 10% reported four adversities, and 9% reported 5–7 adversities. A traumatizing occurrence that left the individual scared for subsequent years was the most commonly reported adversity (44%), followed by parental divorce (31%), a major illness or accident that required hospitalization and parental substance use (each 24%), sexual abuse (23%), parental unemployment (21%), and the child's removal from the home and physical abuse (each 10%). There were no significant differences in mood, sleep, the pain variables, and covariates (which were age, gender, body mass index [BMI], income, and race) between excluded participants and the resulting sample that reported some degree of childhood adversity ($N = 265$). Importantly, the associations described below were significant and in the same directions when individuals reporting no early adversity were included in the sample.

For the overall sample, the median total household income for the past year was \$40,000–\$59,999; 22% of participants were in this category, with an additional 6% having an income

of \$4999 or less, and an additional 1% having an income of \$150,000 or greater. Marital status was also diverse: 33% were never married, 30% were married, 10% were cohabiting with a partner as if married, with some divorced (13%), separated (5%), single (5%), and widowed (3%) participants.

Measures

Early life adversity—As noted earlier, a slightly adapted version of the Childhood Traumas section of Turner and colleagues' scale (1995) was used to measure early adversity. Eight items assessed whether major illness or accidents, parental divorce, unemployment, drug use, traumatic events, and physical and sexual abuse occurred during the childhood and teenage years; scores ranged from 0 to 8, with higher scores indicating a greater number of adversities. The original measure has been used in prior studies examining adverse childhood and lifetime experiences (see Seery et al., 2010).

Mood disturbance—Depressive, anxious, and angry feelings from the past week were assessed via standardized measures for emotional health from the patient-reported outcomes measurement information system (PROMIS) on a scale from 7–35 (for anxiety) and 8–40 (for depressed mood and anger), with higher scores on each scale indicating greater recent depressive symptoms, anxiety, and anger, respectively (Cella et al., 2007). These measures had high internal consistency (α 's = 0.89–0.92). The PROMIS scales measure facets of physical, mental, and social health and have demonstrated reliability, precision, and construct validity with other widely used instruments (Cella et al., 2010).

Sleep disturbance—Sleep disturbance was assessed via the PROMIS measure for sleep disturbance on a scale from 8 to 40. The eight items assessed overall sleep quality and difficulty in falling and/or staying asleep; higher scores indicated greater sleep disturbance, and the scale exhibited high internal consistency (α = 0.88; Cella et al., 2010, 2007).

Optimism—Dispositional optimism was measured via the Life Orientation Test (LOT), a widely used measure consisting of positive, negative, and filler items on a scale of 6–30, with higher scores indicating higher optimism (Scheier & Carver, 1985). This scale has acceptable internal consistency (α = 0.76) and test–retest reliability (r = 0.79; Scheier & Carver, 1985).

Perceived control—Perceived control and mastery was measured using the 12-item Midlife Development Inventory. Sample items from the personal mastery subscale include “I can do just about anything I really set my mind to”; and “when I really want to do something, I usually find a way to succeed at it.”; Sample items from the perceived constraints subscale include the items “other people determine most of what I can and cannot do”; and “there is little I can do to change the important things in my life.”; Scores on the total scale ranged from 12 to 84, with higher scores indicating greater perceived control and personal mastery. This scale exhibited acceptable internal consistency in three national probability samples of adults (α 's = 0.53–0.86; Lachman & Weaver, 1998a, b).

Pain—Pain intensity was assessed using the PROMIS Pain Intensity scale; three items measured current and past week levels of average and worst pain on a scale of 3–15, where a score of three indicated no pain and higher scores indicated greater perceived pain. Pain interference was measured via the PROMIS Pain Interference scale; four items measured how much pain interfered with daily and social activities, work, and household chores in the past week on a scale of 4–20, where a score of four indicated no pain interference and higher scores indicated greater perceived pain interference (Cella et al., 2007). These scales demonstrate high internal consistency (α 's = 0.80 and 0.97, respectively; Cella et al., 2010).

Statistical analyses

Structural equation modeling (SEM) was used to create a latent factor of mood disturbance and to examine primary hypotheses. The advantages of using SEM include the ability to utilize latent variables, as well as to analyze several multiple regression equations simultaneously (Byrne, 2001). In evaluating the adequacy of all models, we primarily considered two fit indices: the comparative fit index (CFI) and the root-mean-square error of approximation (RMSEA). A CFI of 0.90 or greater was considered to indicate good fit, and values of 0.95 or greater was considered excellent fit. The 90% confidence interval around the RMSEA point estimate was considered to indicate good fit to the data if it included values of 0.10 or less, with values less than 0.06 indicating excellent fit. The Chi-square statistic was used to compare models, but was not used as an indicator of model fit because of its sensitivity to sample size (Byrne, 2001; Hu & Bentler, 1998; Kline, 2015).

SPSS 22.0 software was used for all descriptive statistics, correlations, and factor analyses. SEM analyses were conducted using the maximum likelihood estimation (MLE) procedure in AMOS 22.0 (Arbuckle, 2013). The mood disturbance measurement model consisted of depressed mood, anxiety, and anger, and the factor loading of one measured indicator was set to 1.0. The first structural model assessed the association of early adversity with pain intensity and pain interference; the pain variables were correlated in this model. The second structural model examined whether the latent construct of mood disturbance and the measured variable of sleep disturbance explained the relationship between early adversity and pain intensity and interference; mood and sleep disturbances were correlated in this model.

Standard demographic and health related variables (e.g., age, gender, race/ethnicity, education, income, and BMI) were examined as potential covariates because of their likely linkages with the core study variables. Higher BMI, female gender, and older age are often linked with higher self-reported pain (see Emery et al., 2017; Fillingim et al., 2009), and sequelae of early adversity may vary by gender, socioeconomic status (SES), and race (see Adler & Snibbe, 2003; Heim et al., 2008; Slopen et al., 2010). Only gender, age, BMI, income, and Black race were significantly associated with the core study variables in the structural models and were thus retained as covariates. Income was dummy coded based on the median level of \$40,000–\$59,999 as either above or below \$40,000, with a higher number representing individuals with an income below \$40,000.

A third structural model tested optimism as a moderator of the full model described above, with early adversity, mood and sleep disturbances, pain intensity and interference, and

covariates. A fourth structural model separately tested perceived control as a moderator of the full model. As displayed in Table 1, the mean for optimism was 22.10 ± 4.73 ($N = 238$), and the mean for control was 61.76 ± 13.71 ($N = 237$). Because AMOS only allows for multiple group analysis for moderation, a low optimism group (scores ranging from 7 to 22) and a high optimism group (scores ranging from 23 to 30), as well as a low control group (scores ranging from 18 to 64) and a high control group (scores ranging from 65 to 84) were created using median splits. Optimism and control groups were then separately examined as moderators of the full structural model using the multiple group analysis feature in AMOS. A Chi-square difference test was used to compare the Chi-square values and degrees of freedom for this unconstrained structural model with a model in which all paths were constrained to assess any significant differences between groups. In the unconstrained model, differences among key paths of interest were examined using Chi-square thresholds. Given the limitations of using a median split for moderation, any path of interest that showed a significant moderated effect was further examined as a continuous moderator using regression in SPSS.

Missing data

All scales with at least four items were prorated for missing data, such that the mean scales were calculated even if participants were missing items, as long as no more than 20% of the total scale was missing. The total scales were calculated by taking the average of all available items and multiplying this score by the number of scale items. For PROMIS scales with five or more items, raw scale scores were prorated for missingness if at least 50% of the items were answered (see Scott et al., 2015). After these procedures, the total sample of 265 had largely complete data, with minimal data seemingly missing at random. The MLE procedure in SEM has been shown to effectively handle data missing at random (see Enders & Bandalos, 2001).

Results

Table 1 shows means for study variables and covariates. All variables were within the acceptable range for skewness and kurtosis, signifying normal distribution. The median for pain intensity was 6 (11% of participants), with 29% reporting no pain, and 1% reporting the maximum score of 15. The median for pain interference also was 6 (6% of participants), with 46% reporting no pain interference and 2% reporting the maximum score of 20. Correlations among study variables are displayed in Table 2. Early adversity, pain, and mood and sleep disturbances were positively correlated with each other ($ps < 0.05$).

Mood disturbance measurement model

The mood disturbance latent factor composed of depressed mood, anxiety, and anger symptoms was an excellent fit, $\chi^2(1, N = 265) = 0.82$, $p = 0.37$, CFI = 1.00, and RMSEA = 0.00 (0.00–0.16). The factor loadings relating each measured indicator to this latent construct were significant (β s between 0.78 and 0.92, $ps < 0.01$).

Structural models

In the first structural model with covariates (not shown), early adversity was significantly associated with pain intensity ($\beta = 0.15, p < 0.05$) and with pain interference ($\beta = 0.18, p < 0.01$). The direct paths from early adversity to the pain variables were no longer significant upon adding mood and sleep disturbances as statistical mediators, and these direct paths were thus removed. In the final model (Fig. 1), early adversity was associated with mood disturbance ($\beta = 0.29, p < 0.01$) and with sleep disturbance ($\beta = 0.22, p < 0.01$). In turn, both mood and sleep disturbances were associated with pain intensity ($\beta = 0.26, p < 0.01$ and $\beta = 0.13, p < 0.05$, respectively) and pain interference ($\beta = 0.27, p < 0.01$ and $\beta = 0.13, p < 0.05$). Overall, 13% of the variance in pain intensity and 14% of the variance in pain interference was explained by these variables. The final structural model had excellent fit, $\chi^2(48, N = 265) = 65.63, p = 0.05$, CFI = 0.98, RMSEA = 0.04 (0.01–0.06).

Moderation by optimism and perceived control

Table 1 shows mean differences in study variables and covariates between the low and high optimism and perceived control groups. Both the low optimism and low perceived control groups had significantly greater levels of mood disturbance and were more likely to have lower income than those in the high optimism and high control groups.

The full structural model with early adversity, mood and sleep disturbances, pain intensity and interference, and covariates that was tested for moderation by optimism had good fit: $\chi^2(96, N = 222) = 144.62, p = 0.00$, CFI = 0.92, and RMSEA = 0.05 (0.03–0.06). This model was compared to a model in which all regression weights and covariances were constrained to be equal for both groups: $\chi^2(126, N = 222) = 171.86, p < 0.00$. Only the path between sleep disturbance and pain interference was significantly different between groups, $\chi^2(97) = 148.82, p < 0.05$; this path was significant for the low but not the high optimism group ($\beta = 0.40, p < 0.01$ and $\beta = -0.02, p = 0.84$, respectively). Overall, 21% of the variance in pain intensity and 18% of the variance in pain interference was explained in the low optimism group versus 9% of the variance in pain intensity and 18% of the variance in pain interference in the high optimism group. For confirmation using a continuous moderator, the path from sleep disturbance to pain interference was tested for moderation by optimism using regression. There was a significant interaction, $\beta = -0.02, p < 0.05$, in the expected direction: Individuals with greater levels of optimism evidenced a weaker relationship between sleep disturbance and pain interference.

The full structural model with early adversity, mood and sleep disturbances, pain intensity and interference, and covariates that was tested for moderation by perceived control had good fit, $\chi^2(96, N = 224) = 139.69, p = 0.00$, CFI = 0.93, and RMSEA = 0.05 (0.03–0.06). This model was compared to a model in which all regression weights and covariances were constrained to be equal for both groups, $\chi^2(126, N = 224) = 180.13, p < 0.00$. Only the path between sleep disturbance and pain interference was significantly different between groups, $\chi^2(97) = 144.61, p < 0.05$; this path was significant for the low control but not the high control group ($\beta = 0.24, p < 0.01$ and $\beta = -0.05, p = 0.58$, respectively). Overall, 17% of the variance in pain intensity and 15% of the variance in pain interference was explained in the low control group versus 16% of the variance in pain intensity and 11% of the variance

in pain interference in the high control group. For confirmation using a continuous moderator, the path from sleep disturbance to pain interference was tested for moderation by perceived control using regression. There was a significant interaction, $\beta = -0.19$, $p < 0.01$, in the expected direction: Individuals with greater levels of perceived control evidenced a weaker relationship between sleep disturbance and pain interference. Standardized regression weights for all paths of interest for both moderation models are displayed in Table 3.

Discussion

The present research examined the association between early life adversity and pain symptomatology in adulthood, as well as several vulnerability and resilience factors implicated in these associations. Among a sample of diverse adults who reported at least minimal early adversity and varying degrees of pain, we found that higher levels of recalled early adversity were associated with higher recent pain intensity and interference. We also found evidence to support our prediction that mood and sleep disturbances may help explain these associations. A latent construct of mood disturbance (indicated by recent depressed mood, anxiety, and anger) and sleep disturbance statistically accounted for the direct associations between early adversity and the pain variables. We also expected that optimism and perceived control might serve as resilience factors, such that those with greater optimism or perceived control would evidence attenuated associations between early adversity and pain symptomatology, between early adversity and mood and sleep disturbances, between mood disturbances and pain symptomatology, and between sleep disturbance and pain symptomatology. We found only partial support for this prediction. Individuals with either lower optimism or lower perceived control demonstrated a significantly stronger association between sleep disturbance and pain interference. However, optimism and perceived control did not moderate the paths between early adversity, mood disturbance, and pain intensity or interference, or the paths between early adversity, sleep disturbance, and pain intensity. Together, these findings suggest that psychological and behavioral factors associated with risk and resilience may help explain linkages between recalled early adversity and pain symptomatology in adulthood.

The significant direct association we observed between early life adversity and pain is consistent with some past research showing a connection between recalled early adversity and pain symptomatology in adulthood (Davis et al., 2004; Sachs-Ericsson et al., 2009, 2017). Adverse childhood experiences, particularly abuse, are related to more medical problems and chronic pain, and this connection may be exacerbated by current life stress, alterations in brain functioning, and unhealthy behaviors (see Sachs-Ericsson et al., 2009). As discussed earlier, some studies have not found significant associations between childhood maltreatment and increased pain in adulthood (see Fillingim & Edwards, 2005; Raphael et al., 2001). Discrepancies between studies may be explained by characteristics between samples or methods. Studies reporting null findings have tended to include only participants with court-documented abuse or have focused on child maltreatment, suggesting that it may be subjectively perceived adversity, such as that utilized in the present research, which may more consistently relate to pain. Additionally, we examined a broad, aggregate measure of reported early adversity as a continuous variable, which can be a useful approach because

adversities are often co-occurring and may have a collective influence on health (Green et al., 2010; Sachs-Ericsson et al., 2009).

As predicted, observed results were consistent with a model whereby mood and sleep disturbances accounted for the association between early adversity and pain. Previous work has demonstrated potential biological and psychological mechanisms that may explain these findings. Early trauma may alter interactions among nervous, endocrine, and immune systems in ways that may increase sensitivity to stress and risk for depression and anxiety, and thus may also heighten pain symptomatology (for reviews, see Heim & Nemeroff, 2001; Sachs-Ericsson et al., 2009). Neurobiological and immune alterations resulting from early trauma may also lead to fatigue, dysregulations in the sleep/wake cycle and circadian rhythms via elevations of corticotropin-releasing hormone (Germain et al., 2008; Kajeepeta et al., 2015; Silverman et al., 2010). In turn, poor sleep can increase attention towards pain and may produce hyperalgesic changes in opioid systems (Affleck et al., 1996; Lautenbacher et al., 2006). Future experimental studies are needed to examine whether such potential mechanisms underlie the associations modeled in the present research.

Partially supporting our hypotheses, we found a stronger connection between sleep disturbance and pain interference in individuals with lower optimism and lower perceived control. This is consistent with the perspective that optimism and perceived control are protective (or that a lack of optimism and perceived control are problematic). Past research has shown that survivors of early trauma who possessed resilient characteristics, such as an internal locus of control, were better able to cope with their sleep problems (Chambers & Belicki, 1998). Optimism and perceived control may help prevent poor sleep from worsening pain interference by enabling individuals to better utilize cognitive coping resources to manage aspects of pain and to minimize attention to their pain (see Affleck et al., 1996). Further, optimism has been shown to play a role in effective coping with pain through associations with protective health behaviors, such as reduced alcohol use and smoking (Smith & Zautra, 2008; Sturgeon & Zautra, 2010). Similarly, optimists may have better sleep quality through their use of proactive coping with stress, as well as their use of positive self-regulatory behaviors (see Uchino et al., 2017). Biological mechanisms also may be relevant in associations between resilience resources, sleep, and pain. For example, inflammation has been linked with sleep disturbances and pain (Cho et al., 2015; Gatchel et al., 2007); and optimism and control, as well as the associated positive health and coping behaviors described above, may be linked with reduced inflammation and subsequently fewer negative health outcomes (see Dunkel-Schetter & Dolbier, 2011). Investigating biological and psychological mechanisms by which resilience resources affect sleep and pain using longitudinal designs are promising avenues for future research.

As noted above, however, optimism and perceived control did not moderate the other paths of interest in the present study. The paths between early adversity and mood and sleep disturbances were not moderated by optimism and perceived control. The degree to which an individual demonstrates a resilient response depends on the context and specifics of the challenge, with few individuals being resilient to all possible stressors (Rutter, 1987; Sturgeon & Zautra, 2010). It is possible that optimism and perceived control are not sufficient to protect against mood and sleep disturbances in the context of a significant

stressor such as early adversity. The paths from mood disturbance to the pain variables were also not moderated by optimism and perceived control. It could be the case that optimism and control may not exert unique influences on pain above the influence of mood. In the present research, mood disturbances were significantly confounded with higher optimism and perceived control, whereas sleep disturbances were not; mood disturbance appeared to be equally detrimental for pain among those with high and low optimism or perceived control. Finally, it is of interest that optimism and control moderated the path between sleep disturbance and pain interference, but not the path between sleep disturbance and pain intensity. It is possible that optimism and control may help reduce the perceived functional limitations related to poor sleep quality and pain, but not the intensity of the pain itself. This is consistent with past research showing that individuals who possess a positive outlook and who perceive more control over their pain also have evidenced fewer behavioral impairments and better physical functioning (Sturgeon & Zautra, 2010).

Limitations and future directions

The present study had several limitations that highlight the need for future research. First, we did not recruit a sample based on the degree of early adversity, which limits generalizability to other samples. To maximize comparability to other studies of early life adversity and pain and to enable us to examine dose–response relationships between early adversity, mood, sleep, and pain, we excluded individuals reporting no early adversity. Results were comparable when the full sample was included, however. It is also important to note that all data were cross-sectional, preventing the ability to draw causal connections from this work. Data also were self-reported and retrospective and therefore subject to recall bias. A recent study concluded that in comparison to prospective measures of childhood adversity, retrospective measures may underestimate the influence of the adversity on objectively assessed health outcomes (e.g., through tests and biomarkers) and overestimate the influence of the adversity on subjectively assessed outcomes (e.g., through self-report; Reuben et al., 2016). Research using prospective longitudinal designs and/or experimental methods is needed to better establish directional pathways and to suggest mechanisms underlying risk and resilience in the context of early adversity and pain.

An additional limitation of the current work is that our measure of early adversity did not account for the severity or chronicity of each reported adversity. Further, we did not examine the association of each specific adversity with pain due to limited power for such analyses. For similar reasons, we did not assess the influence of other personality factors (e.g., neuroticism), medical comorbidity, or etiology of pain (i.e., fibromyalgia vs. diabetic neuropathy) on these results. Furthermore, our pain measures were limited to assessing pain intensity and interference in the past week, making it difficult to distinguish recent from chronic pain. Future work may benefit from using ecological momentary assessment to elucidate momentary or daily covariation between pain, sleep, and mood in everyday life. Finally, analyses were limited by our use of a dichotomized variable for moderation, which could have decreased our ability to detect within-group variability. However, our multiple group moderation analyses were supported by supplementary regression analyses with continuous moderators.

Conclusion

This study adds to a growing literature examining the relationships between early life adversity and pain symptomatology in adulthood by examining mediators and moderators of this association. Accounting for demographic factors and SES, we found that higher levels of recalled early adversity were associated with higher recent pain intensity and interference, and that recent mood and sleep disturbances accounted for these associations in a large, diverse sample of adults with varying degrees of early adversity and pain. Further, we found that the resilience resources of optimism and perceived control only buffered the relationship between sleep disturbance and pain interference. This work adds yet more evidence that those who have experienced early life adversity are particularly likely to be struggling with not only mood but concomitant sleep and pain issues. Future research examining these associations prospectively and/or experimentally may eventually help illuminate modifiable targets to reduce vulnerabilities and enhance psychological or behavioral protective resources in the context of early adversity and pain in adulthood.

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References

- Adler NE, & Snibbe AC (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science*, 12, 119–123. 10.1111/1467-8721.01245
- Affleck G, Urrows S, Tennen H, Higgins P, & Abeles M. (1996). Sequential daily relations of sleep, pain intensity, and attention to pain among women with fibromyalgia. *Pain*, 68, 363–368. 10.1016/S0304-3959(96)03226-5 [PubMed: 9121825]
- Anda RF, Felitti VJ, Bremner JD, Walker JD, Whitfield C, Perry BD, et al. (2006). The enduring effects of abuse and related adverse experiences in childhood: A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience*, 256, 174–186. 10.1007/s00406-005-0624-4 [PubMed: 16311898]
- Arbuckle JL (2013). *IBM SPSS Amos 22 User's Guide*. Crawfordville, FL: Amos Development Corporation.
- Benca RM, Okawa M, Uchiyama M, Ozaki S, Nakajima T, Shibui K, et al. (1997). Sleep and mood disorders. *Sleep Medicine Reviews*, 1, 45–56. [PubMed: 15310523]
- Byrne BM (2001). *Structural equation modeling with AMOS*. Mahwah, NJ: Lawrence Erlbaum Associates.
- Cella D, Riley W, Stone A, Rothrock N, Reeve B, Yount S, et al. (2010). The patient-reported outcomes measurement information system (PROMIS) developed and tested its first wave of adult self-reported health outcome item banks: 2005–2008. *Journal of Clinical Epidemiology*, 63, 1179–1194. 10.1016/j.jclinepi.2010.04.011 [PubMed: 20685078]
- Cella D, Yount S, Rothrock N, Gershon R, Karon C, Reeve B, et al. (2007). The patient-reported outcomes measurement information system (PROMIS): Progress of an NIH roadmap cooperative group during its first two years. *Medical Care*, 45, S3–S11. 10.2307/40221453
- Chambers E, & Belicki K. (1998). Using sleep dysfunction to explore the nature of resilience in adult survivors of childhood abuse or trauma. *Child Abuse and Neglect*, 22, 753–758. 10.1016/S0145-2134(98)00059-3 [PubMed: 9717612]

- Chapman DP, Wheaton AG, Anda RF, Croft JB, Edwards VJ, Liu Y, et al. (2011). Adverse childhood experiences and sleep disturbances in adults. *Sleep Medicine*, 12, 773–779. 10.1016/j.sleep.2011.03.013 [PubMed: 21704556]
- Chen E, & Miller GE (2013). Socioeconomic status and health: Mediating and moderating factors. *Annual Review of Clinical Psychology*, 9, 723–749. 10.1146/annurevclinpsy-050212-185634
- Cho HJ, Seeman TE, Kiefe CI, Lauderdale DS, & Irwin MR (2015). Sleep disturbance and longitudinal risk of inflammation: Moderating influences of social integration and social isolation in the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Brain, Behavior, and Immunity*, 46, 319–326. 10.1016/j.bbi.2015.02.023
- Cicchetti D. (2013). Annual research review: Resilient functioning in maltreated children—past, present, and future perspectives. *Journal of Child Psychology and Psychiatry*, 54, 402–422. 10.1111/j.1469-7610.2012.02608.x [PubMed: 22928717]
- Davis DA, Luecken LJ, & Zautra AJ (2004). Are reports of childhood abuse related to the experience of chronic pain in adulthood? A meta-analytic review of the literature. *The Clinical Journal of Pain*, 21, 398–405. 10.1097/01.ajp.0000149795.08746.31
- Dunkel-Schetter C, & Dolbier C. (2011). Resilience in the context of chronic stress and health in adults. *Social and Personality Psychology Compass*, 5, 634–652. 10.1111/j.1751-9004.2011.00379.x [PubMed: 26161137]
- Emery CF, Olson KL, Bodine A, Lee V, & Habash DL (2017). Dietary intake mediates the relationship of body fat to pain. *Pain*, 158, 273–277. 10.1097/j.pain.0000000000000754 [PubMed: 28092647]
- Enders CK, & Bandalos DL (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling: A Multidisciplinary Journal*, 8, 430–457. 10.1207/S15328007SEM0803_5
- Fernandez E. (2002). Anxiety, depression, and anger in pain: Research findings and clinical options. Dallas, TX: Advanced Psychological Resources.
- Fillingim RB, & Edwards RR (2005). Is self-reported childhood abuse history associated with pain perception among healthy young women and men? *The Clinical Journal of Pain*, 21, 387–397. 10.1097/01.ajp.0000149801.46864.39 [PubMed: 16093744]
- Fillingim RB, King CD, Ribeiro-Dasilva MC, Rahim-Williams B, & Riley JL 3rd. (2009). Sex, gender, and pain: A review of recent clinical and experimental findings. *Journal of Pain*, 10, 447–485. 10.1016/j.jpain.2008.12.001 [PubMed: 19411059]
- Gatchel RJ, Peng YB, Peters ML, Fuchs PN, & Turk DC (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, 133, 581–624. 10.1037/0033-2909.133A581 [PubMed: 17592957]
- Germain A, Buysse DJ, & Nofzinger E. (2008). Sleep-specific mechanisms underlying posttraumatic stress disorder: Integrative review and neurobiological hypotheses. *Sleep Medicine Reviews*, 12, 185–195. 10.1016/j.smrv.2007.09.003 [PubMed: 17997114]
- Graham-Engeland JE, Zawadzki MJ, Slavish DC, & Smyth JM (2015). Depressive symptoms and momentary mood predict momentary pain among rheumatoid arthritis patients. *Annals of Behavioral Medicine*, 50, 12–23. 10.1007/s12160015-9723-2
- Green JG, McLaughlin KA, Berglund PA, Gruber MJ, Sampson NA, Zaslavsky AM, et al. (2010). Childhood adversities and adult psychiatric disorders in the national comorbidity survey replication I: Associations with first onset of DSM-IV disorders. *Archives of General Psychiatry*, 67, 113–123. 10.1001/archgenpsychiatry.2009.186 [PubMed: 20124111]
- Heim C, & Nemeroff CB (2001). The role of childhood trauma in the neurobiology of mood and anxiety disorders: Preclinical and clinical studies. *Biological Psychiatry*, 49, 1023–1039. 10.1016/S0006-3223(01)01157-X [PubMed: 11430844]
- Heim C, Newport DJ, Mletzko T, Miller AH, & Nemeroff CB (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology*, 33, 693–710. <https://doi.org/10.1016/j.psyneuen.2008.03.008> [PubMed: 18602762]
- Hu L-T, & Bentler PM (1998). Fit indices in covariance structure modeling: Sensitivity to underparameterized model misspecification. *Psychological Methods*, 3, 424–453. 10.1037/1082-989X.3.4.424

- Jones GT, Power C, & Macfarlane GJ (2009). Adverse events in childhood and chronic widespread pain in adult life: Results from the 1958 British Birth Cohort Study. *Pain*, 143, 92–96. 10.1016/j.pain.2009.02.003 [PubMed: 19304391]
- Kajeeepeta S, Gelaye B, Jackson CL, & Williams MA (2015). Adverse childhood experiences are associated with adult sleep disorders: A systematic review. *Sleep Medicine*, 16, 320–330. 10.1016/j.sleep.2014.12.013 [PubMed: 25777485]
- Kline RB (2015). *Principles and practice of structural equation modeling*. New York City: Guilford Publications.
- Lachman ME, & Weaver SL (1998a). The sense of control as a moderator of social class differences in health and well-being. *Journal of Personality and Social Psychology*, 74, 763–773. 10.1037/0022-3514.74.3.763 [PubMed: 9523418]
- Lachman ME, & Weaver SL (1998b). Sociodemographic variations in the sense of control by domain: Findings from the MacArthur studies of midlife. *Psychology and Aging*, 13, 553–562. 10.1037/0882-7974.13.4.553 [PubMed: 9883456]
- Lautenbacher S, Kundermann B, & Krieg J-C (2006). Sleep deprivation and pain perception. *Sleep Medicine Reviews*, 10, 357–369. 10.1016/j.smrv.2005.08.001 [PubMed: 16386930]
- Logan-Greene P, Green S, Nurius PS, & Longhi D. (2014). Distinct contributions of adverse childhood experiences and resilience resources: A cohort analysis of adult physical and mental health. *Social Work in Health Care*, 53, 776–797. 10.1080/00981389.2014.944251 [PubMed: 25255340]
- Lumley MA, Cohen JL, Borszcz GS, Cano A, Radcliffe AM, Porter LS, et al. (2011). Pain and emotion: A biopsychosocial review of recent research. *Journal of Clinical Psychology*, 67, 942–968. 10.1002/jclp.20816 [PubMed: 21647882]
- Masten AS, Burt KB, Roisman GI, Obradovic J, Long JD, & Tellegen A. (2004). Resources and resilience in the transition to adulthood: Continuity and change. *Development and Psychopathology*, 16, 1071–1094. 10.1017/S0954579404040143 [PubMed: 15704828]
- Raphael KG, Widom CS, & Lange G. (2001). Childhood victimization and pain in adulthood: A prospective investigation. *Pain*, 92, 283–293. [PubMed: 11323150]
- Reich JW, Zautra AJ, & Hall JS (2010). *Handbook of adult resilience*. New York, NY: The Guilford Press.
- Reuben A, Moffitt TE, Caspi A, Belsky DW, Harrington H, Schroeder F, et al. (2016). Lest we forget: Comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. *Journal of Child Psychology and Psychiatry*, 57, 1103–1112. 10.1111/jcpp.12621 [PubMed: 27647050]
- Rutter M. (1987). Psychosocial resilience and protective mechanisms. *American Journal of Orthopsychiatry*, 57, 316–331. 10.1111/j.1939-0025.1987.tb03541.x [PubMed: 3303954]
- Sachs-Ericsson N, Cromer K, Hernandez A, & Kendall-Tackett K. (2009). A review of childhood abuse, health, and pain-related problems: The role of psychiatric disorders and current life stress. *Journal of Trauma and Dissociation*, 10, 170–188. 10.1080/15299730802624585 [PubMed: 19333847]
- Sachs-Ericsson NJ, Sheffler JL, Stanley IH, Piazza JR, & Preacher KJ (2017). When emotional pain becomes physical: Adverse childhood experiences, pain, and the role of mood and anxiety disorders. *Journal of Clinical Psychology*, 73, 1403–1428. 10.1002/jclp.22444 [PubMed: 28328011]
- Scheier MF, & Carver CS (1985). Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychology*, 4, 219–247. 10.1037/0278-6133.4.3.219 [PubMed: 4029106]
- Scott SB, Graham-Engeland JE, Engeland CG, Smyth JM, Almeida DM, Katz MJ, et al. (2015). The effects of stress on cognitive aging, physiology and emotion (ESCAPE) project. *BMC Psychiatry*, 15, 146 10.1186/s12888-0150497-7 [PubMed: 26138700]
- Seery MD, Leo RJ, Holman EA, & Silver RC (2010). Lifetime exposure to adversity predicts functional impairment and healthcare utilization among individuals with chronic back pain. *Pain*, 150, 507–515. 10.1016/j.pain.2010.06.007 [PubMed: 20594645]

- Silverman MN, Heim CM, Nater UM, Marques AH, & Sternberg EM (2010). Neuroendocrine and immune contributors to fatigue. *PM&R*, 2, 338–346. 10.1016/j.pmrj.2010.04.008 [PubMed: 20656615]
- Slopen N, Lewis TT, Gruenewald TL, Mujahid MS, Ryff CD, Albert MA, et al. (2010). Early life adversity and inflammation in African Americans and Whites in the Midlife in the United States survey. *Psychosomatic Medicine*, 72, 694. 10.1097/PSY.0b013e3181e9c16f [PubMed: 20595419]
- Smith MT, & Haythornthwaite JA (2004). How do sleep disturbance and chronic pain inter-relate? Insights from the longitudinal and cognitive-behavioral clinical trials literature. *Sleep Medicine Reviews*, 8, 119–132. 10.1016/S1087-0792(03)00044-3 [PubMed: 15033151]
- Smith BW, & Zautra AJ (2008). Vulnerability and resilience in women with arthritis: Test of a two-factor model. *Journal of Consulting and Clinical Psychology*, 76, 799–810. 10.1037/0022-006X.76.5.799 [PubMed: 18837597]
- Southwick SM, Bonanno GA, Masten AS, Panter-Brick C, & Yehuda R. (2014). Resilience definitions, theory, and challenges: Interdisciplinary perspectives. *European Journal of Psychotraumatology*. <https://doi.org/10.3402/ejpt.v5.25338>
- Strine TW, & Chapman DP (2005). Associations of frequent sleep insufficiency with health-related quality of life and health behaviors. *Sleep Medicine*, 6, 23–27. 10.1016/j.sleep.2004.06.003 [PubMed: 15680291]
- Sturgeon JA, & Zautra AJ (2010). Resilience: A new paradigm for adaptation to chronic pain. *Current Pain and Headache Reports*, 14, 105–112. 10.1007/s11916-010-0095-9 [PubMed: 20425199]
- Turner RJ, Wheaton B, & Lloyd DA (1995). The epidemiology of social stress. *American Sociological Review*, 60, 104.
- Uchino BN, Cribbet M, de Grey RG, Cronan S, Trettevik R, & Smith TW (2017). Dispositional optimism and sleep quality: A test of mediating pathways. *Journal of Behavioral Medicine*, 40, 360–365. 10.1007/s10865-0169792-0 [PubMed: 27592128]
- Zautra A, Hall J, & Murray K. (2008). Resilience: A new integrative approach to health and mental health research. *Health Psychology Review*, 2, 41–64. 10.1080/17437190802298568

Fig. 1.

Structural model. *Note* structural model of early life adversity, mood and sleep disturbances, and pain intensity and pain interference with covariates, $\chi^2(48, N=265) = 65.63, p = 0.05$, CFI = 0.98, RMSEA = 0.04 (0.01–0.06). Unidirectional arrows between variables represent regression coefficients, whereas bidirectional arrows represent covariances. Darker arrows represent key paths of interest. For simplicity of presentation, error variances are not shown. $^{\dagger}p < 0.10$; $*p < 0.05$; $**p < 0.01$

Table 1

Characteristics of the study sample based on optimism and perceived control

	Overall sample (N = 265)		Low optimism (N = 123)		High optimism (N = 99)		Low perceived control (N = 117)		High perceived control (N = 107)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Early life adversity (range 0–8)	2.44	1.43	2.56	1.54	2.31	1.32	2.46	1.38	2.44	1.42
Depressed mood (range 8–40)	16.31	7.33	17.98	7.75	14.80	6.57**	18.89	7.37	12.85	4.84**
Anxiety (range 7–35)	17.37	6.04	18.99	6.06	15.84	5.46**	18.98	5.89	15.38	5.27**
Anger (range 8–40)	19.22	6.95	20.80	7.26	17.80	5.93**	20.77	6.94	17.06	5.74**
Sleep disturbance (range 8–40)	21.57	3.33	21.53	3.39	21.59	3.16	21.44	3.37	21.79	3.22
Optimism (range 6–30)	22.10	4.73	18.59	3.09	25.42	1.91**	20.39	4.67	24.07	3.87**
Perceived control (range 12–84)	61.76	13.71	56.45	14.50	66.93	10.45**	50.80	9.26	72.60	4.76**
Pain intensity (range 3–15)	6.62	3.11	6.92	3.13	6.54	3.11	6.68	3.11	6.49	3.07
Pain interference (range 4–20)	7.64	4.56	7.99	4.60	7.62	4.56	7.93	4.63	7.45	4.52
Age	46.40	11.24	46.74	10.68	45.80	11.24	46.81	11.22	46.59	10.84
Gender	65% female		63% female		65% female		70% female		66% female	
BMI	32.01	8.28	32.05	7.92	31.97	8.66	32.38	8.15	32.11	8.66
Black race	60% Black		59% Black		65% Black		57% Black		64% Black	
Income	48% below 40k		58% below 40k		43% below 40k*		59% below 40k		38% below 40k**	

* $p < 0.05$;

** $p < 0.01$.

Significance indicators in the high optimism column indicate comparisons between low optimism and high optimism groups, and significance indicators in the high perceived control column indicate comparisons between low perceived control and high perceived control group

Table 2

Correlations between key study variables and covariates

	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Early life adversity	.19**	.26**	.27**	.21**	-.17**	-.15*	.17*	.20**	.09	.10	-.20**	.08	.16*
2. Depressed mood	-	.72**	.67**	.14*	-.40**	-.53**	.24**	.25**	.02	-.03	.01	.11	.24**
3. Anxiety	-	-	.77**	.23**	-.41**	-.43**	.28**	.29**	-.06	.06	-.12	.06	.17*
4. Anger	-	-	-	.20**	-.37**	-.38**	.18**	.21**	-.07	.06	.00	.07	.15*
5. Sleep disturbance	-	-	-	-	.09	-.04	.21**	.19**	.06	.01	-.07	-.16*	.02
6. Optimism	-	-	-	-	-	.53**	-.10	-.07	.10	.00	.02	.04	-.18**
7. Perceived control	-	-	-	-	-	-	-.10	-.12	-.02	-.10	.09	-.04	-.24**
8. Pain intensity	-	-	-	-	-	-	-	.78**	.16*	.07	-.05	.11	.16*
9. Pain interference	-	-	-	-	-	-	-	-	.16*	.07	-.08	.22**	.15*
10. Age	-	-	-	-	-	-	-	-	-	.03	.03	.03	.02
11. Gender	-	-	-	-	-	-	-	-	-	-	-.01	.09	-.05
12. Black race	-	-	-	-	-	-	-	-	-	-	-	.08	-.03
13. BMI	-	-	-	-	-	-	-	-	-	-	-	-	.23**
14. Income	-	-	-	-	-	-	-	-	-	-	-	-	-

* Correlation is significant at the 0.05 level (2-tailed);

** correlation is significant at the 0.01 level (2-tailed)

Table 3
Estimated standardized regression weights between paths of interest in the moderation models

Model	Pathway	Estimate	SE	P
Low optimism	Early life adversity → mood disturbance	.26	.36	<.01
	Early life adversity → sleep disturbance	.28	.19	<.01
	Mood disturbance → pain intensity	.31	.05	<.01
	Mood disturbance → pain interference	.25	.07	<.01
	Sleep disturbance → pain intensity	.21	.08	<.05
High optimism	Sleep disturbance → pain interference	.40	.12	<.01
	Early life adversity → mood disturbance	.24	.44	<.05
	Early life adversity → sleep disturbance	.09	.25	.37
	Mood disturbance → pain intensity	.24	.07	<.05
	Mood disturbance → pain interference	.35	.09	<.01
	Sleep disturbance → pain intensity	.003	.10	.98
	Sleep disturbance → pain interference	-.02	.14	.84
	Early life adversity → mood disturbance	.35	.40	<.01
	Early life adversity → sleep disturbance	.28	.22	<.01
	Mood disturbance → pain intensity	.27	.05	<.01
Low perceived control	Mood disturbance → pain interference	.27	.08	<.01
	Sleep disturbance → pain intensity	.22	.08	<.05
	Sleep disturbance → pain interference	.24	.12	<.01
	Early life adversity → mood disturbance	.25	.24	<.05
	Early life adversity → sleep disturbance	.14	.22	.15
	Mood disturbance → pain intensity	.36	.11	<.01
	Mood disturbance → pain interference	.32	.15	<.01
	Sleep disturbance → pain intensity	.01	.09	.91
	Sleep disturbance → pain interference	-.05	.12	.58
High perceived control				