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## Lifetime exposure to adversity predicts functional impairment and healthcare utilization among individuals with chronic back pain

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### ABSTRACT

Previous research has demonstrated an association between lifetime exposure to adverse events and chronic back pain (CBP), but the nature of this relationship has not been fully specified. Adversity exposure typically predicts undesirable outcomes, suggesting that lack of all adversity is optimal. However, we hypothesized that among individuals faced with CBP, a history of a low level of lifetime adversity would yield protective effects, manifested as lower impairment and healthcare utilization. Adult members of a national panel ( $N = 396$ ) endorsed a history of CBP when reporting their physical health status in an online survey; they further reported their functional impairment and healthcare utilization. Respondents had previously completed a survey of lifetime exposure to adverse events. Significant U-shaped quadratic relationships emerged between adversity and self-rated functional impairment ( $p < 0.001$ ), disabled employment status ( $p < 0.001$ ), frequency of physician/clinic visits for CBP ( $p < 0.01$ ), prescription (but not over-the-counter) analgesic use ( $p < 0.01$ ), and comorbid depression treatment seeking ( $p < 0.01$ ). Specifically, people with some lifetime adversity reported less impairment and healthcare utilization than people who had experienced either no adversity or a high level of adversity. Additional analyses failed to support alternative explanations of the findings. Implications for understanding and promoting resilience in the context of CBP are discussed.

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### 1. Introduction

Complaints of back pain are among the most common reasons for patient presentations to ambulatory medical settings in industrialized countries [3]. Most of these remit spontaneously or respond to simple treatment interventions; however, for a subset of patients, these symptoms remain chronic [33]. The costs of chronic back pain (CBP) are monumental when healthcare, absenteeism, lost wages, and disability are considered [7,14]. Attempts to understand the persistence, refractoriness, and sequelae of CBP (e.g., disability) have underscored the importance of psychosocial variables [29,36].

Among psychosocial variables, research has examined the relationship between exposure to adverse events and CBP. Self-reported endorsement of recent exposure to stressful life events has been linked to the onset of CBP [8,24] and is associated with

greater perceived severity and chronicity of symptoms [4,13,27]. Higher rates of self-reported exposure to adverse events have been demonstrated among CBP patients as compared with non-patients [13] and among CBP patients demonstrating psychological distress (e.g., depression) relative to CBP patients without concomitant distress [4,25,26,45]. Moreover, CBP patients report an increased rate of traumatic events in childhood, including sexual or physical abuse [27,28,41]. Demonstrating a graded relationship, a greater number of childhood traumas predicted greater likelihood of adult CBP [23]. Similarly, childhood physical and sexual abuse rendered CBP patients refractory to surgical interventions [41,42].

Overall, these data suggest a plausible psychobiological link between adversity and CBP. What remains unclear is the exact nature of this relationship and the range of CBP-related outcomes that are implicated. Methodological shortcomings common in previous research include reliance on small [4,24–26,41,45] and clinically based samples [8,13,24–26,41], limiting the generalizability of reported results. Furthermore, failure to consider psychopathological states such as depression and anxiety may result in spurious relationships between endorsed adversity exposure and CBP-related outcomes [8,23,24,28,41,42]. Finally, the approaches typically used

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to assess adversity in CBP research may obscure important differences between low levels of adversity exposure and a history of no experience with adverse life events.

One theory argues that exposure to stressors can have beneficial effects. Specifically, limited exposure may “toughen” individuals, leaving them more emotionally stable and better able to cope with both difficult stressors and minor challenges; in contrast, both sheltering from all stressors and exposure to continuous chronic stressors lead to lack of “toughness” [10]. Notably, previous research has ignored the potential protective effects of lifetime adversity on CBP patients’ responses to their pain [5,22,35].

Rather than investigate the etiology of CBP, our purpose was to examine the relationship between cumulative lifetime adversity and negative consequences of CBP (i.e., perceived functional impairment and healthcare utilization). Existing research investigating CBP with community samples has largely overlooked the relationship between adversity exposure and the degree to which medical treatment is sought. We also tested the hypothesis that exposure to some adversity may yield beneficial outcomes in the face of CBP. Specifically, consistent with toughness theory, we speculated that individuals endorsing low levels of cumulative lifetime adversity exposure would be less negatively affected by CBP than those endorsing no prior adversity or high levels of adversity exposure.

## 2. Methods

### 2.1. Overview of objectives

The goals of the present investigation were threefold. Using a national sample of patients endorsing CBP, we first tested whether linear relationships existed between cumulative lifetime adversity and self-rated functional impairment, employment status, frequency of medical treatment, analgesic use, and treatment-seeking for comorbid depression and/or anxiety. Second, we assessed whether these linear models oversimplified the associations between lifetime history of adversity exposure and the above-mentioned outcomes; specifically, we tested for quadratic relationships. Third, we conducted additional analyses to test alternative explanations and to explore additional facets of the research question, including the roles of comorbid depression and anxiety, and childhood physical and sexual abuse, in the relationships between adversity exposure and CBP-related outcomes.

### 2.2. Data collection

The study sample was drawn from a nationally representative Web-enabled panel created through traditional probability methods (i.e., using random-digit dialing; RDD), by Knowledge Networks Inc. (KN), an online survey research company [43,44]. To ensure the representation of population segments that would not otherwise have Internet access, KN provides panel households with an Internet connection and Web TV to serve as a computer monitor. In exchange, panel members complete 3–4 short surveys a month. Unlike typical Internet panels, in which people who already have Internet access choose to opt-in, no one can volunteer for the KN panel; all participants are selected using RDD, making the KN probability-based panel demographically comparable to non-Internet based probability samples. Once participants have been selected for the panel, responding to any given survey is voluntary, and the provision of Internet service is not dependent on completion of any specific survey. Upon entry into the KN panel, respondents provide demographic information. The responses of “seasoned” versus “naive” participants do not differ [9].

The assessments were administered by KN online or via paper-and-pencil surveys mailed to respondents. Respondents were in-

formed about the study and its risks and benefits prior to completing each survey; subsequent completion of the surveys was considered informed consent to participate. The Institutional Review Board at the University of California, Irvine, approved study procedures.

Starting in September 2001, our research team collected longitudinal data from a national sample of the adult US population selected from the KN panel [43,44]. A total of 1576 respondents reported their physical health status, healthcare utilization, and functional impairment in September 2003 (representing a 74% participation rate); of these, 396 endorsed having a history of CBP (see below), constituting the sample used in subsequent analyses. Given that respondents reported their lifetime exposure to adversity retrospectively, CBP-related outcomes could have biased recall of prior lifetime adversity if current distress facilitated the recall of adverse events. We attempted to mitigate this possibility by assessing adversity at least 1 year before the outcome measures (i.e., by September 2002), thereby creating a prospective test of our hypotheses. Additional waves of data were collected on subsamples of the full survey panel before September 2002 [44]; as these waves provide restricted sample sizes and are not relevant for current analyses, they are not discussed further.

### 2.3. Measures

#### 2.3.1. CBP history

As in previous research investigating non-clinical samples [27,28], respondents’ CBP history was identified via survey. Specifically, history of chronic back pain or back problems was assessed as part of a larger health survey, modified from the Centers for Disease Control’s National Center for Health Statistics annual National Health Interview Survey (NHIS) [34] that asked respondents “Has a medical doctor ever diagnosed you as suffering from any of the following ailments?” with prompts for 35 physical and mental health ailments, including “chronic back pain or back problems”. Additional questions asked if respondents had diagnosed themselves with any of these conditions and if their self-diagnoses had been confirmed by a physician. We included respondents in our CBP sample if they endorsed any CBP item. We adopted this approach for several reasons. First, the survey was worded for lay people, who are less likely than medical professionals to appreciate the difference between experiencing symptoms acknowledged by a physician versus receiving an actual diagnosis, as well as finer distinctions in defining chronicity and different types of back pain and problems. We thus used these items as proxies for experiencing nonspecifically defined CBP. Second, given our focus on predicting how people respond to CBP rather than on the etiology of CBP, we wanted to include respondents who had failed to seek medical treatment rather than limiting the sample to only those reporting a physician diagnosis. Of the CBP sample, 83.1% reported physician diagnosis or confirmation and 16.9% did not. Finally, similarly worded items have been used successfully in previous CBP research [23]. A limitation of our strategy is that it sacrificed precision in assessing the nature of respondents’ CBP, such as the degree of chronicity and specificity of symptoms. However, our strategy provided the advantage of allowing us to incorporate more sufferers of CBP in our sample, regardless of their level of understanding of diagnostic labels for their symptoms. Although this is an important tradeoff, it is one we deemed appropriate given our research question.

#### 2.3.2. Cumulative lifetime adversity

Lifetime exposure to cumulative adversity was assessed by asking respondents whether they ever experienced each of 37 negative events and the age(s) at which they occurred. Up to four instances of each event were tallied, regardless of duration. The

measure was modified from the Diagnostic Interview Schedule trauma section [37], expanded to include a wider variety of stressful events using primary care patients' reports of lifetime stress [17] and has provided rates of specific events comparable to those in other community samples [6,21]. The list of events included seven categories: one's own illness or injury; loved one's illness or injury; violence (e.g., physical assault, forced sexual relations); bereavement (e.g., parent's death); social or environmental stress (e.g., serious financial difficulties, lived in dangerous housing); relationship stress (e.g., own divorce, parents' divorce); and disaster (e.g., major fire, flood, earthquake, or other community disaster). Thus, the current study assessed adversity using a wider range of major negative life experiences than is typical in previous research. We also treated adversity total as a continuous variable rather than as a categorical variable. Assessing a small number of adverse events or creating categories by collapsing across parts of the range of adversity totals may obscure important differences between people with low versus no exposure to adversity.

### 2.3.3. Functional impairment

Respondents completed two items from the SF-36 Health Survey [50,51] that assessed self-rated functional impairment in work and social activities resulting from physical health. Using the mean of these items, we calculated a continuous scale ranging from 1 to 5 ( $\alpha = 0.88$ ,  $M = 2.03$ ,  $SD = 1.14$ ). Respondents also reported their employment status, coded as 1 if they endorsed "disabled" and 0 if they chose any of the other eight possible options (i.e., paid employee, self-employed, owner/partner in small business/practice/farm, work without pay in family business/farm, unemployed but looking for work, retired, homemaker, other).

### 2.3.4. Healthcare utilization

Respondents were asked if they sought physician or clinic treatment for CBP, and if so, how frequently, using an 11-point scale ranging from "I don't go to a doctor anymore for this problem" to "more than twice a week". We treated this measure as a continuous scale ranging from 0 to 10 ( $M = 1.47$ ,  $SD = 2.19$ ). Respondents who reported not receiving medical care for their CBP were coded as 0.

Respondents were also asked if they took prescription analgesics, over-the-counter (OTC) analgesics, or both for CBP. From this question, we created two dichotomous variables: one for prescription analgesics and one for OTC analgesics (coded 1 = yes, 0 = no).

Given the prevalence and important treatment implications of comorbid anxiety disorder and depression among people with a history of CBP [14,29], we assessed whether respondents reported currently seeking physician or clinic treatment for anxiety disorder or depression as part of the larger health survey described above. Each was represented as a separate dichotomous variable (1 = yes, 0 = no). Respondents were first asked if a physician had diagnosed them with anxiety or depression; if so, respondents were asked how frequently they sought physician or clinic treatment. Respondents were coded positive if they endorsed currently seeking treatment for the respective condition at least once per year. Our anxiety variable included responses for anxiety and post traumatic stress disorders.

## 2.4. Analytic strategy

Analyses were conducted with logistic regression for dichotomous outcome variables and standard multiple regression for continuous outcome variables. We first tested the linear lifetime adversity term alone, which corresponds to the relationship that has typically been reported in previous research. We subsequently added the term for quadratic lifetime adversity, which provides an appropriate test of the quadratic relationship. To formally assess

the shape of this quadratic relationship (i.e., U-shaped), we tested the momentary simple slopes of the curve at two points, both of which were within the sample's range: a history of no (0) adversity and a value representing "high" adversity. The difference between no and low adversity is important conceptually, making the 0 point on the curve particularly relevant to investigate. We used the value at the mean + 1 *SD* to test the curve at a high level of adversity. A standard choice for continuous predictor variables, this reflects a point in the distribution that is relatively high (i.e., above the mean), yet not so high as to be unreliably extreme [2].

To make results more interpretable, lifetime adversity was divided by its standard deviation and continuous outcome variables were converted to *z*-scores ( $M = 0$ ,  $SD = 1$ ). Coefficients thus reflect effect sizes in units of standard deviations. For dichotomous outcome variables, odds ratios (ORs) represent the relative likelihood of the outcome as a function of each standard deviation change in adversity. For continuous outcome variables, *B*s represent the standard deviations of change in outcome predicted for each standard deviation change in adversity.

Finally, to facilitate interpretation of results for dichotomous outcomes in terms of relative risk, we repeated primary analyses using "modified" Poisson regression (i.e., using robust error variances) instead of logistic regression [54]. The results of these analyses are reported in Table 1.

## 3. Results

### 3.1. Sample characteristics

The CBP sample ( $N = 396$ ) ranged in age from 18 to 87 years old (median 54.5 years), and was 53.0% female. Approximately 81% of the sample self-identified as White (non-Hispanic), 9.0% as Hispanic, 4.6% as African-American (non-Hispanic), and 5.4% as "other", which included Asian. Median household income was \$40,000–\$49,999. Approximately 65% of the sample was married, 15.7% was divorced or separated, 11.8% was single, and 7.2% was widowed. Just over 10% of the sample attained less than a high school degree, 37.5% held a high school degree, 31.9% attended some college, and 20.4% held a college or advanced degree. Approximately 8.6% and 13.1% reported currently seeking treatment for comorbid anxiety and depression, respectively.

The number of lifetime adverse events ranged from 0 to 71 (median 9, interquartile range 8); 3.8% reported experiencing no adverse events. Across all events reported by all participants, the percentage of total events from each category was: 36.4% bereavement, 15.0% loved one's illness or injury, 14.0% violence, 12.4% relationship stress, 8.9% social or environmental stress, 8.6% own illness or injury, and 4.8% disaster. Total adversity count was highly positively skewed, so a natural logarithmic transformation was performed (transformed  $M = 2.22$ ,  $SD = 0.73$ ) to create a distribution that more closely approximated normal and to decrease the influence of extreme scores [47].

### 3.2. Functional impairment

#### 3.2.1. Self-rated functional impairment

We first conducted a standard regression analysis to test the linear relationship between lifetime adversity and self-rated functional impairment as a result of respondents' physical health. Consistent with the sort of relationship typically reported in previous findings, greater adversity predicted greater functional impairment ( $B = 0.11$ ,  $p < 0.05$ ). To investigate the hypothesized quadratic relationship between lifetime adversity and functional impairment, we tested a standard multiple regression model with linear and quadratic lifetime adversity terms predicting functional impairment.

**Table 1**  
Quadratic relationships between cumulative lifetime adversity, functional impairment, and healthcare utilization.

Outcome variable and model terms	B (continuous outcome)	OR (dichotomous outcome)	95% CI	Likelihood ratio for change in model fit $\chi^2$ (df = 1)	Relative risk (dichotomous outcome)
<i>Self-rated functional impairment</i>					
Quadratic lifetime adversity <sup>a</sup>	0.12***	–	0.06, 0.19	14.95***	–
No lifetime adversity	–0.53**	–	–0.87, –0.19	–	–
High lifetime adversity (mean + 1 SD)	0.47***	–	0.26, 0.68	–	–
<i>Disabled employment status (yes/no)</i>					
Quadratic lifetime adversity <sup>b</sup>	–	1.47***	1.26, 1.71	25.78***	1.32***
No lifetime adversity	–	0.14**	0.06, 0.29	–	0.24***
High lifetime adversity (mean + 1 SD)	–	3.13***	1.81, 5.41	–	2.23***
<i>Frequency of physician/clinic back pain treatment</i>					
Quadratic lifetime adversity <sup>c</sup>	0.10**	–	0.04, 0.16	10.90***	–
No lifetime adversity	–0.40*	–	–0.72, –0.08	–	–
High lifetime adversity (mean + 1 SD)	0.40***	–	0.20, 0.60	–	–
<i>Prescription analgesic use for back pain (yes/no)</i>					
Quadratic lifetime adversity <sup>d</sup>	–	1.24**	1.09, 1.41	10.91***	1.13***
No lifetime adversity	–	0.40**	0.20, 0.79	–	0.60**
High lifetime adversity (mean + 1 SD)	–	2.30***	1.47, 3.59	–	1.60***
<i>Over-the-counter analgesic use for back pain (yes/no)</i>					
Quadratic lifetime adversity <sup>e</sup>	–	0.97	0.86, 1.09	0.27	0.98
No lifetime adversity	–	1.18	0.61, 2.28	–	1.10
High lifetime adversity (mean + 1 SD)	–	0.91	0.60, 1.38	–	0.94
<i>Currently seeking treatment for anxiety disorder (yes/no)</i>					
Quadratic lifetime adversity <sup>f</sup>	–	1.24*	1.00, 1.54	3.10	1.15
No lifetime adversity	–	0.57	0.15, 2.17	–	0.80
High lifetime adversity (mean + 1 SD)	–	3.30***	1.82, 5.97	–	2.48***
<i>Currently seeking treatment for depression (yes/no)</i>					
Quadratic lifetime adversity <sup>g</sup>	–	1.27**	1.09, 1.48	8.19**	1.19***
No lifetime adversity	–	0.38*	0.17, 0.88	–	0.50**
High lifetime adversity (mean + 1 SD)	–	2.58***	1.53, 4.34	–	2.01***

Dichotomous outcome variables are coded 1 = yes, 0 = no; odds ratios (ORs) from logistic regression and relative risk values from modified Poisson regression are reported. For continuous outcome variables, Bs from standard regression are reported. The incremental change in overall model fit from adding the quadratic lifetime adversity term to the linear lifetime adversity term in logistic and standard regression was further tested with likelihood ratio tests. This yielded nearly identical patterns of significance as when testing the quadratic coefficient in the model, except that the increase in model fit for currently seeking treatment for anxiety disorder approached ( $p = 0.08$ ) rather than reached significance. For each outcome, “quadratic lifetime adversity” refers to the coefficient for quadratic cumulative lifetime adversity in the model. Under each quadratic term, coefficients represent the simple slopes of the relationship between cumulative lifetime adversity and outcome at no lifetime adversity (0) and high lifetime adversity (mean + 1 SD). ORs and relative risk values less than 1 or negative Bs at no lifetime adversity indicate that as adversity increases from 0, outcomes *improve* (i.e., less impairment and utilization of healthcare); ORs and relative risk values greater than 1 or positive Bs at high lifetime adversity indicate that as adversity increases from 1 SD above the mean, outcomes *worsen* (i.e., greater impairment and utilization of healthcare).

<sup>a</sup> Model  $F(2, 365) = 9.81$ \*\*\*.

<sup>b</sup> Model  $\chi^2(2, N = 396) = 26.37$ \*\*\*.

<sup>c</sup> Model  $F(2, 387) = 07.81$ \*\*\*.

<sup>d</sup> Model  $\chi^2(2, N = 389) = 14.06$ \*\*\*.

<sup>e</sup> Model  $\chi^2(2, N = 389) = 0.27$ .

<sup>f</sup> Model  $\chi^2(2, N = 396) = 18.10$ \*\*\*.

<sup>g</sup> Model  $\chi^2(2, N = 396) = 12.54$ \*\*\*.

\*  $p < 0.05$ .

\*\*  $p < 0.01$ .

\*\*\*  $p < 0.001$ .

As hypothesized, results revealed a significant U-shaped quadratic relationship ( $p < 0.001$ ). To assess the extent of any protective influence of lifetime adversity, we tested the simple slope of the curve at 0, which represented a history of no adversity. This slope was significant in the predicted direction ( $p < 0.01$ ): People with low lifetime adversity reported less impairment than people who had experienced no adversity. To establish the reversal of this relationship, such that additional adversity predicted greater instead of lower impairment, we tested the simple slope at a high level of lifetime adversity (mean + 1 SD) [2]. This slope was significant and in the opposite direction of the no-adversity slope ( $p < 0.001$ ), such that high lifetime adversity predicted greater impairment than low adversity. See Fig. 1A. For statistical details of all quadratic analyses, see Table 1.

### 3.2.2. Employment status

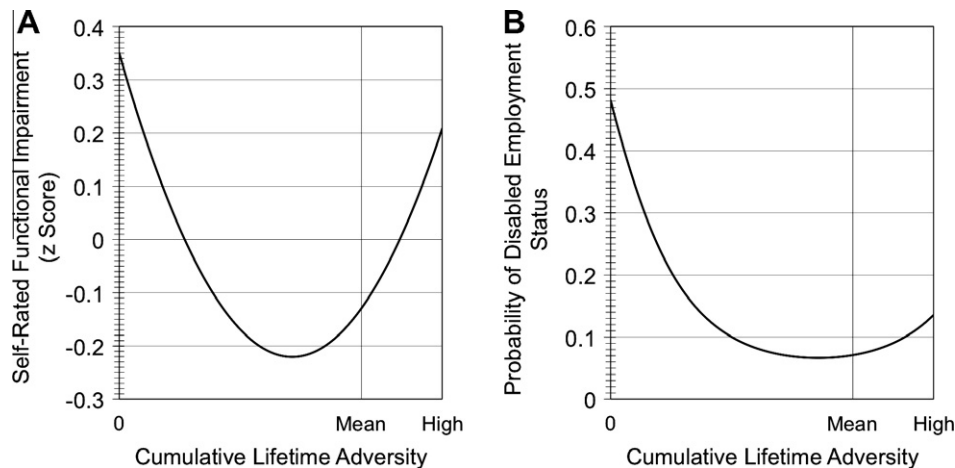
To assess an additional measure of functional impairment, we tested logistic regression models with lifetime adversity predicting

whether or not respondents reported their employment status as “disabled” (11.87% of the sample). When testing only the linear relationship, no significant association emerged. However, when we added the quadratic term to the model, results revealed the expected U-shaped quadratic relationship ( $p < 0.001$ ), such that people with low adversity were less likely to characterize themselves as disabled than both people with a history of no adversity ( $p < 0.001$ ) and those with a high level of adversity ( $p < 0.001$ ). See Fig. 1B.

### 3.3. Healthcare utilization

#### 3.3.1. Frequency of physician/clinic treatment

We examined several facets of healthcare utilization. First, using standard regression, we tested the relationship between lifetime adversity and reported frequency of visits to a physician or clinic for treatment of CBP. Testing the linear relationship revealed a significant association ( $B = 0.11$ ,  $p < 0.05$ ), such that greater



**Fig. 1.** The quadratic relationships between cumulative lifetime adversity and (A) reported functional impairment due to physical health and (B) probability of disabled employment status. *Note:* on the adversity scale, “0” represents no lifetime adversity and “High” represents mean + 1 SD; both points are within the sample’s range.

adversity predicted more frequent utilization of treatment services. However, when we added the quadratic term to the model, the hypothesized U-shaped curve emerged ( $p < 0.01$ ), such that low adversity predicted less frequent utilization of treatment services than both a history of no adversity ( $p < 0.05$ ) and high adversity ( $p = 0.001$ ). See Fig. 2A.

### 3.3.2. Medication use

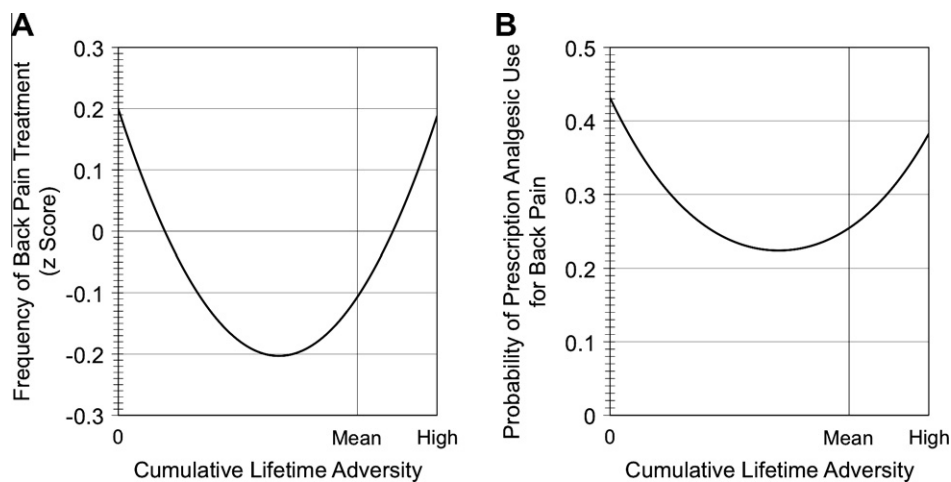
Second, using separate logistic regressions, we tested the relationship between adversity and the use of prescription analgesics (30.59% of the sample) and OTC analgesics (41.65% of the sample; coded 1 = yes, 0 = no) to treat CBP. Results revealed a marginally significant linear relationship between adversity and prescription analgesic use ( $OR = 1.22, p = 0.08$ ), such that greater adversity tended to be associated with greater likelihood of using prescription analgesics. Results also revealed a significant U-shaped curve for prescription analgesic use ( $p < 0.01$ ). Consistent with findings described above, people with low adversity were less likely to use prescription analgesics to treat CBP than either those with a history of no adversity ( $p < 0.01$ ) or those with a high level of adversity ( $p < 0.001$ ). See Fig. 2B. In contrast, no significant effects of adversity emerged for OTC analgesic use.

### 3.3.3. Treatment-seeking for comorbid anxiety disorder and depression

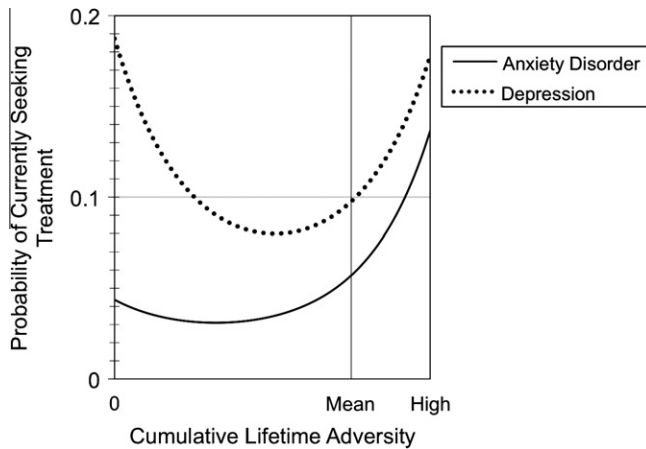
Finally, we used separate logistic regressions to examine the relationship between adversity and the likelihood of seeking treatment for comorbid (1) anxiety disorder (8.59% of the sample) and (2) depression (13.13% of the sample; coded 1 = yes, 0 = no). Significant linear terms emerged for both anxiety disorder ( $OR = 2.34, p < 0.001$ ) and depression ( $OR = 1.40, p < 0.05$ ), such that greater adversity predicted greater likelihood of seeking treatment for these psychiatric conditions. Results also revealed significant quadratic terms for both anxiety disorder ( $p < 0.05$ ) and depression ( $p < 0.01$ ), but only depression revealed a U-shaped curve in which low adversity predicted lower likelihood of treatment seeking than both a history of no adversity ( $p < 0.05$ ) and high adversity ( $p < 0.001$ ). For anxiety disorder, low adversity did not significantly differ from no adversity, but did predict significantly lower likelihood of treatment seeking than high adversity ( $p < 0.001$ ). See Fig. 3.

### 3.4. Alternative explanations for the quadratic pattern

We conducted several additional analyses to address alternative explanations for our quadratic results and to explore additional facets of the research question.



**Fig. 2.** The quadratic relationships between cumulative lifetime adversity and (A) frequency of seeking physician or clinic treatment for CBP and (B) probability of currently using prescription analgesics for CBP. *Note:* on the adversity scale, “0” represents no lifetime adversity and “High” represents mean + 1 SD; both points are within the sample’s range.



**Fig. 3.** The quadratic relationships between cumulative lifetime adversity and probability of currently seeking treatment for comorbid anxiety disorder and depression. Note: on the adversity scale, “0” represents no lifetime adversity and “High” represents mean + 1 SD; both points are within the sample’s range.

#### 3.4.1. Interactions with anxiety disorder and depression status

A possible alternative explanation for our findings is that only respondents seeking treatment for anxiety disorder or depression are responsible for effects, particularly given previously demonstrated relationships between anxiety and/or depression and CBP [14,29]. Indeed, as reported above, we found linear and quadratic relationships between adversity and likelihood of seeking treatment for anxiety and depression. To test the extent to which our results for functional impairment and other healthcare utilization varied as a function of respondents’ anxiety and depression status, we separately examined the interactions between anxiety/depression status and adversity. A significant difference in the shape of the adversity curve between those seeking treatment for anxiety/depression and those not doing so would manifest as a significant interaction. For anxiety, no such interactions approached significance ( $ps > 0.3$ ). For depression, only the interaction predicting prescription analgesic use approached significance ( $p = 0.08$ ), such that the U-shaped adversity curve reported above emerged for respondents *not* seeking treatment for depression ( $p < 0.01$ ), but not for those seeking treatment ( $p = 0.93$ ). No other interactions approached significance ( $ps > 0.2$ ). There is thus no evidence to support that respondents seeking treatment for comorbid anxiety or depression were responsible for the overall pattern of findings.

#### 3.4.2. Excluding potential back injuries from adversity count

Adversities categorized as reflecting injury or illness to oneself, such as direct injury to the back, could conceivably precipitate or exacerbate CBP independent of other adversity, potentially complicating the interpretation of our analyses. To confirm that injuries or illnesses did not unduly influence our findings, we repeated analyses after excluding these events from the total adversity count. This had no meaningful effect on the results, suggesting that adverse events that potentially resulted in back injuries cannot account for our findings.

#### 3.4.3. Adversity history as a proxy for age

It is plausible that lifetime adversity could covary closely with age, given that greater age could increase the opportunity for any given adversity to have occurred at some previous time. In other words, our observed effects could reflect an association between the outcome variables and age rather than lifetime adversity itself. However, age and lifetime adversity were not significantly correlated in our sample ( $r = 0.06$ ,  $p = 0.23$ ). Furthermore, controlling for both linear and quadratic age terms had little effect on the re-

ported results. This suggests that age cannot account for the observed relationship between adversity and outcome variables.

#### 3.4.4. Statistical artifact

To confirm that the natural logarithmic transformation of lifetime adversity count did not create spurious nonlinear relationships at low levels of adversity – the level of primary interest for testing protective effects of adversity – we repeated analyses with an untransformed lifetime adversity count for which values above 10 (1 above the median) were recoded to equal 10. This left low values unaffected while eliminating variability between high values (including extreme scores). Although a less powerful approach than transforming the variable, this allowed us to focus specifically on the differences between no adversity and low adversity by decreasing the influence of high adversity scores on the statistical models. In a pattern consistent with the results described above, significant quadratic terms emerged for self-rated functional impairment ( $p < 0.05$ ), disabled employment status ( $p < 0.001$ ), frequency of CBP treatment ( $p < 0.01$ ), and prescription analgesic use ( $p < 0.01$ ). The slope at a history of no lifetime adversity was significant for all of these quadratic terms ( $ps < 0.05$ ), such that low adversity predicted less impairment and healthcare utilization than no adversity. In contrast to previously described results, quadratic terms for treatment seeking for comorbid anxiety disorder ( $p = 0.39$ ) and depression ( $p = 0.56$ ) did not reach significance. In addition, the quadratic term for OTC analgesic use approached significance ( $p = 0.08$ ). Respondents with low adversity were marginally less likely than those with no adversity to use OTC analgesics ( $p = 0.08$ ). Thus, although these analyses did not fully replicate those using the transformed adversity variable, they do not support the possibility that the observed quadratic relationships were a statistical artifact of transformation.

#### 3.4.5. Currently seeking medical treatment for CBP

An important advantage of our methodology is that it includes respondents who endorsed CBP but had not sought medical treatment for it. However, limiting the sample to only those currently seeking treatment for CBP makes the group more homogeneous and potentially easier to classify. Thus, we repeated analyses using only these respondents ( $N = 173$ ), which yielded a nearly identical pattern of findings, despite the smaller sample and the lower power to detect effects. All relationships were in the same direction and with same significance pattern except for the logistic regression for currently seeking treatment for comorbid depression, in which the quadratic term approached significance ( $p = 0.10$ ) and respondents with low adversity did not significantly differ from those with no adversity ( $p = 0.50$ ). The interaction between quadratic adversity and current treatment-seeking status only reached significance for frequency of currently seeking treatment, which is – by definition – expected. This suggests that the observed quadratic relationships largely hold for this more narrowly defined subsample, and that there is no evidence to support the position that the shapes of the quadratic curves differ significantly as a function of current CBP treatment status.

#### 3.4.6. Childhood adversity

Childhood adversity (e.g., physical or sexual abuse) has been demonstrated to have a particularly negative impact on subsequent development of back pain and its chronicity/refractoriness [23,28,41,42]. In our sample, 24.5% of respondents reported a history of physical or sexual abuse during childhood (i.e., occurring before 18 years of age). Using the total count of child abuse (natural log transformed  $M = 0.24$ ,  $SD = 0.45$ ) to predict our outcome variables revealed the following linear effects, such that more child abuse was associated with greater impairment and healthcare utilization: greater self-rated functional impairment ( $B = 0.10$ ,

$p = 0.05$ ), greater frequency of visits to a physician or clinic for treatment of CBP ( $B = 0.15, p < 0.01$ ), and greater likelihood of seeking treatment for comorbid anxiety disorder ( $OR = 1.99, p < 0.001$ ) and depression ( $OR = 1.80, p < 0.001$ ). The linear relationship between child abuse count and the likelihood of using prescription analgesics approached significance ( $OR = 1.21, p = .07$ ). No other linear or quadratic terms reached significance ( $ps > 0.16$ ). These findings are consistent with previous research in which child abuse has predicted negative consequences for CBP. Importantly, there was no evidence to suggest that greater child abuse itself predicted protection from negative outcomes.

We further examined childhood adversity of other types (i.e., all assessed events except physical or sexual abuse). Using the total count of other adversities experienced during childhood (natural log transformed  $M = 1.00, SD = 0.75$ ) to predict outcomes yielded the following effects for linear adversity, such that greater adversity was associated with greater healthcare utilization: a significant effect for likelihood of seeking treatment for comorbid anxiety disorder ( $OR = 1.56, p < 0.05$ ), a marginally significant effect for comorbid depression ( $OR = 1.29, p = 0.08$ ), and a marginally significant effect for frequency of visits to a physician or clinic for treatment of CBP ( $B = 0.09, p = 0.08$ ). No other linear relationships approached significance ( $ps > 0.15$ ). Examining the quadratic effects of childhood adversity other than physical or sexual abuse revealed significant U-shaped relationships in the form reported above for disabled employment status ( $OR = 1.29, p = 0.05$ ) and seeking treatment for comorbid anxiety ( $OR = 1.56, p < 0.01$ ) and depression ( $OR = 1.37, p = 0.01$ ). No other quadratic relationships reached significance ( $ps > 0.14$ ). Thus, analyses using childhood adversity excluding child abuse yielded findings that partially replicated the quadratic patterns observed for total lifetime adversity.

Finally, we investigated the relationship between a history of physical or sexual child abuse (coded 1 = yes, 0 = no) and the amount of all other lifetime adversity reported. A regression predicting the untransformed total lifetime adversity count excluding child abuse events was significant ( $t[394] = 8.75, p < 0.001$ ), such that respondents with a history of child abuse reported a mean of 6.67 additional other types of adverse events than respondents without a history of childhood physical or sexual abuse. Analogous to the interactions reported above between adversity and depression/anxiety status, we further tested if the nature of the U-shaped adversity curve on functional impairment and healthcare utilization differed significantly between respondents reporting a history of child abuse and those reporting no child abuse. Specifically, we tested for significant interactions between (1) quadratic lifetime adversity excluding child abuse events and (2) child abuse history status (yes versus no). Analyses yielded no significant interactions ( $ps > 0.16$ ). In sum, these results suggest that although any amount of child abuse predicts negative consequences for CBP, low levels of other adversities experienced in childhood can nonetheless predict protection from negative outcomes, and that such protection seems to occur even among those with a history of child abuse.

#### 4. Discussion

Nonspecific back pain in adults can become disabling and is often associated with psychosocial factors [14,49]. Previous research has suggested that a graded relationship exists between adversity exposure and subsequent development of CBP [23] and that exposure to adverse life events correlates with greater CBP severity [4,13,27]. Importantly, in the present investigation we focused not on the etiology of CBP, but instead on predicting outcomes among respondents who already endorsed a history of CBP. Our results yielded linear relationships for self-rated functional impairment and healthcare utilization, such that a history of more

adversity predicted greater impairment and utilization. This pattern is consistent with the results from previous work.

In contrast to the extant literature, however, our findings also suggest that simple linear relationships may obscure adversity's role in protecting against negative consequences of CBP. Specifically, U-shaped curves emerged between lifetime adversity and outcomes, such that low but non-zero levels of adversity were associated with less impairment and less healthcare utilization than both a history of no prior adversity and high levels of adversity.

One potential alternative explanation in research assessing the relationship between adversity and CBP is recall bias. The quadratic relationships we observed cast doubt on recall or reporting biases as an explanation, however. Although such biases could account for why reports of high adversity and high negative outcomes might co-occur [31,52], they cannot account for why respondents who reported no previous adversity reported worse outcomes than respondents who reported some adversity. We also found no evidence to support other alternative explanations for the observed quadratic patterns, including age, comorbid depression and/or anxiety, or statistical artifact.

Our results provide additional insight into the relationships between CBP, adversity, and comorbid depression and anxiety [14,29]. A number of explanations have been hypothesized in previous work: greater adversity predisposing CBP patients to emotional distress [48]; recall biases on the part of anxious/depressed persons leading to heightened endorsement of adversity; or the interaction of emotional distress, physical impairments arising from CBP, and ineffective coping strategies causing individuals to further mismanage their affairs and thereby incur more negative events [4,16]. Consistent with previous research [4,25,26,45], respondents with high levels of lifetime adversity in our study were more likely than those with low levels to report currently seeking treatment for comorbid anxiety and depression. However, our analyses also revealed that for depression, low adversity predicted lower – rather than higher – healthcare utilization compared to no adversity. For anxiety, there was no evidence of greater utilization for low versus no adversity. Furthermore, with the exception of prescription analgesic use for depression, there was no evidence to support differences in the results between respondents who sought treatment for anxiety/depression versus those who did not.

It is noteworthy that the quadratic relationships did not hold for all adversities. The present study extends the findings of previous research underscoring the increased risk of adult CBP among individuals reporting childhood sexual and physical abuse [23,27,28]. Analysis of childhood physical and sexual abuse revealed only linear relationships with greater impairment and healthcare utilization.

We believe several elements of our methodology facilitated finding the quadratic relationships. First, in contrast to previous research, our study assessed a broad array of adverse life events and accounted for multiple instances of each event type. We did so to optimize measurement sensitivity for detecting differences at low levels of adversity. Had we included only few negative experiences, respondents with no and low adversity would have been more likely to yield identical totals, making it impossible to differentiate between them and thereby obscuring the protective effect of low adversity. Second, by temporally separating the assessment of adversity from other measures, we decreased the possibility that current CBP biased recall of adverse life events. Third, our CBP sample was drawn from a population-based panel rather than from specialty pain clinic or hospitalized pain center patients. Clinic samples are self-selected biased samples, which inherently exclude people not seeking treatment for CBP, thereby potentially obscuring differences in functional impairment and healthcare utilization.



Several additional aspects of our adversity measure are worthy of discussion. First, self-reported assessments typically have limitations relative to interviews, including greater intra-category variability and lower reliability [11]. Nonetheless, sensitive topics are more likely to be acknowledged in self-reported assessments of adversity than in interviews [40]. By decreasing social desirability concerns, Web-based data collection improves accuracy of reports over less anonymous methods [40]. Facilitating maximal reporting of events should also help differentiate between low and no adversity. Attaining accurate recall of past adversity is a challenge regardless of methodology. However, as described above, recall bias cannot easily account for a quadratic pattern of results.

Second, our adversity measure represented the number of events experienced rather than the detailed characteristics of events, such as the meaningfulness or emotional significance ascribed to an event. Although relevant variability in adversity may not be captured, simple counts avoid potential ambiguities. For example, isolating effects of single adversities is difficult, given that events are not experienced in a vacuum, but rather in the context of individuals' adversity history [12,15]. Attempting to rate event severity objectively is challenging because everyone experiences adversities differently. What may seem discrete or limited to observers may become chronic or more severe if individuals ruminate about it. Relying on individuals to judge severity for themselves potentially confounds severity with individuals' response to adversity [20]. Nonetheless, more detailed measures of cumulative adversity could provide other important information.

Third, despite its breadth, our adversity measure did not necessarily include all relevant life events. Minor challenges faced in the vicissitudes of life and other events that do not meet standard definitions of "traumatic" can still make important contributions [10,30]. It is thus likely impossible to identify a precise "ideal" number of adverse events.

Other limitations of our work include reliance on self-reported CBP history and lack of direct assessment of depression and anxiety symptoms, which can fail to detect important clinical information, such as CBP specificity and chronicity. In future research, obtaining more detailed assessments of such conditions will be necessary. More sensitive measures should increase power to identify different patterns of effects across patients. Future research could also account for additional chronic pain conditions. Some conditions, such as chronic widespread pain, have been linked to adversity exposure and may possess overlapping characteristics with other conditions [1,19].

We speculate that the observed patterns of quadratic relationships between adversity and CBP-related outcomes may reflect the possibility that resilience, a phenomenon largely ignored in previous CBP research, is occurring. Resilience involves having psychological and social resources that help one tolerate adversity [38]; experiencing some adversity may itself promote development of subsequent resilience and better CBP-related outcomes. Although our data cannot specify the mechanisms linking low adversity with better outcomes, possibilities include that low adversity could teach effective coping skills, help engage social support networks, create a sense of mastery over past adversity, and/or foster beliefs in the ability to cope successfully in the future [32]. For example, experiencing adversity could make subsequent stressful events and daily hassles seem more manageable rather than overwhelming. Symptoms of CBP formerly experienced as stressful and debilitating could be reappraised as minor annoyances that do not substantially interfere with life, leading to lower perceived CBP impairment and need for healthcare. However, higher levels of adversity could negate these benefits by overtaxing coping skills and support networks, and creating feelings of hopelessness and loss of control. Subsequent hassles or adversity would be more likely to seem overwhelming, resulting in higher per-

ceived impairment and need for healthcare. It is also possible that resilience facilitates active rather than passive coping, which has been associated with better CBP outcomes [18]. Future research will be necessary to assess resilience and its influences on the experience of CBP.

It appears that cumulative adversity is related to chronic pain and that this relationship merits additional investigation. These data suggest that some adversity exposure may protect against future impairment and disability, comorbid psychiatric disturbances, and heavy utilization of healthcare. The current findings do not directly address the mechanisms that explain the relationship, nor do they speak to how cumulative adversity relates to the full range of outcomes that are essential to comprehending the experience of the CBP patient. Nonetheless, understanding such mechanisms and their implications for pain management – the ultimate purpose of the present investigation – is necessary for developing individualized treatments and interventions. Promoting resilience may then become one focus of psychotherapeutic approaches [53]. Consistent with our data, resilience may not be attained by avoiding stress entirely, but instead by encountering and managing stressful situations, thereby fostering abilities to overcome subsequent, inevitable challenges during life [39]. In addition, research endeavors have recently investigated the neurobiological bases for resilience, and although these have yet to be completely explicated, some of the presumptive biological substrates are likewise involved in mediating pain transmission [46]. Further research assessing underlying mechanisms and the impact of interventions is an important goal in the context of chronic pain, having the potential to reduce the use of analgesics and medical resources, perceived disability, and comorbid psychopathology associated with pain.

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