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### **Citation**

Gorecki, M. (2025). *The Long-Term Impact of Adverse Childhood Experiences on Chronic Pain in Adulthood*.

Version: Not Applicable (or Unknown)

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Downloaded from: <https://hdl.handle.net/1887/4212862>

**Note:** To cite this publication please use the final published version (if applicable).



Universiteit Leiden

Psychologie  
Faculteit der Sociale Wetenschappen



# The Long-Term Impact of Adverse Childhood Experiences on Chronic Pain in Adulthood

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### Abstract

While the link between adverse childhood experiences (ACEs) and chronic pain has been well established, several aspects of that relationship remain unclear despite the high prevalence of both. Aiming to fill this knowledge gap, we investigated whether ACE severity is associated with chronic pain severity and persistence over time. Moreover, we explored the specificity of ACE types in pain prediction. Finally, the mediating role of post-traumatic stress disorder (PTSD) symptoms in the ACEs–chronic pain relationship was examined. This longitudinal cohort study analyzed the sample from the Netherlands Study of Depression and Anxiety, including 2981 subjects ( $M_{\text{age}} = 41.9$ ,  $SD_{\text{age}} = 13.1$ , 66.4% female). The Childhood Trauma Interview was used at baseline to assess ACE severity and types (sexual, physical, and psychological abuse and emotional neglect). The Chronic Pain Grade Questionnaire measured chronic pain severity and persistence at five measurement points across nine years. After controlling for life events, multiple regression analyses revealed that higher ACE severity was significantly associated with greater chronic pain outcomes ( $p < .001$ ), exhibiting small effect sizes. Pairwise comparison showed that all three abuse types predicted chronic pain with a similar strength but significantly stronger than emotional neglect (all  $p$ 's  $< .025$ ), which was not associated with either pain outcome (all  $p$ 's  $> .492$ ). In mediation analysis, we found positive indirect associations between ACEs and chronic pain through PTSD symptoms. Only the direct effect of sexual abuse on chronic pain persistence was non-significant after including PTSD symptoms ( $p = .091$ ), indicating full mediation. Our results highlight the temporal character of the dose-response relationship between ACE exposure and chronic pain. Furthermore, we provide evidence for the specificity of ACE types in pain prediction, with abuse appearing to have a greater influence than emotional neglect. Lastly, this study supports a mediating role of PTSD symptoms between ACEs and chronic pain. High-quality research is necessary to investigate further the influence of ACE types on chronic pain. The indicated impact of ACEs and PTSD symptoms on chronic pain should be addressed through personalized treatment and prevention strategies, including trauma-informed care.

*Keywords:* adverse childhood experiences, chronic pain, PTSD symptoms, Netherlands Study of Depression and Anxiety

### **Layman's abstract**

Previous research has found that experiencing childhood trauma is linked to chronic pain in adulthood. However, several aspects of that association are still unclear, despite the fact that half of all people have experienced at least one childhood trauma, and chronic pain affects millions. In our study, we investigated whether people who report more trauma also report higher pain scores over time. Next, we tested whether different types of childhood trauma have different effects on chronic pain. Finally, we examined whether symptoms of post-traumatic stress disorder (PTSD), which include distressing memories and anxiety, help explain the relationship between childhood trauma and pain. That means we investigated whether trauma not only influences chronic pain directly but also affects PTSD symptoms, which in turn contribute to chronic pain.

To investigate these aspects, we analyzed a large data set from the Netherlands Study of Depression and Anxiety. In this study, the participants answered questionnaires about their childhood traumas and PTSD symptoms once. The questionnaire about chronic pain was filled in five times over nine years. When we analyzed the data, we found that participants with more childhood trauma reported more chronic pain. We also discovered that different trauma types have different effects: childhood abuse, such as physical or sexual abuse, showed a stronger relationship with chronic pain than neglect, like parental absence. Finally, our results revealed that trauma not only impacts pain directly but also through PTSD symptoms.

Together, these findings provide evidence that childhood trauma is linked to the subjective experience of chronic pain over time. Interestingly, our study highlights that abuse has a stronger impact on chronic pain than neglect, which researchers still debate. The relationship between trauma and pain is very complex and influenced by many factors. This makes it difficult to say precisely how much pain is caused by childhood trauma. Still, the vast majority of research suggests that childhood trauma is a considerable risk factor for the pervasive health condition. Therefore, when treating chronic pain, it is important to consider the effects of trauma and PTSD symptoms.

### **The Long-Term Impact of Adverse Childhood Experiences on Chronic Pain in Adulthood**

Being among the leading causes of disability worldwide, chronic pain is common and shows a global prevalence of 30–50% (Dahlhamer et al., 2018; Treede et al., 2015; Vos et al., 2012). The annual healthcare costs in the United States associated with chronic pain were estimated to be between \$261 and \$300 billion, and the value of lost productivity ranged from \$299 to \$334 billion, exceeding the total costs of heart disease, cancer, and diabetes (Gaskin & Richard, 2012). *Chronic pain* is “an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage” that lasts or recurs for over 3 months (IASP, 2022; Treede et al., 2015). Compared to acute pain, chronic pain has no adaptive purpose and a complex etiology, which has not been sufficiently understood, making efficient treatment challenging (Katz et al., 2015; Turk et al., 2011). However, several risk factors contributing to the development and maintenance of chronic pain have been identified.

Among these risk factors, adverse childhood experiences (ACEs) were suggested to play a crucial role in the occurrence and presentation of chronic pain in adulthood. *ACEs* are potentially traumatic events, such as physical, sexual, and psychological (also referred to as emotional) abuse, as well as emotional neglect, that occur before the age of 18 (Felitti et al., 1998; Tidmarsh et al., 2022). ACE exposure rates are estimated to be 44% in high-income countries (Hillis et al., 2016). On a global scale, each year, every second child is exposed to at least one ACE (Hillis et al., 2016). Further, ACEs have been shown to have a wide range of detrimental, long-lasting effects on an individual's health (Felitti et al., 1998; Gilbert et al., 2009). While deleterious mental health consequences have been extensively researched, physical burdens associated with ACEs have received less scientific attention (Tidmarsh et al., 2022). However, it has been shown that ACEs are related to several physical health conditions, like chronic pain (Noteboom et al., 2021).

A growing body of evidence indicates a severe link between ACEs and chronic pain in adulthood. Two meta-analyses found that individuals reporting abuse or neglect in childhood were more likely to experience chronic pain symptoms and conditions (Bussi eres et al., 2023; Davis et al., 2005). In another study, the chances of chronic pain presentation doubled for people who experienced ACEs (8.7%) compared to those who did not report a history of ACEs (4.6%)

(Groenewald et al., 2020). Additionally, patients suffering from chronic pain reported higher rates of ACE exposure than healthy controls (Davis et al., 2005; Nicolson et al., 2023).

The underlying mechanisms explaining the ACEs–chronic pain relationship involve the interaction between behavioral, neural, and immune processes, which is referred to as psychoneuroimmunology (Irwin & Slavich, 2016). Stressful experiences in early life, such as ACEs, are likely to cause dysregulation of the immune and stress system, leading to an allostatic load and higher inflammation levels in the body, which are associated with the occurrence of chronic pain (Coelho et al., 2014; Ji et al., 2018). Due to a traumatic experience where the coping abilities are exceeded by the faced challenge, e.g., an ACE, the adaptive activation of the hypothalamic-pituitary-adrenal axis in response to acute stress becomes chronic and maladaptive (Lupien et al., 2009).

The stronger the early stress experience, the higher the inflammation rates are, providing a physiological explanation for the well-characterized dose-response relationship between ACE severity and chronic pain (Chen & Lacey, 2018; Danese et al., 2007). It describes the graded fashion of that association, wherein higher ACE severity, determined by both frequency and number of ACEs, is linked to more negative pain outcomes (Bussièrès et al., 2023; Craner et al., 2022; Nicolson et al., 2023; Stickley et al., 2015). ACE severity has been related to clinical complexity, slower recovery, and severity of chronic pain (Tidmarsh et al., 2022). Of the estimated 1.3 billion annual costs arising from ACEs exposure in Europe and North America, 75% were attributed to individuals with two or more ACEs, indicating the high comorbidity of ACEs (Bellis et al., 2019).

Despite these findings, several aspects of the complex relationship between ACEs and chronic pain remain unclear, including the long-term effects of ACEs, the influence of different ACE types, and the role of post-traumatic stress disorder (PTSD).

First, even though a general link between potentially traumatic events in childhood and chronic pain is well established, it is unclear how ACEs impact the pervasive health condition in the long run. While many cross-sectional and case-control studies reported a significant association (Afari et al., 2014; Häuser et al., 2011; Paras et al., 2009), longitudinal research has pointed to a weak or no ACEs–chronic pain relationship (Beal et al., 2020; Raphael & Widom, 2011). A recent systematic review by Marin et al. (2021), exclusively including prospective cohort designs, revealed contradictory results and evaluated the level of evidence as low quality.

The conclusion entailed that there is an urgent need for large prospective cohort studies examining the longitudinal effect of childhood maltreatment on chronic pain.

Second, the relevance of different ACE types in predicting chronic pain has been discussed. Due to their high comorbidity, the specificity of ACE types is challenging to examine (Burke et al., 2017; Gilbert et al., 2009). On the one hand, previous research points to a general impact of ACEs (Beal et al., 2020; Craner et al., 2022; Davis et al., 2005). A meta-analysis by You et al. (2019) found that specific adversity types (sexual, physical, and emotional events) were unrelated to chronic pain levels after controlling for the number of events. On the other hand, a few studies have reported specific effects of ACE types. Specifically, a greater association of physical and sexual abuse with fibromyalgia in adulthood compared to emotional abuse was found in two meta-analyses (Häuser et al., 2011; Kaleycheva et al., 2021). Investigating a Japanese sample from the World Mental Health Survey, the research by Stickley et al. (2015) revealed that out of 11 childhood adversities, only sexual and physical abuse were associated with chronic pain conditions. Other evidence for a specific impact of ACE types stems from a meta-analysis by Tesarz et al. (2020) investigating the impact of PTSD on pain perception. Finding no differences between individuals with PTSD and those without, they reported a significant difference in pain perception among underlying trauma types: Combat-related PTSD was associated with higher pain thresholds, whereas accident-related PTSD predicted lower pain thresholds. The authors concluded that instead of the mere presence of PTSD, the type of underlying trauma might be more relevant for varying pain perception. Although the prediction of chronic pain by specific types of ACEs has not been clarified yet, previous findings indicate that some ACEs, such as sexual and physical abuse, might have a greater impact on chronic pain than other ACEs.

How can the difference in chronic pain prediction among ACE types be explained? One explanation originates from the psychoneuroimmunological perspective, stating an altered immune and stress system as a response to ACE exposure. In their recent review, Cay et al. (2022) postulated two different reactions of the body to early life stress: either an amplifying (hyperalgesia) or suppressing (hypoalgesia) pain reaction. An individual with ACE exposure suffers from hyperalgesia if anxiety arises as the main consequence of the potentially traumatic event. In contrast, hypoalgesia occurs if dissociation is the result of ACE exposure (Defrin et al., 2015). It was suggested that abuse typically leads to anxiety, while neglect and combat

experiences result in dissociation (Cay et al., 2022). Hence, the different pain reactions of the body to a traumatic event—amplifying or suppressing—provide a possible explanation for varying chronic pain development among individuals with different ACE types. Another argument suggesting the specificity of ACE types is that both physical and sexual abuse include a physical component that does not appear in psychological abuse or neglect. In addition to the emotional impact, the physical component was postulated to provide a direct link to chronic pain development (Burke et al., 2017; Katz et al., 2015). Finally, these two adversity types were found to relate more strongly to PTSD, which was proposed to contribute to chronic pain development and its presentation (Koren et al., 2006). Together, the alternating reaction of the body to early life stress, the inclusion of a physical component, and the strong association with PTSD offer an explanation for the possible larger impact of sexual and physical abuse on chronic pain than other types like psychological abuse and neglect.

Lastly, to better understand the ACEs–chronic pain relationship, it is crucial to examine whether and to which extent the traumatic event itself or the response to it contributes to the development and maintenance of chronic pain. As a response to ACE exposure, *PTSD symptoms* such as re-experiencing the trauma, hyperarousal, intrusive thoughts, and avoidance might develop, whose accumulation is classified as PTSD (American Psychological Association, 2025). These symptoms were suggested to contribute to chronic pain by serving as an intermediary through which ACEs result in extended stress reactions (Tidmarsh et al., 2022). Previous studies showed that individuals developing PTSD after a traumatic event are more likely to develop chronic pain than those who do not (Noel et al., 2016; Sareen et al., 2007). However, there is an ongoing scientific debate about whether PTSD and related symptoms mediate the ACEs–chronic pain relationship. On the one hand, some studies suggest that PTSD does not function as a mediator in the association between interpersonal trauma and somatic symptoms (McCall-Hosenfeld et al., 2014). A prospective follow-up study by Raphael and Widom (2011) found that the combined occurrence of abuse or neglect and PTSD increased the risk of developing chronic pain but did not find a mediation role of PTSD. On the contrary, other research found that child maltreatment entirely operated through PTSD symptoms (Alhalal et al., 2018; Beal et al., 2020). Consequently, the evidence of the mediating role of PTSD symptoms is insufficient to draw firm conclusions.

Many previous studies examining the complex ACEs–chronic pain relationship provide evidence for a robust link. Since most evidence comes from cross-sectional studies and the limited longitudinal research shows contradictory results, the long-term effects of ACEs on chronic pain remain unclear. Moreover, the impact of different ACE types and the role of PTSD still lack sufficient understanding. Since these knowledge gaps exist, despite the high prevalence of ACEs (Hillis et al., 2016) and chronic pain (Dahlhamer et al., 2018; Treede et al., 2015), a better clarification of how adverse events in childhood influence the development and occurrence of physical chronic pain is urgently needed. Further understanding might help to improve prevention strategies, such as family-based interventions, and tailor treatment more accurately to individual needs. To achieve this, high-quality longitudinal research is needed first to fill the knowledge gaps of the ACEs–chronic pain relationship.

Therefore, the current study aimed to investigate the long-term impact of ACE severity and types on chronic pain severity and persistence in a large longitudinal sample. Moreover, the mediating role of PTSD symptoms in the ACEs–chronic pain relationship was examined. Specifically, we tested whether (a) higher ACE severity is associated with greater chronic pain severity and persistence, (b) sexual and physical abuse have a stronger impact on chronic pain severity and persistence than psychological abuse and emotional neglect, and (c) PTSD symptoms mediate the relationship between ACE severity as well as types and chronic pain outcomes. To better isolate the influence of childhood adversity on chronic pain, we controlled for important later life stressors.

## **Methods**

### **Design and Sample**

In order to examine the hypotheses, we employed a longitudinal cohort research design to analyze data from the Netherlands Study of Depression and Anxiety (NESDA) (Penninx et al., 2008). Between 2004 and 2007, Dutch citizens aged 18–65 were recruited through mental health care organizations, community settings, and primary care. First, participants received detailed verbal and printed information about the study and provided written consent. Then, participants underwent a half-day face-to-face assessment at baseline, after which four follow-up measurements took place 2, 4, 6, and 9 years later. All measurements comprised medical examination, self-report questionnaires, and cognitive and emotional computer tasks. A follow-up measurement was also conducted 1 year after the baseline, consisting only of written

questionnaires. However, as this follow-up did not include key variables relevant to the present study, we did not incorporate its data into the analysis.

Ensuring representativity only primary clinical diagnosis of a psychiatric disorder (i.e., psychotic disorder, post-traumatic stress disorder, bipolar disorder, obsessive-compulsive disorder, severe addiction disorder, or psychotic disorder) and non-fluency in Dutch were chosen as exclusion criteria. The sample of 2981 Dutch adults at baseline showed retention rates of 87.1% at 2-year follow-up, decreasing to 69.4% after 9 years. A complete description of the assessment procedures can be found in the papers by Penninx et al. (2008, 2021). The NESDA was approved centrally by the Medical Ethical Committee of the VU University Medical Center Amsterdam and carried out following the Declaration of Helsinki.

## **Measures**

### ***ACE Severity and Types***

ACEs were measured retrospectively at baseline with the Childhood Trauma Interview (CTI) developed for the Netherlands Mental Health Survey and Incidence Study (de Graaf et al., 2004, 2010). The CTI was shown to be a valid and reliable measure of interpersonal trauma (Fink et al., 1995). Lasting approximately 10 min, the semi-structured interview assesses the severity of potentially traumatic events before age 16. Participants were shown cards with descriptions of four types of ACEs: psychological abuse (verbally abused, insulted, or threatened), physical abuse (being kicked or hit with hands or an object, beaten up or physically hurt in any other way), sexual abuse (being sexually approached against your will, meaning being touched or having to touch someone sexually) and emotional neglect (lack of parental attention and ignorance of one's problems). Subjects were then asked about the occurrence (yes/no) and the frequency (1 = *once*, 2 = *sometimes*, 3 = *regularly*, 4 = *often*, 5 = *very often*) of each ACE type. For analysis, the responses were recoded into a 3-point scale: 0 = *no*, 1 = *once or sometimes*, 2 = *regularly, often, very often*. By summing the scores (0–2) of each ACE type, the cumulative Childhood Trauma Index (0–8) was obtained, representing ACE severity, and was used for analysis (Hovens et al., 2010).

ACEs were also measured at the 4-year follow-up using the Childhood Trauma Questionnaire (CTQ), a standardized self-report questionnaire including 25 items (Bernstein & Fink, 1998). We chose the CTI scores for analysis instead, as these were assessed at baseline, enabling the inclusion of the data measured before the 4-year follow-up. However, to ensure

good validity, CTI scores were correlated with CTQ scores. Previous studies have shown moderately good correlations between the two instruments (Spinoven et al., 2014).

### ***Chronic Pain Severity and Persistence***

The Chronic Pain Grade Questionnaire (CPG) was used at all measurement points to assess chronic pain outcomes: severity and persistence (Von Korff et al., 1992). The standardized self-report questionnaire is a valid objective instrument for assessing chronic pain during the last 6 months in longitudinal studies (Elliott et al., 2000) and comprises seven items. Chronic pain severity is determined by assessing two pain dimensions: pain intensity and disability. The first three items of the CPG measure pain intensity on a 10-point scale (0 = *no pain* – 10 = *pain as bad as it could be*). The other four items ask about pain disability, with item 4 being an open question (“About how many days in the last 6 months have you been kept from your usual activities (work, school, housework) because of this pain?”) and items 5 to 7 using a 10-point scale (0 = *no change* – 10 = *extreme change*). By averaging and multiplying the scores of items 1 to 3 by 10, a total pain intensity score was calculated, which was repeated with items 5 to 7, resulting in the total pain disability score. After disability points were calculated by adding the points of item 4 to the total pain disability score (see von Korff et al. (1992) for details), the combination of the total pain intensity score and disability points was used to categorize a subject into one of five pain grades: grade 0 (pain-free, no pain in the prior six months), grade I (low disability (<3 disability points), low intensity (<50)), grade II (low disability (<3 disability points), high intensity ( $\geq 50$ )), grade III (high disability, moderately limiting (3–4 disability points)), and grade IV (high disability, severely limiting (5-6 disability points)). The pain grades indicate an individual’s chronic pain severity (Von Korff et al., 1992).

To capture the longitudinal aspect of the current study, the pain grades of all five measurement points were averaged, resulting in a total pain grade (0–5), which was used for analysis. Chronic pain persistence was measured with the open question of item 4. Based on the reported number of days, patients were categorized into one of four categories (0–6 = 0, 7–14 = 1, 15–30 = 2, >30 = 3) (Von Korff et al., 1992), which were averaged across all five measurement points. As with total pain grade, data for chronic pain persistence were included if no more than one measurement time point was missing. The resulting chronic pain persistence score (0–3) was used for analysis.

### ***PTSD Symptoms***

At the 4-year follow-up, PTSD symptoms were assessed with the PTSD Symptom Scale-Interview Version, a 17-item semi-structured interview lasting 20–30 min (Foa et al., 1993). Asking for PTSD symptoms occurring during the last 2 weeks, each item measures one symptom on a 3-point scale (0 = *not at all* – 3 = *very much*), e.g., “Having bad dreams or nightmares about the traumatic event” or “Being overly alert”. By totaling all 17 items, a PTSD symptom severity score (0–51) was determined and used for analysis. The PTSD Symptom Scale-Interview Version has been shown to have excellent validity and reliability (Foa et al., 1993) and correlates strongly with other PTSD measures such as the Clinician-Administered PTSD Scale or the Structured Clinical Interview (Foa & Tolin, 2000).

### ***Covariates and Mental Disorders***

All analyses were controlled for basic covariates such as gender and age at baseline, as well as life events. The prevalence of mental disorders, including anxiety, depression, and borderline pathology, was calculated for descriptive purposes but was not included as a covariate to avoid a restriction of variance.

### ***Life Events***

The List of Threatening Consequences of Brugha assessed important life events at all measurement points (Brugha et al., 1985; Brugha & Cragg, 1990). In a semi-structured interview, participants were asked about 12 different life events with considerable long-term contextual threats, such as serious illness or injury, major financial crises, and separation due to marital difficulties. At baseline, participants were asked about important life events of the past year. At the follow-ups, they were instructed to report the important life events after the last assessment. The initial scoring by the subject is binary (0 = *no*, 1 = *yes*), after which the answers of all 12 items are added up to the life event score. The life event scores of all five measurement points were averaged to obtain the total life event score used for analysis. Data were included if no more than one measurement time point was missing. The widely used List of Threatening Consequences was shown to have sufficient to high validity and strong test-retest reliability (Brugha & Cragg, 1990; Rosmalen et al., 2012).

### ***Mental Disorders***

Classifying disorders according to the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013), the Composite International Diagnostic

Interview (version 2.1) was used to collect diagnoses of anxiety disorder as well as depression. Being the most widely used interview in epidemiological psychiatric studies, the Composite International Diagnostic Interview is a fully structured diagnostic interview with high validity and reliability (Kessler & Üstün, 2004; Wittchen, 1994). At the baseline measurement, patients were asked for lifetime diagnoses (0 = *no*, 1 = *yes*), including all diagnoses until 6 months before baseline.

Borderline pathology was assessed at the 9-year follow-up with the Personality Assessment Inventory (Morey, 2015), a self-administered test containing 24 items rated on a 4-point scale (0 = *false* – 3 = *very true*). Four subscales assess affective instability, negative relationships, identity problems, and self-harm with six items each. A total borderline pathology score (0–72) was obtained by summing all items.

### **Data Analysis Plan**

Before conducting the main analysis, continuous variables (ACE severity, chronic pain severity and persistence, life events, and PTSD symptoms) were winsorized to address outliers ( $z > 3$ ) while retaining all data points (Blaine, 2018). Moreover, the assumptions for parametric tests were checked: linearity, independence, homoscedasticity, normality of residuals, no multicollinearity, and no endogeneity. All assumptions but the normality of residuals were fulfilled. Applying a logarithmic transformation to the pain persistence scores improved residual normality, though some deviations remained (Feng et al., 2014). Descriptive analyses were performed to investigate the characteristics of the sample, including numbers, means, standard deviations, ranges, scale ranges, and frequencies. Lastly, Spearman's rank-order correlations between CTI and CTQ scores were performed to ensure good validity.

To test hypothesis 1, multiple regression analyses were calculated to investigate the relationship between ACE severity and the two chronic pain outcomes. In two separate regressions, it was examined whether the independent variable ACE severity (Childhood Trauma Index of CTI, range 0–8) was associated with the dependent variables chronic pain severity (total pain grade of CPG, range 0–5) and persistence (chronic pain persistence score of CPG, range 0–3). The first hypothesis was confirmed in the case of a significant positive relationship.

Regarding hypothesis 2, two multiple linear regression analyses were performed, one for each chronic pain outcome, including all four ACE types (subscales of CTI, range 0–2) as predictors. Including all ACE types in the models enabled the detection of the unique

contribution of each type, considering their high comorbidity. After that, *t*-tests were used to test for significant differences between the standardized coefficients (betas) for the ACE types of each regression analysis. Standardized regression coefficients were used for comparison as the variables were measured with various scales, and pain persistence scores underwent a logarithmic transformation. In the case of a significant positive beta for physical and sexual abuse being significantly larger than the betas for emotional neglect and psychological abuse, the second hypothesis was confirmed.

Regarding hypothesis 3, for any significant direct effect of ACE severity or ACE types on chronic pain severity and persistence, an additional mediation analysis was performed using model 4 of PROCESS macro to assess the impact of PTSD symptoms (PTSD symptom severity score of the PTSD Symptom Scale-Interview Version, range 0–51) on the association. After calculating the direct effect, the indirect effect of ACE severity and ACE types on the chronic pain variable through PTSD symptoms was computed. Confidence intervals (95%) were calculated using bootstrapping to assess the significance of the indirect effect. The third hypothesis was confirmed if the confidence interval did not include zero.

Statistical significance was determined in all models with  $p < .05$ , and gender, age, and life events (total life event score of the List of Threatening Consequences, range 0–12) were included as control variables. All statistical analyses were performed with two-tailed tests. Bonferroni correction was applied to account for multiple testing, resulting in  $\alpha = 0.025$  for hypotheses 1 and 2 with no adjustment for hypothesis 3. Statistical Package for Social Sciences (Version 29.0.2.0) was used for statistical analysis.

## Results

### Sample Characteristics

The total sample of 2981 subjects had a mean age of 41.9 years ( $SD = 13.0$ ), including 66.4% of female subjects. Half of the sample (48.7%) experienced at least one ACE. The most commonly reported ACE was emotional neglect (38.8%), followed by psychological abuse (24.8%) and sexual abuse (18.4%). Physical abuse was reported least frequently (13.8%). Of the total sample, 59.4% reported a lifetime anxiety diagnosis, and 66.2% had a lifetime depression diagnosis. Other descriptive statistics are depicted in Table 1.

**Table 1***Descriptive Statistics*

Variable	<i>N</i>	<i>M</i>	<i>SD</i>	Range	Scale range
ACE severity	2,970	1.61	2.12	7.97	0–8
Emotional neglect	2,976	0.73	0.94	2.00	0–2
Psychological abuse	2,977	0.45	0.81	2.00	0–2
Sexual abuse	2,976	0.23	0.51	2.00	0–2
Physical abuse	2,979	0.21	0.56	2.00	0–2
Chronic pain severity	2,252	1.52	0.72	3.71	0–5
Chronic pain persistence	2,253	0.34	0.50	1.90	0–3
Life events	2,260	1.43	0.80	3.88	0–12
PTSD symptoms	1,433	16.23	10.61	48.09	0–51
Lifetime anxiety diagnosis <sup>a</sup>	2,981	.59	.49	1.00	0–1
Lifetime depression diagnosis <sup>a</sup>	2,981	.66	.48	1.00	0–1
Borderline pathology	2,256	17.06	10.91	66.00	0–72

*Note.* ACE = adverse childhood experience; PTSD = post-traumatic stress disorder.

<sup>a</sup>Lifetime diagnoses include all diagnoses 6 months before baseline.

### CTI and CTQ Correlations

Spearman's rank-order correlations revealed a significant positive correlation between the total scores of the CTI and the CTQ,  $r_s(2395) = .628, p < .001$ . Similarly, the subscales of both ACE measures were significantly correlated, including emotional neglect ( $r_s(2398) = .55, p < .001$ ), psychological abuse ( $r_s(2398) = .51, p < .001$ ), sexual abuse ( $r_s(2398) = .47, p < .001$ ), and physical abuse ( $r_s(2399) = .51, p < .001$ ).

### ACE Severity and Chronic Pain Outcomes

The multiple regression model, including ACE severity as a predictor, explained 16% of the variance in chronic pain severity,  $R^2 = .16, F(4, 2243) = 107.72, p < .001$ . ACE severity was revealed to have a significant impact on chronic pain severity when controlling for age, gender, and life events (Table 2). In the next multiple regression model, including ACE severity as a

predictor, 10% of the variance in chronic pain persistence was explained,  $R^2 = .10$ ,  $F(4, 2244) = 66.09$ ,  $p < .001$ . After controlling for age, gender, and life events, ACE severity remained a significant predictor (Table 2).

**Table 2**

*Multiple Regression Analyses of the Associations Between ACE Severity and Chronic Pain*

*Outcomes*

Outcome variable	Independent variable	<i>B</i>	<i>SE (B)</i>	$\beta$	95% CI		<i>p</i>	$r_{a(b,c)}^2$ (%)
					<i>LL</i>	<i>UL</i>		
Chronic pain severity	ACE severity	0.062	0.0073	.172	0.0473	0.0760	<b>&lt;.001</b>	2.7
	Life events	0.220	0.0183	.241	0.1837	0.2554	<b>&lt;.001</b>	5.4
	Gender	0.265	0.0299	.172	0.2062	0.3237	<b>&lt;.001</b>	2.9
	Age	0.006	0.0011	.111	0.0040	0.0083	<b>&lt;.001</b>	1.2
Chronic pain persistence	ACE severity	0.008	0.0014	.115	0.0050	0.011	<b>&lt;.001</b>	1.2
	Life events	0.039	0.0036	.228	0.0322	0.0462	<b>&lt;.001</b>	4.8
	Gender	0.035	0.0059	.120	0.0233	0.0463	<b>&lt;.001</b>	1.4
	Age	0.001	0.0002	.088	0.0005	0.0013	<b>&lt;.001</b>	0.8

*Note.* A logarithmic transformation was applied to chronic pain persistence scores. Control variables include life events, gender, and age. The significance threshold was adjusted using Bonferroni correction ( $\alpha = 0.025$ ). Significant *p*-values are in bold.  $N = 2248$ ; CI = confidence interval; *LL* = lower limit; *UL* = upper limit; ACE = adverse childhood experience.

### **ACE Types and Chronic Pain Outcomes**

The multiple regression model, including the four ACE types as predictors, accounted for 17% of the variance in chronic pain severity,  $R^2 = .17$ ,  $F(7, 2240) = 63.37$ ,  $p < .001$ . Psychological abuse, sexual abuse, and physical abuse were revealed to be significant predictors of chronic pain severity after controlling for age, gender, and life events (Table 3). Emotional neglect did not predict chronic pain severity significantly.

**Table 3***Multiple Regression Analyses of the Associations Between ACE Types and Chronic Pain**Outcomes*

Outcome variable	Independent variable	<i>B</i>	<i>SE (B)</i>	$\beta$	95% CI		<i>p</i>	$r_{a(b,c)}^2$ (%)
					<i>LL</i>	<i>UL</i>		
Chronic pain severity	Emotional neglect	0.005	0.0196	.006	-0.0334	0.0436	.795	0.003
	Psychological abuse	0.092	0.0251	.098	0.0425	0.1410	<b>&lt;.001</b>	0.5
	Sexual abuse	0.106	0.0305	.071	0.0461	0.1656	<b>&lt;.001</b>	0.4
	Physical abuse	0.102	0.0332	.072	0.0371	0.1675	<b>.002</b>	0.3
	Life events	0.216	0.0183	.238	0.1806	0.2523	<b>&lt;.001</b>	5.2
	Gender	0.260	0.0302	.170	0.2011	0.3197	<b>&lt;.001</b>	2.6
	Age	0.006	0.0011	.114	0.0042	0.0084	<b>&lt;.001</b>	1.3
Chronic pain persistence	Emotional neglect	-0.003	0.0038	-.018	-0.0102	0.0049	.492	0.02
	Psychological abuse	0.016	0.0049	.090	0.0063	0.0256	<b>.001</b>	0.4
	Sexual abuse	0.014	0.0060	.051	0.0026	0.0259	<b>.017</b>	0.2
	Physical abuse	0.012	0.0065	.044	-0.0011	0.0244	.072	0.1
	Life events	0.039	0.0036	.225	0.0317	0.0457	<b>&lt;.001</b>	4.7
	Gender	0.034	0.0059	.118	0.0226	0.0458	<b>&lt;.001</b>	1.3
	Age	0.001	0.0002	.090	0.0005	0.0014	<b>&lt;.001</b>	0.8

*Note.* A logarithmic transformation was applied to chronic pain persistence scores. Control variables include life events, gender, and age. The significance threshold was adjusted using Bonferroni correction ( $\alpha = 0.025$ ). Significant *p*-values are in bold.  $N = 2247$ ; CI = confidence interval; *LL* = lower limit; *UL* = upper limit.

Follow-up *t*-tests between standardized regression coefficients revealed that the beta for psychological abuse was significantly larger than the beta for emotional neglect,  $t(2244) = -4.21$ ,  $p < .001$ . The betas for physical abuse ( $t(2244) = -2.61$ ,  $p = .010$ ) and sexual abuse ( $t(2244) = -2.39$ ,  $p = .019$ ) were also found to be significantly larger than the beta for emotional neglect. However, the beta for psychological abuse did not significantly differ from the beta for physical abuse ( $t(2244) = 1.08$ ,  $p = .283$ ) or sexual abuse ( $t(2244) = 0.89$ ,  $p = .376$ ). No significant difference was found between the betas for physical abuse and sexual abuse,  $t(2244) = 0.04$ ,  $p = .971$ .

The last multiple regression model, including the four ACE types, predicted chronic pain persistence significantly and accounted for 11% of the variance,  $R^2 = .11$ ,  $F(7, 2241) = 39.13$ ,  $p < .001$ . Controlling for age, gender, and life events, psychological abuse and sexual abuse emerged as significant predictors for chronic pain persistence (Table 3). Physical abuse, approaching significance, and emotional neglect were not significant predictors.

Follow-up *t*-tests comparing standardized regression coefficients showed that psychological abuse had a significantly larger beta than emotional neglect,  $t(2245) = -4.53$ ,  $p < .001$ . Similarly, the betas for physical abuse ( $t(2245) = -2.25$ ,  $p = .025$ ) and sexual abuse ( $t(2245) = -2.31$ ,  $p = .021$ ) were significantly larger than the beta for emotional neglect. In contrast, no significant difference was found between the standardized regression coefficients for psychological abuse and physical abuse,  $t(2245) = 1.84$ ,  $p = .066$ . Additionally, the beta for sexual abuse was not significantly different from the betas for psychological abuse ( $t(2245) = 1.24$ ,  $p = .216$ ) or physical abuse ( $t(2245) = -0.24$ ,  $p = .807$ ).

### **PTSD Symptoms as a Mediator**

Mediation analyses revealed significant positive indirect effects of ACE severity on both chronic pain severity and persistence through PTSD symptoms (Table 4). Furthermore, significant positive indirect effects of psychological, sexual, and physical abuse on chronic pain severity through the mediator were found. Physical and sexual abuse showed a positive indirect effect on chronic pain persistence through PTSD symptoms. The positive direct effects of all ACE variables except for sexual abuse remained significant, suggesting partial mediation. A non-significant direct effect of sexual abuse on chronic pain persistence was found, indicating full mediation through PTSD symptoms.

**Table 4***Mediation Analyses of PTSD Symptoms on the Associations Between ACE Variables and Chronic Pain Outcomes*

Outcome variable	Independent variable	Total effect (c-path)	Effect on PTSD symptoms (a-path)	Effect of PTSD symptoms on chronic pain (b-path)	Direct effect (c'-path)	Indirect effect (axb-path)	% Indirect effect
		$\beta$ <i>p</i>	$\beta$ <i>p</i>	$\beta$ <i>p</i>	$\beta$ <i>p</i>	AB (95% CI)	
Chronic pain severity	ACE severity	.144 <.001	.134 <.001	.147 <.001	.124 <.001	<b>.020</b> <b>[0.0032, 0.0112]</b>	14
	Psychological abuse	.131 <.001	.100 <.001	.151 <.001	.116 <.001	<b>.015</b> <b>[0.0054, 0.0237]</b>	11
	Sexual abuse	.079 .009	.070 .010	.159 <.001	.068 .028	<b>.011</b> <b>[0.0024, 0.0309]</b>	14
	Physical abuse	.149 <.001	.074 .007	.153 <.001	.138 <.001	<b>.011</b> <b>[0.0023, 0.0281]</b>	7
Chronic pain persistence	ACE severity	.087 .001	.134 <.001	.113 <.001	.072 .009	<b>.015</b> <b>[0.0004, 0.0018]</b>	17
	Psychological abuse	.087 .001	.100 <.001	.115 <.001	.075 .005	<b>.012</b> <b>[0.0007, 0.0037]</b>	14
	Sexual abuse	.054 .047	.070 .010	.120 <.001	.046 .091	<b>.008</b> <b>[0.0003, 0.0047]</b>	15

*Note.* A logarithmic transformation was applied to chronic pain persistence scores. Significant indirect effects are in bold.  $N = 1324$ ;

PTSD = post-traumatic stress disorder; ACE = adverse childhood experience; CI = confidence interval.

### Discussion

The present study aimed to investigate the long-term impact of ACEs on chronic pain by using a longitudinal-cohort research design, including a large Dutch sample ( $N = 2981$ ). Additionally, this study examined the mediating role of PTSD symptoms in the ACEs–chronic pain association. Confirming our first hypothesis, we found that higher ACE severity was significantly associated with increased chronic pain severity and persistence. Supporting the second hypothesis, physical and sexual abuse showed a significantly greater impact on both pain outcomes than emotional neglect, which was not associated with either pain outcome. Physical abuse was found to be a non-significant predictor of pain persistence. Against our expectations, physical and sexual abuse did not have a stronger impact on chronic pain than psychological abuse. Lastly, we found that PTSD symptoms positively mediated all significant ACEs–chronic pain associations, confirming our third hypothesis. Whereas the relationship between sexual abuse and chronic pain persistence was fully mediated, all other associations were partially mediated by PTSD symptoms.

The results of our longitudinal study suggest a dose-response relationship between ACE severity and chronic pain outcomes. Although a few studies did not find this cumulative effect of ACE (Lamers-Winkelmann et al., 2012; Raphael et al., 2004), the vast majority of previous studies support our finding (Bussi eres et al., 2023; Craner et al., 2022; Nicolson et al., 2023; Stickley et al., 2015). Whereas most of these studies used a cross-sectional or case-control design, little longitudinal research investigating the ACEs–chronic pain relationship has been conducted (Marin et al., 2021). A recent meta-analysis, including prospective cohort studies, reported contradictory results and low-quality evidence regarding the temporal character of that association (Marin et al., 2021). Therefore, our study significantly contributes to the unanswered question of whether the ACEs–chronic pain relationship lasts over time by revealing a small long-term effect of ACE severity on chronic pain severity and persistence. This finding suggests that bodily reactions to childhood adversity, such as elevated inflammation levels, might persist for several years and lead to enduring physical pain burdens later in life.

Furthermore, our results indicate that all three abuse types predicted chronic pain with similar strength, exhibiting small effect sizes. Contrary to our finding of a generic impact across abuse types, two prior meta-analyses indicated that physical and sexual abuse have a larger impact on chronic pain than emotional abuse (H user et al., 2011; Kaleycheva et al., 2021).

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However, other studies support a non-specific impact across abuse types on chronic pain (Afari et al., 2014; Beal et al., 2020; Bussièrès et al., 2023; You et al., 2019). Together with these findings, our results suggest that the physical component of physical and sexual abuse does not seem to make a significant difference in predicting pain severity and persistence compared to psychological abuse (Burke et al., 2017; Katz et al., 2015). Instead, the involvement of the body might influence the location and type of pain. In a study by Brown et al. (2018), physical and sexual abuse were linked to bodily pain, such as back pain, while emotional abuse was associated with a more general impact on pain across the body.

While revealing a generic influence of abuse, our results also show that all three abuse types had a greater impact on chronic pain severity and persistence than emotional neglect. Aligning with that, Brown et al. (2018) found stronger effects on chronic pain for abuse types than neglect. On the one hand, our results support the postulation that abuse typically leads to hyperalgesia (Cay et al., 2022). All abuse types predicted higher chronic pain outcomes, except physical abuse for pain persistence, which approached significance. On the other hand, our findings contradict the hypothesis that neglect leads to hypoalgesia. Instead, emotional neglect was not associated with chronic pain, which was supported by a recent meta-analysis: Bussièrès et al. (2023) reported that, in sensitivity analysis, only exposure to emotional neglect was no longer significantly associated with chronic pain. Hence, the physiological reactions to emotional neglect appear not to affect an individual's pain experience. Other studies contrast these results by revealing an association between emotional neglect and lower (Brown et al., 2018; Zarchev et al., 2025) and even increased pain levels (Yeung et al., 2016). While our results strongly indicate no impact of emotional neglect on chronic pain, previous research seems contradictory.

There are a few possible reasons for the inconsistent findings regarding the specificity of ACE types in pain prediction. Psychological (or emotional) abuse and neglect were often defined differently (Bussièrès et al., 2023), making the difference in their chronic pain relationship challenging to examine. Further, despite its high prevalence, many studies have failed to include emotional neglect separately in their analysis (Afari et al., 2014; Beal et al., 2020; You et al., 2019). Psychological traumas, including neglect, are sometimes grouped as psychological (or emotional) abuse. This conflation of psychological abuse, which is linked to elevated pain, and emotional neglect, which appears to have no effect, could explain why some former studies

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found a weaker impact of psychological abuse on chronic pain compared to physical and sexual abuse (Häuser et al., 2019).

In conclusion, our study indicates that the long-term effects of ACEs on chronic pain differ based on the specific type of adversity: abuse or neglect. This specificity aligns with prior research showing that different inflammatory biomarkers are released depending on whether an individual experienced abuse or neglect. A meta-analysis by Baumeister et al. (2016) found that an increase in C-reactive protein level was associated with parental absence, while Interleukin-6 and Tumor necrosis factor-alpha elevation was linked to physical and sexual abuse. Of note is that whether the different biomarkers are related to hyper- and hypoalgesia is still unknown.

Moreover, our results suggest that PTSD symptoms serve as a long-term mediator, amplifying the relationship between ACEs and chronic pain. Hence, bodily stress and altered immune reactions to childhood adversity might be extended and compounded by the development of PTSD symptoms. Previous research is inconclusive on whether PTSD functions as a mediator in that relationship (Alhalal et al., 2018; Powers et al., 2014; Raphael & Widom, 2011). The contradictions might be based on varying research designs, sample characteristics, such as age and ethnicity, and the heterogeneity of PTSD (Breslau et al., 2005; Marin et al., 2021; Tesarz et al., 2020). There is consensus in the literature, though, that PTSD is robustly linked to elevated chronic pain (Noel et al., 2016; Sareen et al., 2007), which is in line with our results. Consequently, a complete model of childhood trauma and chronic pain needs to include PTSD symptoms (Marin et al., 2021). Next to that, the effect of PTSD on pain perception has been suggested to depend on the trauma type, especially combat- or accident-related trauma (Defrin et al., 2015; Tesarz et al., 2020). Our results suggest that PTSD symptoms mediate the ACEs–chronic pain relationship regardless of the underlying abuse type. However, only sexual abuse was no longer associated with chronic pain persistence after including PTSD symptoms. One explanation for that might be the strong link between sexual abuse and PTSD (Koren et al., 2006). Considering the logarithmic transformation of the outcome variable and the near-significant direct effect of sexual abuse, this finding should be interpreted with caution.

The present study does not come without limitations. First, it relies on retrospective self-reports of ACEs. Even though retrospective ACE self-reports, such as the CTI, demonstrate good validity and reliability (Dube et al., 2003; Fink et al., 1995), the risk for recall bias remains (Raphael et al., 2004). Abuse experiences are likely to be minimized by subjects, suggesting a

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potential underestimation of ACE exposure (Brewin et al., 1993; Heim et al., 2008). Moreover, the analyzed NESDA sample primarily consisted of Dutch citizens (97%) and showed high rates of mental disorders, especially anxiety and depression, which constrains the generalizability to other samples (Heer et al., 2014; Penninx et al., 2021). Mental disorders have been associated with chronic pain and may affect the relationship between ACEs and chronic pain (Heer et al., 2014; Noteboom et al., 2021). We did not include other potentially influencing factors like ethnicity, pain catastrophizing, or lifestyle (Janssen et al., 2022; Tidmarsh et al., 2022). Since, at baseline, subjects reported life events from the last year, our analysis did not account for earlier life stressors after age 16. Lastly, the results regarding pain persistence should be interpreted cautiously due to the non-normality of residuals and the applied logarithmic transformation.

Noteworthy strengths of our study include its large sample size and longitudinal cohort research design, which provide insight into the temporal relationship between ACEs and chronic pain. Additionally, we included multiple types of abuse and neglect, allowing the investigation of how the nature of ACEs affects chronic pain.

In light of the identified limitations and strengths, various directions for future research are proposed. To reduce the risk of recall bias in ACE assessment, future research should combine official records, such as court documentation, and standardized assessments, including sibling or parent reports (Gilbert et al., 2009). Besides identifying the types of ACE exposure, it is crucial to assess the severity, duration, and age of exposure (Scott-Storey, 2011). In addition, due to the complexity of the ACEs–chronic pain relationship, future studies should use longitudinal designs and account for important confounders like lifestyle, pain catastrophizing, or mental disorders. Also, as our finding that emotional neglect does not influence chronic pain diverges from existing literature, additional studies are necessary to replicate and confirm this result. Upcoming research should examine whether the inflammatory biomarkers associated with specific ACEs are linked to differences in chronic pain. Future studies, including physiological measures of pain, could explore whether types of abuse differentially affect the type and location of chronic pain.

Although the effect size of the ACE predictors in our study was small, their significance should not be underestimated, as the ACEs–chronic pain relationship is intricate and influenced by multiple interrelated factors. Additionally, the association between ACEs and chronic pain may be biased downward as ACE exposure is likely to be underreported in retrospective data

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(Brewin et al., 1993). Consequently, the long-term effect of ACEs on chronic pain demands clinical attention. Increasing clinicians' awareness of the ACEs–chronic pain association is essential for helping patients understand the link between their life experiences and pain burdens, a crucial first step in the treatment process (National Institute for Health and Care Excellence, 2021). Besides that, chronic pain services should focus on trauma-informed care, which follows an integrative biopsychosocial approach to strengthen an individual's empowerment and safety over time (Machtiger et al., 2015). With an emphasis on prevention, the traditional clinical perspective of “What is wrong with you?” is reframed to “What happened to you?” (Menschner & Maul, 2016). Further, trauma screenings for chronic pain patients should aim to identify both various ACE types and the presence of PTSD symptoms. Finally, pain management services may need to consider the specificity of ACE types, and addressing PTSD symptoms should be an integral part of pain treatment.

### **Conclusion**

Overall, our results provide evidence for the long-term impact of ACEs on chronic pain severity and persistence. While the indicated link between ACE severity and the pervasive health condition aligns with previous research, the specificity of ACE types remains debated. Our study suggests that abuse types may have a greater influence on chronic pain than emotional neglect. In addition, the mediating role of PTSD symptoms between ACEs and chronic pain is supported by our results. Despite the need for more high-quality research on the ACEs–chronic pain relationship, the enduring impact of ACEs is significant and requires serious attention in chronic pain treatment.

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