

RESEARCH

Open Access



# Somatic symptoms among young adults: an observational study examining the roles of trauma type and psychological distress

Lauren A. Perez<sup>1</sup> and Yvette Z. Szabo<sup>1\*</sup>

## Abstract

**Background** This study extends previous research examining the interplay between trauma and somatic symptoms by focusing on trauma type (i.e., whether the trauma was interpersonal in nature [e.g., assault, sexual violence, combat] or not) and the extent to which psychological distress accounts for these associations. Additionally, we novelly focus on clusters of somatic symptoms.

**Methods** A sample of predominantly Hispanic/Latinx young adults ( $n = 214$ ) completed a series of brief validated questionnaires assessing demographics, stressful life events (Stressful Life Events Screening Questionnaire – revised), somatic symptoms (Patient Health Questionnaire – 14), and psychological distress (Patient Health Questionnaire – 4) as part of an online survey. Data were first analyzed using linear regression, followed by structural equation modeling to estimate indirect effects, with bootstrapping used to generate confidence intervals.

**Results** Results support a significant indirect effect of interpersonal trauma (IP) on somatic symptoms through psychological distress. While both IP and psychological distress contributed to cardiopulmonary and pain/fatigue clusters, gastrointestinal symptoms were accounted for by psychological distress. Exploratory analyses revealed unique associations by gender, with partial mediation of associations between IP and somatic symptoms by psychological distress observed more clearly in women.

**Conclusions** The present study extends extant research demonstrating that greater exposure to interpersonal trauma exposure is significantly and strongly associated with increased somatic symptoms, psychological distress partially accounts for these associations. With replication, these findings inform theoretical frameworks of the psychological underpinnings of somatic symptom development and can be used to foster advancements in patient care.

**Keywords** Somatic symptom clusters, Interpersonal trauma, Physical health outcomes, Adults, Psychological distress

According to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) traumatic events are those that involve exposure to actual or threatened death, serious injury, or sexual violence [5]. Approximately 70–89% of Western adults will experience a

traumatic event at least once in their lifetime [8, 30]. While many individuals are resilient following a traumatic event [10], many others will go on to develop adverse mental and physical health outcomes (e.g., anxiety, depression, post-traumatic stress disorder or PTSD, somatization; [5, 28, 39, 42]). Existing research on the impact of trauma type has noted that exposure to interpersonal trauma (interpersonal in nature [e.g., assault, sexual violence, combat]) is associated with higher rates of PTSD, suicidality, and increased risk for poor physical health outcomes (e.g., cardiovascular, gastrointestinal,

\*Correspondence:

Yvette Z. Szabo  
yszabo@calstatela.edu

<sup>1</sup> California State University, Los Angeles, 5151 State University Dr, Los Angeles, CA 90032, USA



© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by-nc-nd/4.0/>.

and pain-related illness; [23, 41, 62]). Non-interpersonal traumatic events, such as natural disasters or car accidents are among the most reported of all traumatic events. These events have been associated with negative health outcomes, but to a lesser extent than interpersonal traumatic events for mental health [58]. Further, the association between trauma exposure, particularly trauma type (i.e., interpersonal, non-interpersonal), and physical health outcomes is far less understood.

The present study proposes a comprehensive exploration of the contributions of trauma type as it relates to one domain of physical health, somatic symptoms, which individuals, particularly trauma survivors, often experience without organic or diagnosable cause [31]. Further, these unexplained symptoms account for one-third of medical visits in a primary care setting overall and result in greater unmet clinical needs [25, 32]. In primary care patients, the prevalence rate of psychological disorders has been found to be significantly higher in those with chronic somatic diseases (56.8%) compared to physically healthy patients (48.9% [18]). The experience of somatic symptoms has previously been linked to trauma exposure, regardless of trauma type, but is reported more frequently in survivors of interpersonal trauma [42, 52].

Recent work has shed light on possible pathways linking the experience of trauma to somatic symptoms. Previous research has suggested that the experience of somatic symptoms following trauma is explained by the presence or severity of mental health symptoms (e.g., anxiety, depression, PTSD) [36, 39, 44]. Additionally, recent findings have suggested a linear relationship between the number of physical symptoms reported with the degree of psychological distress [24]. Thus, our study sought to contribute to the understanding of the relationships between trauma exposure, mental health, and somatic symptoms, particularly focusing on differences between interpersonal and non-interpersonal events across the lifespan.

The extant literature has examined somatic symptoms as a total score, characterizing the degree to which participants somaticize in general. However, somatic symptoms have been previously categorized as representing distinct clusters or sets of symptoms. One of the most commonly used measures of somatic symptoms, the Patient Health Questionnaire-15, clusters symptoms into three different subscales: pain or pain/fatigue, cardiopulmonary or symptoms relating to the heart and/or lung, and gastrointestinal (GI) or symptoms related to the stomach and/or intestines [31, 38]. It is plausible that trauma exposure may relate to some clusters more strongly than others. Similarly, psychological distress may account for some,

but not all symptoms. Some preliminary research has found anxiety and depressive disorders to be associated with pain, palpitations, dizziness, nausea [59], and pain-related items associated with depression and anxiety [61]. Associations between symptom clusters and trauma type, and the extent to which psychological distress accounts for these links, have not been examined, and are requisite for later intervention development.

The present study examines the unique contributions of different trauma types on somatic symptoms, as well as the degree to which these associations are accounted for by psychological distress symptoms. This work extends previous literature by considering both interpersonal and non-interpersonal traumatic events among a sample of young adults, who are likely at an age before the onset of many physical conditions. Additionally, the present study fills a critical gap in previous research with a focus on clusters of somatic symptoms. Finally, the current project contributes to the generalizability of these associations by examining them within a predominantly Hispanic/Latinx sample. This builds on other research using predominantly white samples [60] or that did not report race or ethnicity [26, 53]. Additionally, previous research has suggested there may be ethnic/racial differences in stress responses, specifically that Hispanic/Latinx individuals are more likely to perceive and express psychological distress through physical manifestations [1, 25].

We considered covariates of age, gender and SES. Previous research has reported gender differences in trauma exposure [15], somatic symptoms [7], and mental health prevalence (e.g., PTSD, depression, American Psychiatric Association, 2022) [15]. Previous research suggests women report higher somatic symptoms than men even when gynecologic and reproductive symptoms are removed, especially among those with exposure to adverse/traumatic events [39].

Lower SES has been previously correlated with a higher frequency of adverse childhood experiences (ACE; e.g., stressful/traumatic events that occur before the age of 18 – abuse, violence, neglect), which can contribute to adverse physical and psychological health outcomes [37]. Consistent with recommendations not to use race/ethnicity as a proxy [49], we did not include race and ethnicity as a covariate but report this descriptively to characterize our sample. We used first-generation college student status as a proxy for socioeconomic status (SES), as previous research has found that first-generation students tend to come from backgrounds with lower incomes, sustain more student debt, qualify for more financial aid than their non-first-generation counterparts, and often have more limited access to health resources than their peers [46].

## Hypotheses

Based on previous research, we had three primary hypotheses. First, we hypothesized that interpersonal trauma would be associated with higher somatic symptoms but expected this relationship to be less evident for non-interpersonal trauma. Secondly, we hypothesized that psychological distress symptoms would partially, but not completely, account for the association between trauma exposure and somatic symptoms. Lastly, based on limited previous research, we hypothesized that interpersonal trauma would be uniquely associated with higher reporting of gastrointestinal and pain/fatigue clustered symptoms, but not cardiopulmonary symptoms.

## Method

### Participants

Participants were adults 18 years of age and older attending a minority-serving institution in Southern California. Participants were recruited from the psychology subject pool website Sona Systems (Sona Systems, n.d.), where students enrolled in psychology coursework can receive course credit or extra credit for participation in research studies. The present project received approval from the Cal State LA Institutional Review Board. All participants provided informed consent before advancing to the rest of the questionnaire.

A total of 277 participants started the survey, and 214 were retained for analysis. Participants were excluded for completing less than 75% of the survey ( $n = 20$ ), not meeting eligibility criteria ( $n = 22$ ), completing the survey in less than 500 s (8.33 min;  $n = 9$ ), missing both attention check items ( $n = 3$ ) or reporting poor effort in their responses ( $n = 3$ ). We also removed individuals who did not complete the trauma questionnaire, as this was our primary variable of interest ( $n = 6$ ).

Participants in the analytic sample were on average about 20 years old, primarily women and about half identified as first-generation college students. Please see Table 1 for demographics and participant characteristics.

### Measures

#### Demographics

Participants self-reported their date of birth (for calculation of age), race and ethnicity, gender identity, and first-generation college student status. Age and gender were used as covariates in models.

**Table 1** Sample Characteristics

	<i>n</i>	<i>M/SD</i>
Gender		
Women	155 (72.4%)	
Men	53 (24.7%)	
Non-binary	2 (0.93%)	
Prefer not to Say	2 (0.93%)	
Prefer to Self-describe	2 (0.93%)	
Race		
American Indian/Alaska Native	7 (3.3%)	
Asian	18 (13%)	
Black/African American	15 (7.0%)	
Native Hawaiian/Pacific Islander	5 (2.3%)	
White	81 (37.9%)	
Multi-racial	2 (0.93%)	
Ethnicity		
Hispanic/Latino	181 (84.9%)	
Middle Eastern/North African	2 (0.93%)	
Bi/multi-ethnic	4 (1.9%)	
None of these	26 (12.2%)	
First-gen		
Yes	170 (79.4%)	
No	40 (18.6%)	
Not Sure	4 (2.1%)	
Age		19.91 (2.56)
Interpersonal trauma (recoded)		1.32 (1.61)
Non-interpersonal trauma (recoded)		0.73 (0.90)
Somatic symptoms (PHQ-14)		9.24 (5.58)
Cardiopulmonary subscale		2.09 (2.20)
Gastrointestinal subscale		2.00 (1.81)
Pain/Fatigue subscale		4.01 (2.19)
Psychological Distress symptoms		5.10 (3.54)

*N* = 214 except race ( $n = 138$ ), ethnicity ( $n = 213$ ), age ( $n = 200$ ), somatic symptoms ( $n = 213$ ), Gi symptoms ( $n = 212$ ), pain/fatigue symptoms ( $n = 209$ ), distress symptoms ( $n = 210$ ) and PTSD symptoms ( $n = 212$ ). For race/ethnicity, participants could choose all that apply. Further, many participants who were missing data for the race question, were not missing data for the ethnicity questions which may suggest these participants did not believe these items accurately reflected their ethnic/racial identity

### Data quality

There were two attention checks items in the survey and the last question of the survey asked them to rate the extent to which they answered questions to the best of their ability on a scale of 1 = *strongly disagree* to 5 = *strongly agree* (participants with a 1 or a 2 were excluded). Attention check items were added once the study was ongoing, therefore 70 participants did not complete the items, and 207 participants did. As another layer of data quality, we examined the length of time to complete the survey, as well as the percentage of the survey completed for all participants.

### Trauma exposure

Participants completed the Stressful Life Events Scale – Revised [20], which is adapted from Goodman et al. [19]. The questionnaire asked participants whether each of 13 items has happened to them (recoded so 0=no, 1=yes). For the present study's purposes, the researchers created 2 scores by summing items that reflected interpersonal trauma (i.e., physical force/weapon used against you, physically forced to have intercourse, been touched or forced to touch someone else against wishes, repeatedly beat/attacked by caregiver as a child, physically harmed as an adult, family or partner put you down/ignored you, threatened with gun/knife, present when someone else was killed or assaulted, any situation where your life was in danger such as combat) or non-interpersonal trauma (i.e., life-threatening illness, life-threatening accident, a close family member/friend die suddenly, or other horrifying/helpless situation). Possible scores ranged from 0–9 for interpersonal and 0–4 for non-interpersonal. Because these variables were positively skewed with low endorsement at the upper end of the range, they were recoded to 0–6+ for interpersonal and 0, 1, 2, 3+ for non-interpersonal events. This approach has been used in recent research (e.g., [11]) to improve the distribution of scores and maintain interpretability of the scores compared to log transformation.

### Psychological distress symptoms

The 4 item-Patient Health Questionnaire (PHQ-4) was used to assess anxiety (2 items) and depression (2 items) in the past two weeks [33]. Each item is rated on a scale of 0=*not at all* to 3=*nearly every day* in terms of how often the participant experienced that symptom. Scores were summed to create a total score, with higher scores indicating more frequent distress symptoms. The use of PHQ-4 in the present study demonstrated moderate to good reliability and strong internal consistency ( $\omega_{\text{total}}=0.91$ ,  $\omega_{\text{hierarchical}}=0.78$ ). This measure has been previously validated as a clinical screener for depression and anxiety in college students [12, 29], and the total score has historically related to healthcare utilization, functional impairment and disability in community samples [32].

### Somatic symptoms

The Patient Health Questionnaire (PHQ-15) was used to assess physical symptoms [31]. For each item, participants rated how often they were bothered by the listed items during the past four weeks on a scale of 0=*not bothered at all* to 2=*bothered a lot*. Consistent with previous research [22], we calculated a total score by summing together 14 items as one item assesses menstrual

pain and is not relevant to individuals without a uterus. A subsample also completed measures again approximately two weeks later ( $n=94$ ) demonstrating good test–retest reliability ( $\text{ICC}=0.82$ ) and moderate to good reliability and internal consistency within the present sample (PHQ-14  $\omega_{\text{total}}=0.87$ ,  $\omega_{\text{hierarchical}}=0.62$ ). We examined the factor structure of the measure (see below) and used subscale scores as recommended by the original scale authors [32] with includes cardiopulmonary (five items,  $\omega_{\text{total}}=0.75$ ), gastrointestinal symptoms (three items;  $\omega_{\text{total}}=0.78$ ) and pain/fatigue scales (five items;  $\omega_{\text{total}}=0.79$ ).

### Procedures

Participants clicked a link that routed them to Qualtrics which allowed them to complete the survey after reviewing the informational cover letter. Participants were informed of the purpose of the study: to examine the construct validity of several emotion regulation measures within the university community and explore associations between emotion and psychological distress cross-sectionally and over time. It also randomly assigned an ID and automatically granted credit upon survey completion. Data was collected from April to December of 2023.

### Data analysis

For participants missing less than 25% at the scale level for psychological distress or somatic symptoms (i.e., participants skipped an item or two of a questionnaire that measures a theoretical construct), missing data was imputed with the average of the participant's answered items. For items that measure exposure (i.e., trauma exposure) or demographics, no imputation was conducted. Missing data at the scale level was generally low: Somatic symptoms (0.47%), Psychological distress (1.9%), Age (6.5%), First-Gen status (0%) and Gender (0%) as well as Cardiopulmonary symptoms (0%), GI symptoms (0.09%), and Pain/Fatigue symptoms (2.3%). Categorical variables were coded as follows: Gender (1=man, 2=woman, 3=non-binary, 4=prefer not to describe, 5=prefer to self-describe) and First generation status (1=yes, 2=no, 3=not sure). To reduce bias by restricting to only participants with complete data, we retained all participants who had data for at least one IV and one DV in each model, so  $n$  varies by model (particularly for those including covariates) and is clearly noted in the note of a table or via the degrees of freedom of the model. Interpersonal trauma, non-interpersonal trauma, as well as cardiopulmonary were skewed and kurtotic. Skewness and kurtosis were reduced with recoding of the stressful life events measure and normality of residuals was examined for regression models.

All analyses were conducted in R (version 4.3.1). ICC estimates were calculated using R statistical package Psych [48] based on a single-rating ( $k=1$ ), absolute-agreement, two-way mixed-effects model. The first aim was tested with linear regression, with models first run without covariates and then with covariates. To assess the extent to which mental health symptoms account for associations between trauma and somatic symptoms, we followed recommendations by Shrout and Bolger [54]. First, we conducted Pearson correlations to establish an association between trauma (independent variable) and psychological distress (proposed mediator), as well as between psychological distress (mediator) and somatic symptoms (dependent variable). Structural equation modeling was conducted using the lavaan package [50] to examine indirect effects of trauma on somatic symptoms via psychological distress, with bootstrapping used to estimate confidence intervals. Finally, because gender emerged as a significant predictor in several models, we ran exploratory analyses stratifying by gender. Due to a small prevalence of gender minority individuals, we limited analyses to women ( $n=155$ ) and men ( $n=53$ ) only.

## Results

### Descriptive Statistics

Table 1 includes the means and standard deviations of the primary variables of interest. Scores on the somatic symptom measure were consistent with medium somatic symptoms [32]. For the measure of psychological distress, the average score was consistent with mild distress [33].

### Factor analysis

Because several factor structures have been reported for the PHQ-15, we first conducted several CFAs examining the factor structure [14, 31, 38]. As shown in Supplemental Table 1, the models were largely comparable but the 3-factor model by the scale Kroenke and colleagues [32] demonstrated the best-fit indices and was used in analyses.

### Trauma Type, Psychological Distress, Somatic Symptoms

The first aim of the study was to examine the contributions of the frequency of each interpersonal and non-interpersonal traumatic event to somatic symptoms and consider the extent to which psychological distress explains those symptoms. As shown in Table 2, the model with both types of events predicted a significant proportion of the variance in somatic symptoms. However, only interpersonal trauma was a significant individual predictor such that, consistent with hypotheses, greater experience of interpersonal trauma was associated with more somatic symptoms. In contrast, non-interpersonal trauma was not related to somatic symptoms. Results were unchanged in models including covariates, though gender was related to somatic symptoms such that women reported higher levels of symptomology compared to men.

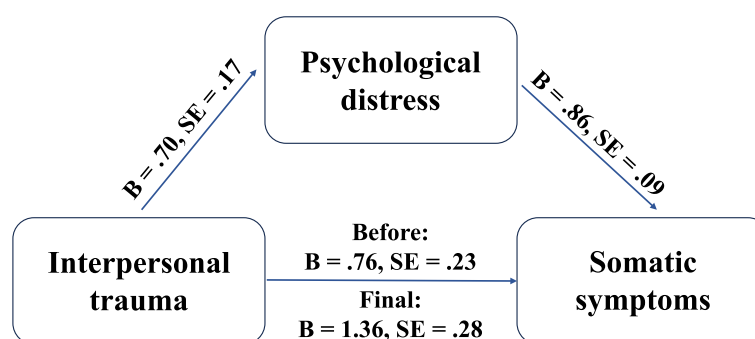
To test whether psychological distress accounts for associations between interpersonal trauma and somatic symptoms, we first examined the correlation between trauma and psychological distress symptoms (i.e., to establish the relationship between IV and conceptual/statistical mediator). This association was significant for interpersonal trauma ( $r(208)=0.37$ ,  $p<0.001$ ) as well as non-interpersonal trauma ( $r(208)=0.27$ ,  $p<0.001$ ). Then, we examined the correlation between psychological distress and somatic symptoms (i.e., the mediator and outcome), which was also significant,  $r(207)=0.64$ ,  $p<0.001$ . Thus, subsequent models included both trauma types. As shown in Table 2, when psychological distress symptoms were included in the model, the model predicted 20% of the variance in somatic symptoms and 25% in the adjusted model. Further, both interpersonal trauma and psychological distress were predictors of somatic symptoms. There was a significant indirect effect of interpersonal trauma on somatic symptoms through psychological distress symptoms ( $b=0.61$ ,  $SE=0.16$ ,  $p<0.001$ ). As represented in Table 2, non-interpersonal

**Table 2** Contributions of Trauma Type

DV: Somatic symptoms	Without covariates				With covariates			
	$F(2, 210) = 26.66$ , $p < .001$ , $R^2 = .20$				$F(5, 194) = 12.83$ , $p < .001$ , $R^2 = .25$			
	B (SE)	$\beta$	$p$	CI of B	B (SE)	$\beta$	$p$	CI of B
Intercept	6.99 (.47)		<.001	[6.06, 7.91]	6.31 (3.58)		.08	[-0.74, 13.37]
Interpersonal	1.34 (.26)	.39	<.001	[0.84, 1.84]	1.37 (.27)	.40	<.001	[0.83, 1.91]
Non interpersonal	0.63 (.45)	.10	.16	[-0.26, 1.53]	.47 (.47)	.07	.32	[-0.50, 1.39]
Gender					1.92 (.61)	.18	.01	[0.52, 3.02]
Age					-0.15 (.15)	-.07	.31	[-0.45, 0.14]
First generation					0.51 (.78)	.04	.52	[-1.03, 2.05]

$\beta$  = standardized regression coefficient; bold = statistically significant





**Fig. 1** Effect of interpersonal trauma on somatic symptoms through psychological distress, adjusted model

trauma was not significant. Thus, interpersonal trauma is related to somatic symptoms, partially due to psychological distress, but also related to somatic symptoms directly (shown in Fig. 1 and Supplemental Table 2). Results were unchanged in models considering covariates and none of the covariates were significant in the model.

### Subscale scores

Our second aim examined the extent to which interpersonal trauma and non-interpersonal trauma are uniquely associated with clusters of somatic symptoms. As shown in Table 3, the models with interpersonal trauma and non-interpersonal trauma were statistically significant, suggesting trauma is significantly associated with cardiopulmonary, GI, and pain/fatigue symptoms. Models predicted between 16 and 17% of the variance in the clusters. Interpersonal trauma, but not non-interpersonal trauma, was the only significant predictor and findings were unchanged when considering covariates. Gender was a significant predictor of GI symptoms and marginally significant for pain/fatigue, such that, women were more likely to experience symptoms than men. Further, experiencing more interpersonal trauma is associated with greater experience of each of the clusters of somatic symptoms; with pain/fatigue and cardiopulmonary symptoms being the most strongly associated, supporting our hypothesis. We next examined the extent to which psychological distress accounted for associations with the clusters of somatic symptoms. As depicted in Table 4, there was also evidence of a significant indirect effect of interpersonal trauma on somatic symptoms through psychological distress for all three clusters. For cardiopulmonary and pain/fatigue symptoms, when psychological distress symptoms were included in the model, both interpersonal trauma and psychological distress were predictors of somatic symptoms. However, for GI symptoms, when psychological distress symptoms were included in the model, interpersonal trauma was no

longer significantly associated with GI symptoms. This suggests that for all subscales, interpersonal trauma is related to somatic symptoms through psychological distress. However, while psychological distress fully accounts for these associations with GI symptoms, interpersonal trauma is uniquely related to the experience of cardiopulmonary and pain/fatigue symptoms. Models were unchanged when covariates were added and none of the covariates were significant in the models.

### Exploratory analyses by gender

Analyses for women can be found in Supplemental Tables 3-6. Broadly, associations with women mirrored the overall pattern for aims 1 or 2 – interpersonal trauma, but not non-interpersonal trauma, was associated with somatic symptoms, and this was partially accounted for by psychological distress. Further, interpersonal trauma was associated with cardiopulmonary, GI, and pain/fatigue symptoms and while psychological distress fully accounted for associations with GI symptoms, it partially accounted for the other subscales. Findings were unchanged after adjusting for covariates.

Analyses for men can be found in the Supplemental Tables 7-10. Two key differences emerged. First, for aim 1, psychological distress fully accounted for associations between interpersonal trauma and somatic symptoms. For the subscales, psychological distress was significantly associated with all three subscales and interpersonal trauma was not. However, the indirect effect did not reach significance until covariates were included. When covariates were added, the mediation effect became significant.

### Discussion

The present study examines the contributions of trauma type (e.g., interpersonal vs. non-interpersonal) on the reporting of somatic symptoms and the degree to which psychological distress accounts for these associations in

**Table 3** Unique associations between trauma type and somatic symptom clusters

	Without covariates			With covariates		
	B (SE)	$\beta$	<i>p</i>	B (SE)	$\beta$	<i>p</i>
<b>Cardiopulmonary</b>						
	$R^2 = .14, F(2, 211) = 17.33, p < .001$			$R^2 = .16, F(5, 194) = 7.63, p < .001$		
Intercept	1.35 (.19)***		< .001	2.14 (1.47)		.15
Interpersonal	.43 (.10)***	.31	< .001	.47 (.11)***	.35	< .001
Non-interpersonal	.23 (.19)	.10	.21	.20 (.19)	.08	.30
Gender				.24 (.26)	.06	.35
Age				-.08 (.06)	-.09	.22
First generation				.23 (.32)	.05	.48
<b>GI</b>						
	$R^2 = .10, F(2, 209) = 10.91, p < .001$			$R^2 = .16, F(5, 193) = 7.12, p < .001$		
Intercept	1.50 (.16)***		< .001	.102 (1.25)		.47
Interpersonal	.30 (.09)***	.26	< .001	.28 (.09)**	.25	.003
Non-interpersonal	.14 (.16)	.07	.37	.11 (.16)	.05	.51
Age				-.04 (.05)	-.05	.48
Gender				.76 (.22)***	.24	< .001
First generation				-.08 (.27)	-.02	.78
<b>Pain/Fatigue</b>						
	$R^2 = .14, F(2, 211) = 18.15, p < .001$			$R^2 = .18, F(5, 194) = 8.42, p < .001$		
Intercept	3.25 (.19)***		< .001	2.47 (1.43)		.08
Interpersonal	.43 (.10)***	.32	< .001	.42 (.11)***	.32	< .001
Non-interpersonal	.22 (.18)	.09	.22	.14 (.19)	.06	.44
Age				-.03 (.06)	-.04	.62
Gender				.64 (.25)**	.17	.01
First generation				.23 (.31)	.05	.46

$\beta$  = standardized regression coefficient; \*\* = significant at the  $p < .01$  level, \*\*\* = significant at the  $p < .001$  level

a racially and ethnically diverse sample of young adults. To the best of our knowledge, this is the first study to investigate these associations by looking at clusters of somatic symptoms – cardiopulmonary, pain/fatigue, and GI.

Consistent with our hypotheses, greater experiences of interpersonal trauma were significantly associated with more somatic symptoms. Importantly, these associations remained virtually unchanged when covariates were added to the model. This extends previous research on interpersonal trauma, which has been predominantly examined within white populations [42], as well as other research that did not focus on trauma across the lifespan [44].

The present study also allowed for empirical examination of the relative contributions of trauma type. Non-interpersonal trauma was similarly correlated with psychological distress and somatic symptoms and generally was in the hypothesized direction in models. However, it was not significantly associated with outcomes when interpersonal trauma was considered. This

may be due to myriad reasons. Interpersonal trauma is linked with greater emotional dysregulation, shame, and are more at risk for reoccurrence than non-interpersonal traumatic events – which are factors that contribute to both the development of psychopathology and poor physical health outcomes [6, 13, 43]. This recurrent nature may suggest that endorsement of events like sexual abuse may represent multiple traumatic events, which could amplify their impact. Additionally, interpersonal trauma is often ongoing, so it is important to consider the possibility of conflation in type and frequency in the measure, given that most traumas assessed in the present study were reported as interpersonal. Further, we must consider whether our findings reflect a restriction of range for non-interpersonal trauma given fewer types of events were assessed. As the timing of trauma may play a significant role in its effects on health, it's crucial to reconcile these findings with studies emphasizing the impact of sensitive periods for trauma exposure (i.e., middle childhood trauma more likely to predict adverse outcomes; [16]). While our study considers trauma across

**Table 4** Effect of interpersonal trauma on somatic symptom clusters through psychological distress

	Without covariates		With covariates	
	B (SE)	p	B (SE)	p
<b>Pain Fatigue</b>				
Indirect	Interpersonal: 0.21 (.06), $p = .001$ Non-interpersonal: 0.11 (.10), $p = .30$		Interpersonal: .20 (.06), $p = .001$ Non-interpersonal: .07 (.10), $p = .50$	
Total	Interpersonal: 0.42 (.10), $p < .001$ Non-interpersonal: 0.19 (.20), $p = .34$		Interpersonal: .42 (.12), $p = .001$ Non-interpersonal: .11 (.21), $p = .62$	
Interpersonal	0.21 (.09)	.02	0.21 (.12)	.07
Non-interpersonal	0.08 (.16)	.61	0.04 (.18)	.83
Distress	0.30 (.04)	< .001	0.29 (.04)	< .001
Gender			0.29 (.38)	.44
Age			-0.01 (.05)	.85
First generation			0.11 (.27)	.69
<b>Cardiopulmonary</b>				
Indirect	Interpersonal: 0.18 (.05), $p < .001$ Non-interpersonal: .10 (.09), $p = .32$		Interpersonal: .18 (.05), $p = .002$ Non-interpersonal: .06 (.09), $p = .51$	
Total	Interpersonal: 0.43 (.12), $p = .001$ Non-interpersonal: .23 (.23), $p = .33$		Interpersonal: .47 (.13), $p < .001$ Non-interpersonal: .20 (.23), $p = .40$	
Interpersonal	0.25 (.12)	.04	0.29 (.13)	.02
Non-interpersonal	0.13 (.20)	.53	0.14 (.21)	.51
Distress	0.27 (.04)	< .001	0.26 (.05)	< .001
Gender			-0.12 (.26)	.64
Age			-0.06 (.06)	.26
First generation			0.21 (.28)	.46
<b>GI</b>				
Indirect	Interpersonal: .15 (.05), $p = .001$ Non-interpersonal: .08 (.08), $p = .28$		Interpersonal: .15 (.05), $p = .001$ Non-interpersonal: .05 (.08), $p = .50$	
Total	Interpersonal: .30 (.09), $p = .001$ Non-interpersonal: .13 (.17), $p = .43$		Interpersonal: .28 (.09), $p = .002$ Non-interpersonal: .09 (.17), $p = .60$	
Interpersonal	0.14 (.08)	.09	0.13 (.09)	.18
Non-interpersonal	0.05 (.14)	.72	0.04 (.15)	.78
Distress	0.22 (.03)	< .001	0.22 (.04)	< .001
Gender			0.48 (.28)	.09
Age			-0.02 (.04)	.58
First generation			-0.14 (.23)	.54

N ranges from 196–209

the lifespan, the average age in our study was 20; we cannot rule out that most participants reported traumatic events occurring before the age of 18. Future research should consider larger samples to compare symptomology in groups with and without childhood trauma and with and without interpersonal trauma to tease apart the contribution of age, trauma type, and critical periods for somatic symptoms.

Our findings support that the experience of interpersonal trauma is associated with related to a greater experience of psychological distress, which in turn is related to the experience of more somatic symptoms, consistent with previous research [42]. Similar to other studies (e.g., [39]), both psychological distress and interpersonal

trauma were statistically significant predictors of somatic symptoms when they were included together, suggesting that psychological distress only partially accounts for the association between trauma and somatic symptoms. While other forms of psychopathology not measured in the current study (e.g., PTSD) might contribute, previous research suggests these do not fully explain the associations either [44]. Loeb et al. [40] proposed that interpersonal trauma may influence somatic symptoms through alternative pathways, which could include psychological factors, such as the emotional experience of guilt and betrayal, or biological mechanisms. For instance, high levels of guilt from interpersonal trauma have been linked to mixed sympathetic and parasympathetic activation



[55]. Additionally, sleep disturbances have been identified as a potential pathway influencing somatic symptom development [2]. Biological research shows that childhood trauma can dysregulate the hypothalamic–pituitary–adrenal (HPA) axis [45], and inflammation has been suggested as a link between trauma and physical health [28]. Further research exploring these potential pathways will inform theory and intervention.

A primary contribution of the present study is the analysis of clusters of somatic symptoms. Interpersonal trauma emerged as a significant predictor of all three subscales, partially supporting the hypothesis and suggesting that the experience of interpersonal trauma is related to a general tendency for individuals to experience heightened somatic symptoms across various domains. Similar to our results using the total score, we observed significant direct and indirect effects for cardiopulmonary symptoms and pain/fatigue. However, the association between interpersonal trauma and GI symptoms was fully accounted for by psychological distress. Previous research has highlighted the link between psychological distress and persistent GI symptoms and frequent healthcare-seeking over time [34], these findings suggest addressing psychological distress may ameliorate symptoms. Some research suggests the type of physical symptom experienced can mean that different physiological systems are involved (e.g., increase in sympathetic nerve activity has been associated with the onset of chronic fatigue; [57]). Additionally, the central nervous system is responsible for modulating sensory, motor, and secretory functions of the GI tract [4], as well as holds a significant role in the second-to-second regulation of cardiac activity [56]. However, future prospective research would be needed to understand the development of these symptoms and whether distinct aspects of trauma and/or individual factors influence the type of somatic symptom experienced.

Another critical contribution of this study was the exploratory analyses stratified by gender. Similar to previous research suggesting women report somatic symptoms more frequently than men, particularly following an interpersonal traumatic event [17], women exhibited higher scores for GI and Pain/Fatigue subscales. Frequency of exposure to interpersonal trauma was more strongly associated with somatic symptoms than non-interpersonal trauma for both men and women. However, this was partially accounted for by psychological distress in women and fully accounted for in men. Further, psychological distress accounted for associations with all three subscales among men and there was no evidence of direct effects of interpersonal trauma in these models. However, indirect effects only became evident when covariates were included in the model though

neither first generation status and age were significant. This may suggest that these covariates, or similar constructs like SES or time since trauma, may be influencing the trauma-symptom relationship in ways not captured by significance alone. A different pattern emerged for women. For women, psychological distress partially accounted for associations when considering pain/fatigue and cardiopulmonary symptoms, mirroring the findings in the main models. Overall, the present results suggest that psychological distress plays a critical role in linking interpersonal trauma with physical health outcomes in both women and men. However, the present findings suggest unique pathways may exist between the experience of trauma and subsequent somatic symptoms based on one's gender and possibly other confounds. It is important to note that these analyses were exploratory and our sample had a relatively larger number of women. It is possible that with a larger sample size of men, a direct association would have been significant or that other more nuanced patterns may have emerged. While our exploratory findings with women have been supported in the broader literature (i.e., women are more likely to develop adverse psychological/physical outcomes post-trauma), replication with a higher sample of men is requisite for the generalization of our exploratory analyses.

### Limitations

Due to the study's cross-sectional design, we cannot determine how these associations might change over time. Additionally, the data's self-report nature means these findings are based on subjective reports. Attention checks were introduced after data collection had already begun, resulting in approximately 25% of participants not completing an attention check; however, all data was screened for quality by excluding responses from participants who did not complete large portions of the survey or completed the survey implausibly quickly. The present study has limited consideration of health covariates, such as the number of diagnosed health conditions or medication use. However, since the average onset of chronic diseases is midlife [21, 27, 51] and our sample's mean age was much below this, it's unclear the extent to which this would impact our findings. Further, the current study had limited assessment of SES. While future research should consider the role of income, education and occupation, there are empirical recommendations suggesting mindful use of SES given its multifaceted construct [49]. Further, the PHQ-15 is a measure of symptom severity, but the symptoms reported due to medically explained and unexplained symptoms can't be distinguished. Future research can consider the impact of trauma on exacerbating already existing medical conditions (e.g., compromised immune system; [47]).

### Future directions and implications

Future research should continue to carefully explore the subtypes of somatic symptoms and their specific associations with trauma exposure and trauma type. In addition to recruiting a larger proportion of men, future studies should adopt a longitudinal approach to better understand how the number, severity, and timing of lifetime traumas relate to the onset and severity of somatic symptoms. Given that psychological distress only partially accounted for somatic symptoms, future research should also consider additional pathways that trauma relates to somatic symptoms, such as biological, social, and psychological pathways highlighted previously or health behaviors.

With replication, the present findings have significant implications for clinical practice as they underscore the importance of psychological determinants of somatic symptoms. One-third of medical visits in a primary care setting are due to somatic symptom complaints that cannot be medically explained [25]. Recent meta-analyses have found that prevention efforts such as education on symptoms, and treatments ranging from cognitive-behavioral therapy to biofeedback have been effective in reducing both somatic symptoms and healthcare utilization [9]. Because psychological distress contributed to all clusters of symptoms and accounted for associations between trauma and GI symptoms, it is important to consider referrals for mental health when a physiological explanation is not present. Across sex and gender, referral for psychiatric treatment may improve symptoms [9], but may not be sufficient for cardiopulmonary, pain/fatigue symptoms, particularly among women. Existing treatments for somatic symptoms (e.g., pain) usually include medication and mindfulness practices, but empirical research highlights adverse effects in trauma survivors, particularly when it comes to feelings related to anxiety [3]. Therefore, clinicians should consider mental health symptoms (e.g., depression, anxiety, PTSD) and concurrent trauma exposure when providing care for adults who present with somatic symptoms. Further, a recent review has suggested that body-orientated therapeutic approaches (i.e., somatic experiencing) in treating post-traumatic symptoms have a positive impact on both affective and somatic symptoms and well-being measures in both traumatized and non-traumatized samples [35]. Nonetheless, more research on interventions suitable for trauma-exposed individuals who present with somatic symptoms is merited.

### Conclusion

This study extends a body of research demonstrating that greater exposure to interpersonal trauma exposure is significantly and strongly associated with increased somatic symptoms. Further, psychological distress partially accounts for these associations. However, this study's examination of symptom clusters and gender provides a more nuanced understanding that can serve as a foundation for future research and theory development. Unique gender findings in the present study suggest that gender may play a significant role in how trauma manifests physically. Associations in the exploratory analysis were more consistently seen in women than men. However, among men these were fully accounted for by psychological distress. By refining somatic symptom patterns with future research, the underlying mechanisms linking clusters to trauma exposure can be more easily identified and help inform clinical prevention and intervention efforts. Future studies utilizing a longitudinal approach are requisite for a more advanced understanding of the links between trauma and somatic symptoms and how they evolve. Consequently, deepening the comprehension of the psychological mechanisms that contribute to physical health outcomes will strengthen the foundation for improving patient care. More specifically, identifying specific patterns in symptom development can provide a path for researchers and clinicians to establish effective treatment plans that are tailored to an individual patient's needs, reduce symptom burden, and optimize the health and well-being of trauma survivors.

### Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s40359-025-02504-7>.

Supplementary Material 1.

### Acknowledgements

Acknowledgements The NIH Bridges to the Doctorate fellowship (T32-GM-146700) and CSU Sally Casanova California Pre-Doctoral Scholarship supported the first author.

### Authors' contributions

YZS designed the study and collected the data. LAP conducted an extensive literature search and investigated theoretical frameworks. LAP and YZS analyzed and interpreted data and contributed to the development of the manuscript. The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

### Funding

National Institutes of Health, T32-GM-146700.

### Data availability

Data for the study is available upon reasonable request and ethics board approval by contacting the corresponding author.

## Declarations

### Ethics approval and consent to participate

The present project received approval from the Cal State LA institutional review board (IRB; exempt protocol #1962543) in accordance with the Common Rule on April 19th, 2023. All participants provided informed consent for their participation.

### Consent for publication

Not Applicable.

### Competing interests

The authors declare no competing interests.

Received: 5 December 2024 Accepted: 18 February 2025

Published online: 26 March 2025

## References

- Abarca G, Tornberg-Belanger S, Ryan D, Price C, Rao D, Ornelas I. Understanding the relationship between social stressors, trauma and somatic symptoms among Latina immigrant women. *Racial Ethnic Health Disparities*. 2022;10(1):387–94.
- Ablin JN, Clauw DJ, Lyden AK, Ambrose K, Williams DA, Gracely RH, et al. Effects of sleep restriction and exercise deprivation on somatic symptoms and mood in healthy adults. *Clin Exp Rheumatol*. 2013;31(6 Suppl 79):S53–9.
- Aizik-Reebs A, Shoham A, Bernstein A. First, do no harm: An intensive experience sampling study of adverse effects to mindfulness training. *Behav Res Ther*. 2021;145:103941. <https://doi.org/10.1016/j.brat.2021.103941>.
- Altat MA, Sood MR. The nervous system and gastrointestinal function. *Dev Disabil Res Rev*. 2008;14(2):87–95. <https://doi.org/10.1002/ddrr.15>.
- American Psychiatric Association. Diagnostic and statistical manual of mental disorders (5th ed., text rev.). American Psychiatric Publishing; 2022. <https://doi.org/10.1176/appi.books.9780890425787>.
- Anda RF, Porter LE, Brown DW. Inside the Adverse Childhood Experience Score: Strengths, Limitations, and Misapplications. *Am J Prev Med*. 2020;59(2):293–5. <https://doi.org/10.1016/j.amepre.2020.01.009>.
- Barsky AJ, Peekna HM, Borus JF. Somatic symptom reporting in women and men. *J Gen Intern Med*. 2001;16(4):266–75. <https://doi.org/10.1046/j.1525-1497.2001.016004266.x>.
- Benjet C, Bromet E, Karam EG, Kessler RC, McLaughlin KA, Ruscio AM, et al. The epidemiology of traumatic event exposure worldwide: Results from the World Mental Health Survey Consortium. *Psychol Med*. 2016;46(2):327–43. <https://doi.org/10.1017/S0033291715001981>.
- Berezowski L, Ludwig L, Martin A, Löwe B, Shedden-Mora MC. Early Psychological Interventions for Somatic Symptom Disorder and Functional Somatic Syndromes: A Systematic Review and Meta-Analysis. *Psychosom Med*. 2022;84(3):325–38. <https://doi.org/10.1097/PSY.0000000000001011>.
- Bonanno GA, Westphal M, Mancini AD. Resilience to Loss and Potential Trauma. *Annu Rev Clin Psychol*. 2011;7(1):511–35. <https://doi.org/10.1146/annurev-clinpsy-032210-104526>.
- Burns C, Hejl C, Szabo Y. Childhood adversity and adult inflammation: Exploring the mediating role of emotion regulation in the MIDUS II study. *J Child Adol Trauma*. 2024;17:319–34. <https://doi.org/10.1007/s40653-023-00594-2>.
- Byrd-Bredbenner C, Eck K, Quick V. GAD-7, GAD-2, and GAD-mini: Psychometric properties and norms of university students in the United States. *Gen Hosp Psychiatry*. 2021;69:61–6. <https://doi.org/10.1016/j.genhosppsych.2021.01.002>.
- Crowell SE, Puzia ME, Yaptangco M. The ontogeny of chronic distress: emotion dysregulation across the life span and its implications for psychological and physical health. *Curr Opin Psychol*. 2015;3:91–9. <https://doi.org/10.1016/j.copsyc.2015.03.023>.
- Dadfar M, Asgharnejadfarid A, Hosseini A, Esfahani MN, Lester D, Kalibatseva Z. Measuring somatic symptoms with the PHQ-15: a comparative study of three Iranian samples. *Mental Health Relig Cult*. 2020;23:1–13. <https://doi.org/10.1080/13674676.2020.1718069>.
- Ditlevsen DN, Elklit A. Gender, trauma type, and PTSD prevalence: a re-analysis of 18 nordic convenience samples. *Ann Gen Psychiatry*. 2012;11(1):26. <https://doi.org/10.1186/1744-859X-11-26>.
- Dunn EC, Nishimi K, Gomez SH, Powers A, Bradley B. Developmental timing of trauma exposure and emotion dysregulation in adulthood: Are there sensitive periods when trauma is most harmful? *J Affect Disord*. 2018;227:869–77. <https://doi.org/10.1016/j.jad.2017.10.045>.
- Eslami B, Di Rosa M, Barros H, Torres-Gonzalez F, Stankunas M, Ioannidi-Kapoulou E, et al. Lifetime abuse and somatic symptoms among older women and men in Europe. *PLoS One*. 2019;14(8). <https://doi.org/10.1371/journal.pone.0220741>.
- Gili M, Comas A, Garcia-Garcia M, Monzon S, Antoni SB, Roca M. Comorbidity between common mental disorders and chronic somatic diseases in primary care patients. *Gen Hosp Psychiatry*. 2010;32(3):240–5. <https://doi.org/10.1016/j.genhosppsych.2010.01.013>.
- Goodman L, Corcoran C, Turner K, Yuan N, Green B. Assessing traumatic event exposure: General issues and preliminary findings for the Stressful Life Events Screening Questionnaire. *J Traumat Stress*. 1998;11(3):521–42.
- Green B, Chung J, Daroowalla A, Kaltman S, DeBenedictis C. Evaluating the Cultural Validity of the Stressful Life Events Screening Questionnaire. *Violence Against Women*. 2006;12(12):191–213.
- Harlow SD, Derby CA. Women's Midlife Health: Why the Midlife Matters. *Womens Midlife Health*. 2015;1(1):5. <https://doi.org/10.1186/s40695-015-0006-7>.
- Hinz A, Ernst J, Glaesmer H, Brähler E, Rauscher FG, Petrowski K, et al. Frequency of somatic symptoms in the general population: Normative values for the Patient Health Questionnaire-15 (PHQ-15). *J Psychosom Res*. 2017;96:27–31. <https://doi.org/10.1016/j.jpsychores.2016.12.017>.
- Husarewycz MN, El-Gabalawy R, Logsetty S, Sareen J. The association between number and type of traumatic life experiences and physical conditions in a nationally representative sample. *Gen Hosp Psychiatry*. 2014;36(1):26–32. <https://doi.org/10.1016/j.genhosppsych.2013.06.003>.
- Hyphantis T, Goulia P, Carvalho AF. Personality traits, defense mechanisms and hostility features associated with somatic symptom severity in both health and disease. *J Psychosom Res*. 2013;75(4):362–9. <https://doi.org/10.1016/j.jpsychores.2013.08.014>.
- Interian A, Allen LA, Gara MA, Escobar JI, Díaz-Martínez AM. Somatic Complaints in Primary Care: Further Examining the Validity of the Patient Health Questionnaire (PHQ-15). *Psychosomatics*. 2006;47(5):392–8. <https://doi.org/10.1176/appi.psy.47.5.392>.
- Jacob L, Haro JM, Koyanagi A. Post-traumatic stress symptoms are associated with physical multimorbidity: Findings from the Adult Psychiatric Morbidity Survey 2007. *J Affect Disord*. 2018;232:385–92. <https://doi.org/10.1016/j.jad.2018.02.063>.
- Kase NG, Gretz Friedman E, Brodman M. The midlife transition and the risk of cardiovascular disease and cancer Part II: strategies to maximize quality of life and limit dysfunction and disease. *Am J Obstet Gynecol*. 2020;223(6):834–847.e2. <https://doi.org/10.1016/j.ajog.2020.06.008>.
- Kendall-Tackett K. Psychological trauma and physical health: A psychoneuroimmunology approach to etiology of negative health effects and possible interventions. *Psychol Trauma Theory Res Pract Policy*. 2009;1(1):35–48. <https://doi.org/10.1037/a0015128>.
- Khubchandani J, Brey R, Kotecki J, Kleinfelder J, Anderson J. The Psychometric Properties of PHQ-4 Depression and Anxiety Screening Scale Among College Students. *Arch Psychiatr Nurs*. 2016;30(4):457–62. <https://doi.org/10.1016/j.apnu.2016.01.014>.
- Kilpatrick DG, Resnick HS, Milanak ME, Miller MW, Keyes KM, Friedman MJ. National Estimates of Exposure to Traumatic Events and PTSD Prevalence Using DSM-IV and DSM-5 Criteria: DSM-5 PTSD Prevalence. *J Trauma Stress*. 2013;26(5):537–47. <https://doi.org/10.1002/jts.21848>.
- Kroenke K, Spitzer RL, deGruy FV, Swindle R. 15-item Patient Health Questionnaire (PHQ-15) [Database record]. *APA PsycTests*; 1998. <https://doi.org/10.1037/t19590-000>.
- Kroenke K, Spitzer RL, Williams JBW. The PHQ-15: Validity of a New Measure for Evaluating the Severity of Somatic Symptoms. *Psychosom Med*. 2002;64(2):258–266. <https://doi.org/10.1097/00006842-200203000-00008>.
- Kroenke K, Spitzer RL, Williams JB, Löwe B. An ultra-brief screening scale for anxiety and depression: the PHQ-4. *Psychosomatics*. 2009;50(6):613–21. <https://doi.org/10.1176/appi.psy.50.6.613>.

34. Koloski NA, Talley NJ, Huskic SS, Boyce PM. Predictors of conventional and alternative health care seeking for irritable bowel syndrome and functional dyspepsia. *Aliment Pharmacol Ther.* 2003;17:841–51. <https://doi.org/10.1046/j.0269-2813.2003.01498.x>.
35. Kuhful M, Maldei T, Hetmanek A, Baumann N. Somatic experiencing – effectiveness and key factors of a body-oriented trauma therapy: A scoping literature review. *Eur J Psychotraumatol.* 2021;12(1). <https://doi.org/10.1080/2008198.2021.1929023>.
36. Lee RY, Oxford ML, Sonney J, Enquobahrie DA, Cato KD. The mediating role of anxiety/depression symptoms between adverse childhood experiences (ACEs) and somatic symptoms in adolescents. *J Adolesc.* 2022;94(2):133–47. <https://doi.org/10.1002/jad.12012>.
37. Lee RY, Oxford ML, Sonney J, Enquobahrie DA, Cato KD. Relationships between recent adverse childhood experiences (ACEs) and somatic symptoms in adolescence. *J Child Fam Stud.* 2024;33:1015–28. <https://doi.org/10.1007/s10826-024-02812-3>.
38. Liao S, Huang W, Ma H, Lee M, Chen T, Chen I, et al. The relation between the patient health questionnaire-15 and DSM somatic diagnoses. *BMC Psychiatry.* 2016;16(1):351. <https://doi.org/10.1186/s12888-016-1068-2>.
39. Loeb TB, Joseph NT, Wyatt GE, Zhang M, Chin D, Thames A, et al. Predictors of somatic symptom severity: The role of cumulative history of trauma and adversity in a diverse community sample. *Psychol Trauma Theory Res Pract Policy.* 2018;10(5):491–8. <https://doi.org/10.1037/tra000334>.
40. Loeb EL, Tan JS, Hessel ET, Allen JP. Getting What You Expect: Negative Social Expectations in Early Adolescence Predict Hostile Romantic Partnerships and Friendships Into Adulthood. *J Early Adolesc.* 2018;38(4):475–96. <https://doi.org/10.1177/0272431616675971>.
41. Luthra R, Abramovitz R, Greenberg R, Schoor A, Newcorn J, Schmeidler J, et al. Relationship Between Type of Trauma Exposure and Posttraumatic Stress Disorder Among Urban Children and Adolescents. *J Interpers Violence.* 2009;24(11):1919–27. <https://doi.org/10.1177/0886260508325494>.
42. McCall-Hosenfeld JS, Winter M, Heeren T, Liebschutz JM. The association of interpersonal trauma with somatic symptom severity in a primary care population with chronic pain: exploring the role of gender and the mental health sequelae of trauma. *J Psychosom Res.* 2014;77(3):196–204. <https://doi.org/10.1016/j.jpsychores.2014.07.011>.
43. Mirabile M, Gnatt I, Sharp J, Mackelprang J. Shame and emotion dysregulation as pathways to posttraumatic stress symptoms among women with a history of interpersonal trauma. *J Interpers Violence.* 2024;39(7–8):1853–76. <https://doi.org/10.1177/08862605231211924>.
44. Morina N, Schnyder U, Klaghofer R, Müller J, Martin-Soelch C. Trauma exposure and the mediating role of posttraumatic stress on somatic symptoms in civilian war victims. *BMC Psychiatry.* 2018;18(1):92. <https://doi.org/10.1186/s12888-018-1680-4>.
45. Murphy F, Nasa A, Cullinane D, Raajakesary K, Gazzaz A, Sooknarine V, et al. Childhood Trauma, the HPA axis and psychiatric illnesses: A targeted literature synthesis. *Front Psych.* 2022;13:748372. <https://doi.org/10.3389/fpsy.2022.748372>.
46. Nam J. First-generation college students: facts and statistics. Best Colleges; 2023. <https://www.bestcolleges.com/research/first-generation-students-facts-statistics/>.
47. Napolitano L, Faist E, Wichmann M, Coimbra R. Immune dysfunction in trauma. *Surg Clin North Am.* 1999;79(6):1385–416. [https://doi.org/10.1016/S0039-6109\(05\)70084-0](https://doi.org/10.1016/S0039-6109(05)70084-0).
48. Revelle W. psych: Procedures for Psychological, Psychometric, and Personality Research. Evanston: Northwestern University; 2024. R package version 2.4.12. <https://CRAN.R-project.org/package=psych>.
49. Ross P, Hart-Johnson T, Santen S, Zaidi N. Considerations for using race and ethnicity as quantitative variables in medical education research. *Perspectives on Medical Education.* 2020;9(5):318–23. <https://doi.org/10.1007/S40037-020-00602-3>.
50. Rosseel Y, Loh WW. A structural after measurement approach to structural equation modeling. *Psychol Methods.* 2024;29(3):561–88. <https://doi.org/10.1037/met0000503>.
51. Ryff C, Singer B, Dienberg G. Positive health: connecting well-being with biology. *Philos Trans R Soc Lond B Biol Sci.* 2004;359(1449): 1383–1394. <https://doi.org/10.1098/rstb.2004.1521>.
52. Sack R, Auckley D, Auger R, Carskadon M, Wright K, Vitiello M, et al. Circadian Rhythm Sleep Disorders: Part I, Basic Principles, Shift Work and Jet Lag Disorders. *Sleep.* 2007;30(11):1460–83. <https://doi.org/10.1093/sleep/30.11.1460>.
53. Sadeghi S, Dolatshahi B, et al. Relationship Between Traumatic Experiences and Somatic Symptoms Severity in Students. *Pract Clin Psychol.* 2017;5(3):211–6. <https://doi.org/10.18869/acadpubjpcp.5.3.211>.
54. Shrout P, Bolger N. Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychol Methods* 7(4). Am Psychol Assoc. 2002:422–45. <https://doi.org/10.1037/1082-989X.7.4.422>.
55. Stewart C, Mitchell D, MacDonald P, Pasternak S, Tremblay P, Finger E. The psychophysiology of guilt in healthy adults. *Cogn Affect Behav Neurosci.* 2023;23(4):1192–209. <https://doi.org/10.3758/s13415-023-01079-3>.
56. Talman W. Cardiovascular regulation and lesions of the central nervous system. *Ann Neurol.* 1985;18(1):1–13. <https://doi.org/10.1002/ana.410180102>.
57. Tanaka M, Tajima S, Mizuno K, Ishii A, Konishi Y, Miike T, et al. Frontier studies on fatigue, autonomic nerve dysfunction, and sleep-rhythm disorder. *J Physiol Sci.* 2015;65(6):483–98. <https://doi.org/10.1007/s12576-015-0399-y>.
58. Thomas EA, Owens GP, Keller EM. Relationships among non-interpersonal and interpersonal trauma types, posttraumatic stress, and posttraumatic growth. *J Clin Psychol.* 2021;77:2592–608. <https://doi.org/10.1002/jclp.23190>.
59. Tsai C. Factor analysis of the clustering of common somatic symptoms: a preliminary study. *BMC Health Serv Res.* 2010;10(1):160. <https://doi.org/10.1186/1472-6963-10-160>.
60. Wamser R, Ferro R. Cumulative trauma, posttraumatic stress, and obstetric and perinatal outcomes. *Psychol Trauma.* 2023. <https://doi.org/10.1037/tra0001579>.
61. Xie Y, Shen Z, Zhu X, Pan Y, Sun H, Xie M, et al. Infralimbic-basolateral amygdala circuit associated with depression-like not anxiety-like behaviors induced by chronic neuropathic pain and the antidepressant effects of electroacupuncture. *Brain Res Bull.* 2024;218:111092. <https://doi.org/10.1016/j.brainresbull.2024.111092>.
62. Yoo Y, Park H, Park S, Cho M, Cho S, Lee JY, et al. Interpersonal trauma moderates the relationship between personality factors and suicidality of individuals with posttraumatic stress disorder. *PLoS One.* 2018;13(1). <https://doi.org/10.1371/journal.pone.0191198>.

## Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.