scientific reports

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The unique contributions of adverse childhood experiences to increases in post-traumatic stress symptoms and problematic substance use after trauma exposure

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This study investigated the complex relationships between adverse childhood experiences (ACE), post-traumatic stress symptoms (PTSS), and substance use disorder (SUD) following a large-scale collective trauma in Israel. We utilized a longitudinal design with a quasi-representative sample of 1,343 Jewish Israeli adults, measuring PTSS and SUD both before (April 2022) and after (December 2023) the October 7th attack. Using regression analysis and structural equation modeling, we tested the sensitization and self-medication hypotheses, and the direct effect of ACE on SUD beyond PTSS. Results demonstrate that higher ACE scores significantly predicted greater elevations in PTSS following the trauma, supporting the sensitization hypothesis. Pre-trauma PTSS significantly predicted increases in SUD, consistent with the self-medication hypothesis. Critically, ACE directly predicted an elevation in SUD even when controlling for PTSS. SEM analysis provided evidence for a direct contribution of ACE to changes in SUD after the collective trauma. This provides new evidence suggesting that early adversity confers a distinct, independent risk for SUD that is not solely mediated by post-traumatic stress. These findings underscore the profound and multifaceted impact of childhood experiences on long-term well-being and highlight the need for trauma-informed interventions that address both immediate stress reactions and the enduring consequences of early adversity.

Adverse childhood experiences (ACE), defined as harmful events that occur during childhood, encompass various forms of abuse (emotional, physical, sexual), neglect (emotional, physical), and household dysfunction[1,2]. ACE represents a significant public health concern with far-reaching implications for long-term well-being[1]. Research consistently demonstrates that exposure to ACE is a robust predictor of various negative outcomes across the lifespan, including elevated risks for developing mental disorders[1,3,4], including depression[1,5], anxiety[3], and post-traumatic stress disorder (PTSD)[6–8]. Beyond directly precipitating post-traumatic stress symptoms (PTSS[6,7], ACE can amplify vulnerability to later trauma. The chronic or intense stress experienced during critical developmental stages can disrupt physiological and psychological development, potentially leading to insecure attachment[9], deficits in emotion regulation skills[10], and maladaptive coping strategies like experiential avoidance and maladaptive risk-taking behaviors[11]. This developmental disruption is central to the sensitization hypothesis, which posits that prior adversity or trauma induces a state of heightened vulnerability, rather than resilience, when an individual faces subsequent traumatic events[12,13], thereby increasing susceptibility to experiencing mental disorders[12,13].

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One significant maladaptive coping mechanism related to ACE, which has severe negative effects on individuals' mental health, is the use of substances for self-medication[14]. Substances may provide a temporary sense of relief or numbness that, although creating a dangerous cycle of overdependence and addiction, allow people to immediately buffer the pain and distress related to early or current adverse experiences[9,14–16]. Indeed, research shows that ACE are a well-established risk factors for substance use disorder (SUD) in adulthood[2,17,18]. Individuals with a history of childhood trauma often report an earlier onset of substance use[19], increased alcohol cravings[19], and higher rates of relapse[19].

Moreover, studies consistently show that trauma survivors exhibiting more severe trauma-related symptoms have higher rates of substance use[19], earlier onset[19], and more severe patterns of usage[19]. Individuals with PTSD often report using alcohol and cannabis to alleviate post-traumatic symptoms such as emotional distress, hyperarousal, and intrusive thoughts[7,14,15,20]. Similarly, opioids are used for symptom management and are particularly prevalent among individuals with PTSD to manage pain[21]. Even stimulant use, such as cocaine, can serve as a strategy to regulate distressing emotions by controlling impulsive behaviors[22]. While substances may offer transient relief, their use may ultimately exacerbate the trauma-related symptoms they are intended to manage, thereby perpetuating a self-exacerbating cycle[23].

Given these established associations, critical questions emerge: To what extent do ACE contribute to the development or increased severity of SUD directly, beyond their influence through PTSS? Does substance use merely an attempt to self-medicate PTSS, or does ACE also confer an independent vulnerability to SUD, regardless of the presence of PTSS? Furthermore, aligning with the sensitization hypothesis, ACE are expected to predict an elevation in PTSS following subsequent traumatic events. However, do ACE also predict increases in SUD symptoms following traumatic events later in life? ACE might independently contribute to an elevation in SUD beyond predisposing to PTSS. Individuals may be using substances to cope with ACE-related distress that does not necessarily manifest in PTSS symptoms (e.g., chronic pain or cognitive deficits) with regard the current trauma. Alternatively, ACE may create generalized predisposition that independently contribute to both PTSS and SUD.

Despite extensive research on bivariate associations between ACE, PTSS, and SUD, investigations into their full triadic interplay following current trauma exposure remain comparatively limited. Moreover, to firmly establish empirical evidence for these complex relationships, particularly regarding changes following a traumatic event, pre- and post-event measurements of both SUD and PTSS are critically needed. Such longitudinal studies are even more scarce due to the inherent unpredictability of traumatic events that precludes planned prospective studies.

Recent work provided some evidence of direct effects of ACE on SUD, beyond the effects of PTSS, but studies were limited by lack of examination of changes in SUD and PTSS following a traumatic event. Mergler et al.[19] showed that childhood trauma and PTSS were associated with more severe patterns of substance use. Interestingly, childhood trauma without concurrent PTSS was linked to certain substance use patterns, albeit to a lesser extent than when PTSS was present. However, this study was limited to individuals already diagnosed with SUD, potentially restricting generalizability. In a related study, Jung et al.[24], demonstrated that the link between ACE and binge drinking was only partially mediated by PTSD, with a remaining ACE direct effect. Similarly, studies have shown that current PTSS partially mediates the link between child and youth sexual abuse and different forms of substance use[25,26]. Ulman et al.[27] extensively examined these links in women with unwanted sexual experiences, revealing nuanced mediation roles for PTSS in the relationship between different trauma types and problematic substance use; however, the specificity of their sample limit broader generalizability. Thus, to the best of our knowledge, there is no robust study examining the effects of ACE on changes in SUD following a traumatic event later in life in the general population, particularly one that rigorously assesses whether this influence extends beyond mediation through PTSS.

The present study aims to address this critical research gap. The unfortunate but unique October 7th traumatic event in Israel provides an unparalleled opportunity to examine this aim within a large population exposed to severe and widespread stress. Leveraging a longitudinal design with pre- and post-event measurements of both PTSS and SUD in a large, quasi-representative national sample of Israeli adults, our study is uniquely positioned to draw insight into the complex interplay of ACE, PTSS, and SUD. While we present ACE as a predictor throughout this study given that the adverse experiences occurred in childhood and therefore preceded all other measured variables, it is important to note that ACE was assessed retrospectively after the October 7th traumatic events. As a result, ACE can also be conceptualized as a retrospectively-assessed moderator, particularly in light of the potential for state-dependent recall bias following trauma exposure. This alternative framing warrants further examination and more nuanced modeling beyond the scope of the current study to disentangle ACE's role as an interacting moderator.

The current study has three key aims. One, to test the sensitization hypothesis by investigating whether ACE predict an elevation in PTSS following the traumatic event. Two, to test the self-medication hypothesis by examining (a) whether pre-event PTSS predicts an elevation in SUD following the traumatic event and (b) whether ACE predict an elevation in SUD after such an event, building upon the known association between ACE and SUD. Three, to examine whether ACE predict an elevation in SUD after a traumatic event, beyond their contribution through PTSS. In this way, we will examine the full triadic interplay of ACE and increases in PTSS and SUD following exposure to a current trauma, and model the causal pathways through which ACE might explain elevation in post-trauma SUD directly or through the mediation of PTSS.

Methods

This article is based on data collected by the Israeli Center on Addiction and Mental Health (ICAMH) at multiple time points. In this study, we used data from two time points, before and after October 7: April 2022 (described in Shmulewitz, D. et al., 2023)[28], and December 2023 (described in Shmulewitz, D. et al., 2024)[29].

Sample

The data were from Jewish Israeli adults, all fluent in Hebrew. Participants were recruited from iPanel, a panel of individuals who are willing to participate in online surveys (iPanel, 2023)[30]. In the pre-trauma (April 2022) wave of measurement (N = 2,659), we assessed problematic substance use and post-traumatic stress symptoms. In the post-trauma (December 2023) wave (N = 4,002), we assessed the same aspects as well as adverse childhood experiences. The December 2023 sample included all participants from the April 2022 wave who agreed to be recontacted and completed the follow-up, forming the longitudinal subsample used in the current analyses (N = 1,343). In addition, the December 2023 wave included new respondents who were not part of the 2022 sample, recruited to support a broader epidemiological study following the traumatic events of October 7. The same recruitment methods, sampling quotas, and core instruments were used across both time points to ensure consistency. Further details on the sample, recruitment procedures, and representativeness can be found in a previous publication using this dataset[29,31] as well as in the official report of the Israel Center for Addiction and Mental Health (ICAMH, 2025)[32]. Quotas were used to match Israel's population of Hebrew-speaking Jewish adults by age, gender, region and religiosity, according to Israel Census Bureau (Gjersing 2012; Fricker 2016). Data from the subset of participants (N = 1,343) who participated at both time points were analyzed in the current study. Because some variables, like age or education could introduce selection bias into our sample, we conducted a missing data analysis using the finalfit[33] and MissMech[34] packages in R. This analysis aimed to account for any potential biases stemming from non-random dropouts of participants between the two waves[35].

Procedure

All methods were carried out in accordance with relevant guidelines and regulations, including the ICC/ESOMAR International Code, and all experiments were performed in accordance with the Declaration of Helsinki. All experimental protocols and survey procedures were approved by the Institutional Review Board of Reichman University. The April 2022 data collection was approved under protocol P_re1_2022050 (approved April 27, 2022). The December 2023 data collection and the longitudinal follow-up study were approved under protocol P_2023185 (approved November 23, 2023). Informed consent was obtained from all participants prior to their inclusion in the study. After consenting to participate in the survey, participants were transferred to the survey itself via Qualtrics[36], in order to maintain confidentiality. The surveys assessed socio-demographics, substance use and related behaviors, psychopathology, and risk and protective factors (such as exposure to October 7th or ACE in 2023 data). At the end of the survey, participants were given online gift cards worth 20 ILS. To maintain quality assurance, participants who failed any of the four attention checks were redirected out of the survey and incomplete surveys were removed. The survey employed a forced-response format in Qualtrics, requiring participants to respond to all items in order to proceed. As a result, the final analytic sample included only fully completed questionnaires, and no partial data were available for inclusion. In 2022, of the 5,133 participants who agreed to participate in the survey, 2,474 were removed due to quotas, not completing the survey or because of response patterns. In 2023, of the 6,765 participants, 2,763 were excluded for similar reasons.

Measures

Sociodemographics such as age, gender, education, and economic level were asked at the beginning of each survey (Supplementary Table 1).

Problematic substance use was assessed twice (before and after October 7 trauma) by the Alcohol, Smoking and Substance Involvement Screening Test (ASSIST; Humeniuk et al.[37]). Participants were asked about non-medical use of tobacco, alcohol, cannabis, sedatives, prescription stimulants and prescription opioid painkillers. For each substance they used non-medically, they were asked six follow-up questions regarding the frequency of use, craving, and negative consequences of use. Responses were weighted and summed into a final score variable[38]. To create one variable measuring problematic use of any substance, a variable was created that indicates the highest score from all substances (ASSIST maximum score).

Post-traumatic stress symptoms were assessed twice (before and after October 7 trauma) using the Posttraumatic Stress Disorder Checklist – DSM-5 (PCL-5[39,40]). The questionnaire included 20 items regarding various PTSD symptoms that the participant may be experiencing, such as intrusive memories and thoughts, emotional distress, physiological reactions, and insomnia. At both time points, participants were asked to report symptoms experienced during the past month. In the 2023 measurement, the instructions additionally referred to symptoms experienced "in the past month following the war," reflecting the national context at the time of data collection. While the items and response scale remained identical across waves and the measure was intended to assess the same construct at both time points, this added phrasing may have led some participants to focus their responses on war-related experiences. For each item, participants were asked to indicate how often they experienced it by choosing: (0) not at all; (1) a little bit (2) moderately; (3) quite a bit; or (4) extremely. Answers to all items were summed into a total score.

Adverse childhood experiences were assessed using eleven items adapted from the World Health Organization ACE questionnaire[41], administered in the post-trauma wave (December 2023[28]). Six items measured frequency of childhood maltreatment (e.g., physical abuse, sexual abuse, emotional abuse, and neglect) using a 1–4 scale. Due to a mistake with the item, the emotional neglect item was excluded, leaving ten eligible questions. Five additional items assessed household dysfunction (e.g., parental mental illness, substance use, incarceration, or violence), each coded dichotomously (0 = no, 1 = yes). Full item wording is provided in the Supplementary Materials. The answers to all items were summed into one final score; childhood maltreatment items contributed more to the total score because they were rated on a broader response scale (1–4), whereas household dysfunction items were coded dichotomously (0–1). Sensitivity analyses testing alternative scoring approaches are reported below.

*Exposure to the October 7th attacks*was assessed using four types of potentially traumatic events: (1) being physically present in an area of southern Israel that was attacked; (2) being exposed to the events while serving in the security forces or emergency response teams; (3) being in a location under widespread missile fire; and (4) experiencing serious injury or death as a result of the attacks. For each type of event, participants indicated whether it happened to: (1) themselves, (2) a close family member, or (3) someone they know. Multiple responses were allowed. Separate subscores were calculated for each exposure group (self, close family, acquaintance) by summing the number of endorsed event types (0–4). These three subscores were then summed to form a composite October 7th exposure index ranging from 0 to 12[31]..

Exposure to the ongoing war was assessed using two items measuring the frequency with which participants reported hearing explosions and hearing rocket warning sirens since October 7th. Each item was rated on a 7-point scale ranging from 1 (not at all) to 7 (a few times a day). The two items were summed to create a continuous index of ongoing war exposure, with higher scores indicating more frequent sensory exposure to war-related threats[31].

Analysis

Multiple regression analyses were conducted to address the three research aims.

To address the first question, whether ACE predicted a pre- to post-trauma elevation in PTSS, a regression model was run with PTSS in 2023 as the outcome and ACE as the predictor, controlling for prior PTSS in 2022, age, gender, exposure to the October 7th events and exposure to the ongoing war. To address the second question, whether pre-trauma PTSS or ACE predicted a pre- to post-trauma elevation in substance use disorder score, we conducted two separate regression models. A regression model was estimated with SUD (ASSIST max score) in 2023 as the outcome, PTSS in 2022 as the predictor, and prior SUD in 2022, age, gender, exposure to the October 7th events and exposure to the ongoing war as covariates. Another regression model was run with SUD in 2023 as the outcome and ACE as the predictor, while controlling for prior SUD in 2022, age, gender, exposure to the October 7th events and exposure to the ongoing war. To address the third question, whether ACE predicted an elevation in SUD beyond their effects on PTSS, a regression model was conducted with SUD in 2023 as the outcome and ACE and PTSS in 2023 as the predictors, while controlling for prior SUD in 2022, age, gender, exposure to the October 7th events and exposure to the ongoing war. Analysis was carried out using the R base package *stats* version 4.3.3[42].

To model the causal pathway through which ACE directly influence SUD, a comprehensive Structural Equation Model (SEM) was specified. The model aimed to examine the direct effect of ACE on the pre- to post-trauma changes in substance use disorder symptoms beyond the effect of concurrent pre- to post-trauma changes in PTSS. The model incorporated two latent change scores: one representing the change in SUD and another representing the change in PTSS. The simplified model is presented in Fig. 1, and all specifications are reported in Supplementary Tables 8 and 9. Analysis was carried out using the R package *lavaan* version 0.6.15[43].

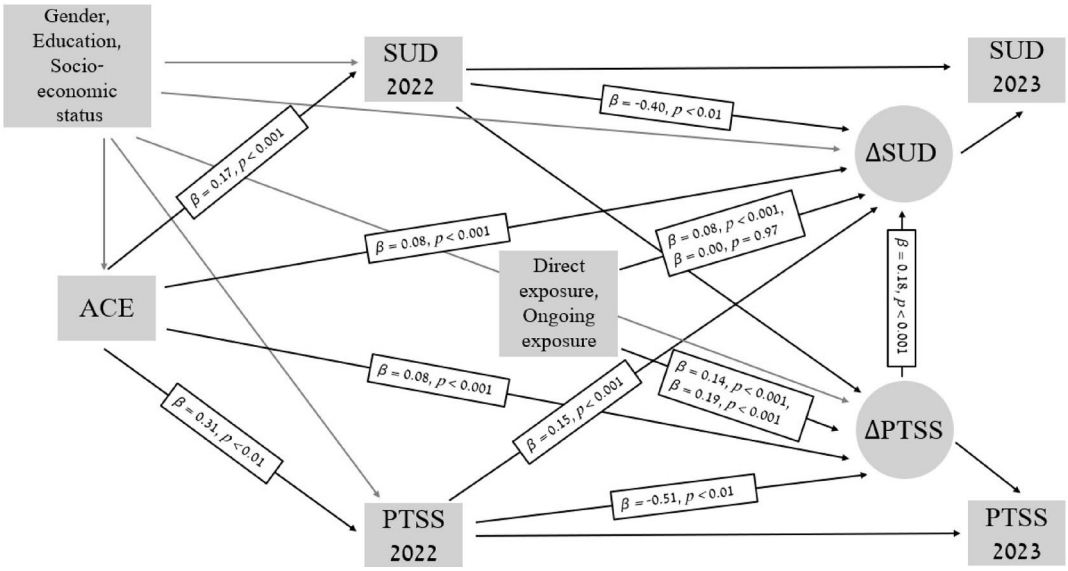

Fig. 1. Hypothesized Structural Equation Model of the Relationships Between Demographics, Adverse Childhood Experiences, Post-Traumatic Stress Symptoms, and Substance Use Disorder Symptoms. *Note*. Circles represent latent variables (change in SUD, change in PTSS). Rectangles represent observed variables (Adverse Childhood Experiences, Substance Use Disorder at baseline and follow-up, Post-Traumatic Stress Symptoms at baseline and follow-up, Direct Exposure to the October 7th events, Ongoing Exposure to the war, and demographic variables). Single-headed arrows indicate hypothesized regression paths. For clarity, double-headed arrows which indicate covariances, specific path labels and fixed parameters are omitted from this visual representation. Presented coefficients are standardized.

Results

Descriptives

About half the sample were women, secular, lived in the Tel Aviv/Central region. About a quarter were aged 18–34, and approximately half of the sample had post-high school, academic education. Potential PTSD (according to clinical cut-offs) was found in 11% of the sample in 2022 and 21.8% in 2023 (Supplementary Tables 1 and 2).

Multiple regression analyses

In addressing the first research question, the regression model examining pre-to post-trauma elevations in PTSS as a function of ACE was significant and explained 35.1% of the variance (Table 1; Fig. 2), with higher ACE scores predicting higher elevations in PTSS (p <.001), thereby supporting the sensitization hypothesis. In addressing the second research question, regressions analyses revealed that pre-trauma PTSS significantly contributed to pre-to post-trauma elevations in SUD and the model explained 47.6% of the variance (Table 2), with higher PTSS in 2022 predicting higher elevations in SUD (p <.01). Findings also indicated that ACE significantly contributed to pre-to post-trauma elevations in SUD (Model's R^2 = 48.0%; Table 3; Fig. 2), with higher ACE scores predicting higher elevations in SUD (p <.001). Both findings support the self-medication hypothesis. Regarding the third research question, ACE remained a significant predictor of higher pre- to post-trauma elevations in SUD even after controlling for the contribution of PTSS in 2023 (p <.01) with model explaining 49.3% of the variance (Table 4).

Latent variable modeling

Structural Equation Modeling (SEM) was conducted to examine the direct effect of ACE on change in SUD, while controlling for PTSS and multiple covariates, including trauma exposure (Fig. 1). The model demonstrated good fit with χ^2 (12) = 74.54, p <.001; CFI = 0.97; GFI = 0.99; RMSEA = 0.062. Baseline SUD significantly predicted pre- to post-trauma changes in SUD (B = − 0.34, SE = 0.02, p <.001). Baseline PTSS also significantly predicted pre- to post-trauma changes change in SUD (B = 0.07, SE = 0.01, p <.001). Likewise, the latent variable of pre- to post-trauma changes in PTSS significantly predicted concurrent changes in SUD (B = 0.07, SE = 0.01, p <.001). Importantly, ACE directly predicted pre- to post-trauma changes in SUD (B = 0.18, SE = 0.06, p <.01), beyond the significant contributions through baseline PTSS and pre- to post-trauma changes in these symptoms. Full model results are presented in Supplementary Tables 8 and 9.

Sensitivity analysis

Analysis of dropouts revealed that those who participated in 2022 but not in 2023 were significantly younger (p<.001) and had differences in education level (p<.001) and economic status (p=.003) (Supplementary Table 3). To account for these differences, we re-ran the regression models including these variables as covariates[33]. The core findings remained significant after accounting for these variables (Supplementary Tables 4–7).

The specification of ACE scoring can meaningfully influence the magnitude and interpretation of results[44] To evaluate the robustness of our findings to alternative ACE specifications, we constructed four theoretically motivated scoring variants (see Supplementary Materials). We then re-estimated all core analyses using each specification. In all cases, the effect of ACE remained statistically significant and directionally consistent, suggesting that the findings are not dependent on any particular operational specification.

Discussion

In this study, we examined the complex interplay between ACE, PTSS, and SUD following a collective traumatic event in the general population of Israel. We confirmed that ACE predicted an elevation in PTSS following the traumatic event, which supports the sensitization hypothesis. We also found that pre-event PTSS predicted an elevation in SUD following the event, consistent with the self-medication hypothesis. Furthermore, we report a novel finding by which ACE predicted an elevation in SUD after the traumatic event. Addressing the identified

	PTSS 2023				
Predictors	**Estimates**	**std. Error**	**CI**	**Statistic**	**p**
(Intercept)	10.82	1.87	(7.15, 14.49)	5.78	< **.001**
Adverse Childhood Experiences	0.55	0.13	(0.29, 0.81)	4.12	< **.001**
PTSS 2022	0.45	0.03	(0.39, 0.50)	16.38	< **.001**
Age	-0.15	0.03	(-0.21, -0.10)	-5.53	< **.001**
Gender [Male]	-5.61	0.75	(-7.07, -4.14)	-7.49	< **.001**
Ongoing Exposure	0.88	0.12	(0.65, 1.11)	7.49	< **.001**
Direct Exposure	1.19	0.21	(0.77, 1.61)	5.58	< **.001**
Observations		1336			
R^2 / R^2 adjusted		0.3517 / 0.3488			

Table 1. Multiple regression of ACE predicting change in PTSS. Note: ACE = Adverse childhood experiences (measured by summed ACE score); SUD = Substance use disorder (measured by maximum score on the ASSIST); estimates reflect prediction of ASSIST max score in 2023 controlling for ASSIST max score in 2022, age, and gender.

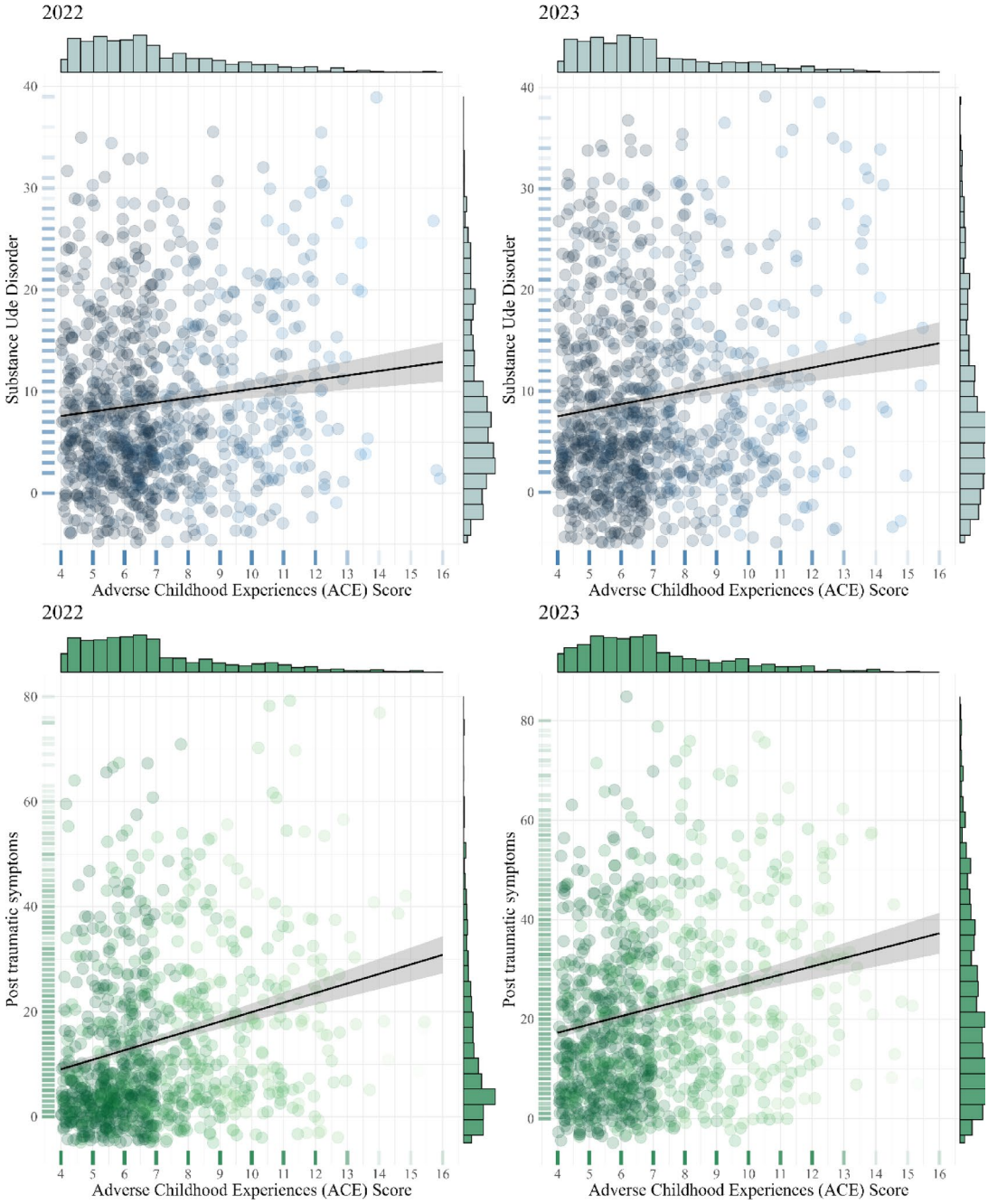

Fig. 2. Relations between ACE and SUD and PTSS before and after the 7th of October.

central research gap, we found that ACE predicted a pre- to post-trauma elevation in SUD beyond the contribution of PTSS. The causal pathway analysis provided evidence for a direct contribution of ACE to changes in SUD after the collective trauma. Importantly, the direct effect refers to the direct association estimated within the specified path model, which accounted for gender, education, economic status, PTSS and SUD levels before the trauma, as well as exposure to the October 7th events and the ongoing war. However, alternative explanations cannot be ruled out, including the possibility of unmeasured mediators, moderators, and measurement limitations. While our findings provide statistical support for a potential causal pathway linking ACE to increased SUD symptoms after trauma exposure, we emphasize that this is only preliminary evidence. Interpretations must be made in light of the study's limitations, as detailed in the Limitations section.

These findings are consistent with established literature. Our observation that pre-event PTSS predicts an elevation in SUD following the traumatic event highlights the enduring vulnerability that prior traumatic stress can encompass and suggests the important role of the self-medication hypothesis in post-trauma substance use[2,14,25]. The finding that ACE predicted an elevation in PTSS underscores the profound impact of early adversity on an individual's subsequent stress responses, and giving further support to the sensitization

Predictors	SUD 2023				
	Estimates	std. Error	CI	Statistic	*p*
(Intercept)	2.29	0.77	(0.77, 3.81)	2.95	**0.003**
PTSS 2022	0.03	0.01	(0.01, 0.06)	2.88	**0.004**
SUD 2022	0.69	0.02	(0.65, 0.74)	30.47	**< 0.001**
Age	−0.03	0.01	(−0.06, −0.01)	−2.59	**0.010**
Gender [Male]	0.55	0.34	(−0.12, 1.22)	1.60	0.111
Ongoing Exposure	0.06	0.05	(−0.05, 0.16)	1.06	0.289
Direct Exposure	0.38	0.10	(0.19, 0.56)	3.95	**< 0.001**
Observations		1336			
R^2/R^2 adjusted		0.4759/0.4735			

Table 2. Multiple regression of PTSS predicting change in SUD. Note: PTSS = Posttraumatic Stress Symptoms (measured by PCL-5 total score); SUD = Substance Use Disorder (measured by maximum score on the ASSIST); estimates reflect prediction of ASSIST max score in 2023 controlling for ASSIST max score in 2022, age, and gender.

Predictors	SUD 2023				
	Estimates	std. Error	CI	Statistic	*p*
(Intercept)	1.14	0.84	(−0.50, 2.79)	1.36	0.173
Adverse Childhood Experiences	0.25	0.06	(0.14, 0.36)	4.33	**< 0.001**
SUD 2022	0.69	0.02	(0.65, 0.73)	31.11	**< 0.001**
Age	−0.04	0.01	(−0.06, −0.01)	−3.08	**0.002**
Gender [Male]	0.61	0.34	(−0.06, 1.28)	1.79	0.073
Ongoing Exposure	0.05	0.05	(−0.05, 0.16)	1.03	0.305
Direct Exposure	0.37	0.09	(0.18, 0.55)	3.85	**< 0.001**
Observations		1336			
R^2/R^2 adjusted		0.4799/0.4776			

Table 3. Multiple regression of ACE predicting change in SUD.

Predictors	SUD 2023				
	Estimates	std. Error	CI	Statistic	*p*
(Intercept)	0.41	0.84	(−1.24, 2.05)	0.49	0.628
SUD 2022	0.66	0.02	(0.61, 0.70)	29.16	**< 0.001**
Adverse Childhood Experiences	0.18	0.06	(0.07, 0.30)	3.12	**0.002**
PTSS 2023	0.07	0.01	(0.04, 0.09)	5.87	**< 0.001**
Age	−0.02	0.01	(−0.05, 0.00)	−2.00	**0.046**
Gender [Male]	1.08	0.35	(0.40, 1.76)	3.11	**0.002**
Ongoing Exposure	0.00	0.05	(−0.11, 0.10)	−0.07	0.940
Direct Exposure	0.27	0.10	(0.09, 0.46)	2.86	**0.004**
Observations		1335			
R^2/R^2 adjusted		0.493/0.4903			

Table 4. Multiple regressions of ACE predicting change in SUD beyond PTSS.

hypothesis[12,13,45]. Notably, PTSS was not attributed to specific trauma types, and the study did not measure PTS symptoms stemming from ACEs specifically. Rather, we modeled the influence of ACE on SUD while accounting for overall post-traumatic stress responses following a traumatic event. Moreover, given the close temporal proximity between the October 7th events and the post-assessment, reported symptoms may reflect an acute stress response rather than longer-term psychopathology. Future studies should re-examine these associations after more time has passed, allowing for differentiation between acute and sustained responses, and should explicitly assess trauma attribution (e.g. PCL-5 item asking respondents to identify the 'worst' traumatic event) and its timing to further clarify the nuanced pathways linking ACE, PTSS, and SUD.

Building upon these foundational findings, our study offers novel insights into the nuanced relationship between these constructs. We found that ACE predicts elevation in post-trauma SUD partially through the ACE

effects on change in PTSS, similar to previous studies showing mediation of the ACE effect on SUD through PTSS[19,45,46]. Furthermore, we found that ACE predicts a direct elevation in SUD symptoms independent of PTSS, suggesting that the impact of early adversity extends beyond its contribution to post-traumatic stress. Critically, our research provides compelling evidence for a causal pathway through which ACE directly elevates SUD symptoms after exposure to adverse events later in life, suggesting that early life experiences can independently heighten vulnerability to substance use following subsequent trauma. Further studies should identify the underlying pathways through which this direct influence of ACE on SUD symptoms occurs.

These potential mechanisms can be considered within an integrated framework of "shared vulnerability," where early adversity disrupts fundamental psychological and biological systems, thereby increasing susceptibility to both PTSS and SUD. One central pathway begins with the disruption of emotion regulation i.e. the ability to influence the experience and expression of emotions. ACE fundamentally impair an individual's ability to tolerate distress and manage impulses[10]. These deficits are closely linked to the development of maladaptive coping mechanisms, such as avoidance and problematic risk-taking, which are well-established risk factors for SUD[10,14,15]. For instance, novelty-seeking and risk-taking patterns have been identified as psychological mechanisms that bridge ACE to earlier substance use initiation[2,7,45].

Similarly, ACE can contribute to SUD through cognitive impairments. Specifically, executive function deficits—impairments in cognitive control, behavioral inhibition, and decision-making—are frequently observed in individuals with a history of ACE[9,10], thereby increasing their susceptibility to SUD[16,18,47]. These cognitive challenges can hinder an individual's ability to resist impulsive behaviors, foresee negative consequences, and engage in adaptive problem-solving, all of which are essential for avoiding and discontinuing substance use. The dysfunction in emotion regulation and cognitive impairments can reinforce one another, leading to increased impulsivity and a learned tendency to avoid problems. This, in turn, can predispose individuals to use substances for self-medication.

This "shared vulnerability" can be further understood within the broader framework of attachment theory[9,16,47]. Evidence suggests that secure attachment is a protective factor against the impact of ACE on SUD, whereas insecure attachment heightens risk[18,47,48]. Individuals with insecure attachment may experience heightened anxiety, difficulty forming stable relationships, and an enduring need for comfort or safety[18,47,48]. Scholars have proposed that in moments of acute stress, substances can serve as a substitute "object" to provide this sought-after emotional relief[9,16]. This perspective helps explain why individuals might turn to substances not just to cope with trauma symptoms, but to fill a deeper relational void derived from early experiences of rejection, abuse or maltreatment[9].

Considering these potential pathways, including but not limited to maladaptive coping, emotion regulation deficits, cognitive impairments, and insecure attachment, underscores the complex and multifaceted ways in which ACE can heighten vulnerability to SUD. These mechanisms may account for the effects of ACE on SUD both directly and indirectly through PTSS. Our findings suggest that both the self-medication hypothesis, wherein individuals use substances to cope with PTSS, and the shared vulnerability hypothesis, where ACE create a generalized risk for both problematic conditions independently, potentially play complementary roles in the increase of SUD after a traumatic event. Future research should prioritize rigorous scientific exploration of these and other potential mechanisms, mapping the direct and indirect causal pathways linking ACE, PTSS, and SUD, including how PTSS directly related to ACE may differentially shape responses to later trauma.

Beyond advancing theoretical understanding, the current findings carry significant implications for policy makers and the development of targeted interventions aimed at reducing both SUD and PTSS. The independent impact of ACE on SUD emphasizes the critical need for comprehensive, trauma-informed approaches that extend beyond addressing acute PTSS to mitigate the pervasive effects of early adversity. Moreover, our results strongly advocate early interventions within at-risk households, aiming to prevent ACE and foster resilient developmental trajectories that safeguard against future mental health and substance use challenges.

Study limitations are noted. Our reliance on self-report measures introduces the possibility of recall and social desirability biases. We also acknowledge the potential for measurement inconsistencies in PTSS assessment across time points, including the absence of trauma attribution in the PCL-5. This design choice allowed us to capture general post-traumatic stress responses in a population exposed to multiple sources of trauma. However, it also limits our ability to determine whether symptoms reported at each wave were related specifically to ACEs, the October 7th events, or other intervening life events. Future waves of data collection will include trauma attribution items to better disentangle the source and timing of post-traumatic responses. Second, data were collected two months post-event and not immediately after. However, this timing allows us to infer that the observed elevations in PTSS and SUD were not merely acute reactions but represent more lasting changes. Third, due to the nature of the study, we could not separate the effects of the October 7th terror attack from the subsequent war. Moreover, as data were collected during an ongoing conflict, PTSS may partly reflect prolonged or evolving stress responses. Future studies should therefore re-evaluate these effects once the war concludes. Fourth, an important limitation concerns the timing of ACE assessment. Although all participants were adults reporting on childhood experiences, the ACE questionnaire was administered after the October 7th trauma. Retrospective reports may be influenced by current emotional states potentially creating a state-dependent recall bias which can affect the salience, accuracy, or intensity of reported memories. While such bias is not unique to this study and is inherent in all retrospective ACE assessments, it warrants caution when drawing strong causal conclusions. Future longitudinal studies examining ACE during childhood are needed to mitigate this. Fifth, the exclusion of non-Hebrew-speaking individuals and those under 18 or over 70 limits the generalizability of our findings to the broader Israeli population, particularly Palestinian Israelis and other Arabic-speaking communities. However, this limitation is partially mitigated by the fact that over 90% of Israeli Jews are fluent in Hebrew[49]; Importantly, we have initiated pilot research within these demographics to develop methods for collecting more inclusive samples of the broader population in future investigations. Finally, while

our sampling procedure may have introduced selection bias, we conducted missing data analysis to account for these differences, which confirmed that the significance of our core findings was not affected (Supplementary Tables 3–7).

Despite these methodological limitations, the current study advances our understanding of the complex interplay between ACE, PTSS, and SUD by leveraging a unique dataset from before and after a large-scale traumatic event in Israel. Our findings demonstrate that pre-event PTSS predicts an elevation in SUD, and that ACE predicts an elevation both in PTSS and SUD after a traumatic event. Critically, we provide novel evidence that ACE exerts a direct influence on SUD symptoms beyond the presence of PTSS, suggesting a distinct and independent pathway through which early adversity confers vulnerability to substance use, following later traumatic exposure, in the general population. This nuanced understanding underscores the profound and multifaceted impact of childhood experiences on long-term psychological well-being and substance use risk, highlighting the importance of comprehensive interventions that address both the immediate effects of post-traumatic stress and the deeper, long-lasting impact of early life adversity.

Data availability

The datasets generated and analyzed during the current study are not publicly available due to institutional restrictions and participant confidentiality agreements, as data access requires approval from the Israeli Center for Addiction and Mental Health, which serves as the data custodian, but are available from the corresponding author on reasonable request.

Received: 7 October 2025; Accepted: 27 January 2026
Published online: 01 February 2026

References

1. Hughes, K. et al. The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. *Lancet Public. Health*. **2**, e356–e366 (2017).
2. Zarse, E. M. et al. The adverse childhood experiences questionnaire: two decades of research on childhood trauma as a primary cause of adult mental illness, addiction, and medical diseases. *Cogent Med.* **6**, 1581447 (2019).
3. Sahle, B. W. et al. The association between adverse childhood experiences and common mental disorders and suicidality: an umbrella review of systematic reviews and meta-analyses. *European Child and Adolescent Psychiatry* vol. 31 1489–1499 Preprint at (2022). https://doi.org/10.1007/s00787-021-01745-2
4. Kalmakis, K. A. & Chandler, G. E. Health consequences of adverse childhood experiences: A systematic review. *Journal of the American Association of Nurse Practitioners* vol. 27 457–465 Preprint at (2015). https://doi.org/10.1002/2327-6924.12215
5. Merrick, M. T. et al. Unpacking the impact of adverse childhood experiences on adult mental health. *Child. Abuse Negl.* **69**, 10–19 (2017).
6. Frewen, P., Zhu, J. & Lanius, R. Lifetime traumatic stressors and adverse childhood experiences uniquely predict concurrent PTSD, complex PTSD, and dissociative subtype of PTSD symptoms whereas recent adult non-traumatic stressors do not: results from an online survey study. *Eur J. Psychotraumatol* **10**, 1606625 (2019).
7. Kalmakis, K. A., Chiodo, L. M., Kent, N. & Meyer, J. S. Adverse childhood experiences, post-traumatic stress disorder symptoms, and self-reported stress among traditional and nontraditional college students. *J. Am. Coll. Health*. **68**, 411–418 (2020).
8. Haim-Nachum, S., Sopp, M. R., Bonanno, G. A. & Levy-Gigi, E. The lasting effects of early adversity and updating ability on the tendency to develop PTSD symptoms following exposure to trauma in adulthood. *Cognit Ther. Res.* **46**, 1101–1112 (2022).
9. Mikulincer, M. & Shaver, P. R. *Attachment in Adulthood: Structure, Dynamics, and Change* (Guilford, 2010).
10. Cloitre, M. et al. Emotion regulation mediates the relationship between aces and physical and mental health. *Psychol. Trauma.* **11**, 82–89 (2019).
11. Mahanta, D., Deb Barman, J. A. & Review Paper on Adverse Childhood Experiences and Its Relation with Coping Strategies A. 12.
12. Hammen, C., Henry, R. & Daley, S. E. Depression and sensitization to stressors among young women as a function of childhood adversity. *J. Consult Clin. Psychol.* **68**, 782–787 (2000).
13. McLaughlin, K. A. et al. Childhood adversities and post-traumatic stress disorder: Evidence for stress sensitisation in the World Mental Health Surveys. *British Journal of Psychiatry* vol. 211 280–288 Preprint at (2017). https://doi.org/10.1192/bjp.bp.116.197640
14. Khantzian, E. J. The self-medication hypothesis of substance use disorders: A reconsideration and recent applications. *Harvard Review of Psychiatry* vol. 4 231–244 Preprint at (1997). https://doi.org/10.3109/10673229709030550
15. Luciano, M. et al. Supplemental material for posttraumatic stress Disorder, drinking to Cope, and harmful alcohol use: A multivariate Meta-Analysis of the Self-Medication hypothesis. *J. Psychopathol. Clin. Sci.* https://doi.org/10.1037/abn0000764.supp (2022).
16. McGovern, T. F. & Addiction Attachment, Trauma, and recovery: the power of connection. *Alcohol Treat. Q.* **38**, 537–539 (2020).
17. Bryant, D. J., Coman, E. N. & Damian, A. J. Association of adverse childhood experiences (ACEs) and substance use disorders (SUDs) in a multi-site safety net healthcare setting. *Addictive Behav. Reports* **12**, 100293 (2020).
18. Zhai, Z. W., Kirisci, L., Tarter, R. E. & Ridenour, T. A. Psychological dysregulation during adolescence mediates the association of parent-child attachment in childhood and substance use disorder in adulthood. *Am. J. Drug Alcohol Abuse*. **40**, 67–74 (2014).
19. Mergler, M. et al. Differential relationships of PTSD and childhood trauma with the course of substance use disorders. *J. Subst. Abuse Treat.* **93**, 57–63 (2018).
20. Hicks, T. A., Zaur, A. J., Keeley, J. W. & Amstadter, A. B. The association between recreational cannabis use and posttraumatic stress disorder: A systematic review and methodological critique of the literature. *Drug and Alcohol Dependence* vol. 240 Preprint at (2022). https://doi.org/10.1016/j.drugalcdep.2022.109623
21. Saraiya, T. C. et al. Trauma-related shame predicts daily non-medical prescription opioid use among individuals with PTSD symptoms. *J. Psychiatr Res.* **171**, 171–176 (2024).
22. Tull, M. T. et al. The role of emotion regulation difficulties in the relation between PTSD symptoms and the learned association between Trauma-Related and cocaine cues. *Subst. Use Misuse*. **51**, 1318–1329 (2016).
23. Chilcoat, H. D. & Breslau, N. Investigations of causal pathways between PTSD and drug use disorders. in *Addictive Behaviors* vol. 23 827–840 (1998).
24. Jung, J. et al. Adverse childhood experiences are associated with High-Intensity binge drinking behavior in adulthood and mediated by psychiatric disorders. *Alcohol Alcohol.* **55**, 204–214 (2021).
25. Keyes, K. M. et al. Exposure to the Lebanon war of 2006 and effects on alcohol use disorders: the moderating role of childhood maltreatment. *Drug Alcohol Depend.* **134**, 296–303 (2014).
26. Guillaume, S. et al. Associations between adverse childhood experiences and clinical characteristics of eating disorders. *Sci Rep* **6**, 35761 (2016).

27. Ullman, S. E., Relyea, M., Peter-Hagene, L. & Vasquez, A. L. Trauma histories, substance use coping, PTSD, and problem substance use among sexual assault victims. *Addict. Behav.* **38**, 2219–2223 (2013).
28. Shmulewitz, D., Eliashar, R., Levitin, M. D. & Lev-Ran, S. Test characteristics of shorter versions of the Alcohol, smoking and substance involvement screening test (ASSIST) for brief screening for problematic substance use in a population sample from Israel. *Subst Abuse Treat. Prev. Policy* **18**, 58 (2023).
29. Shmulewitz, D. et al. Comorbidity of problematic substance use and other addictive behaviors and anxiety, depression, and post-Traumatic stress disorder: A network analysis. *Psychol. Med.* **54**, 4635–4645 (2024).
30. iPanel.
31. Shmulewitz, D. et al. Exposure to online hate speech is positively associated with post-traumatic stress disorder symptom severity. *Sci Rep* **15**, 29869 (2025).
32. Shmulewitz, D. et al. Prevalence of Substance and Behavioral Addictions and Common Mental Health Disorders in Israel, 2022–2025. (2025).
33. Harrison, E., Drake, T., Pius, R. & Finalfit Quickly Create Elegant Regression Results Tables and Plots When Modelling. (2018). https://finalfit.org/
34. Jamshidian, M., Jalal, S. & Jansen, C. *MissMech: An R Package for Testing Homoscedasticity, Multivariate Normality, and Missing Completely at Random (MCAR). JSS Journal of Statistical Software* vol. 56 (2014). http://www.jstatsoft.org/
35. Woods, A. D. et al. Missing data and multiple imputation decision tree. Preprint at https://doi.org/10.31234/osf.io/mdw5r (2021).
36. Qualtrics, X. M.
37. Humeniuk, R. et al. Validation of the alcohol, smoking and substance involvement screening test (ASSIST). *Addiction* **103**, 1039–1047 (2008).
38. Humeniuk, R. *The Alcohol, Smoking and Substance Involvement Screening Test (ASSIST): Manual for Use in Primary Care* (World Health Organization, 2010).
39. Blevins, C. A., Weathers, F. W., Davis, M. T., Witte, T. K. & Domino, J. L. The posttraumatic stress disorder checklist for DSM-5 (PCL-5): development and initial psychometric evaluation. *J. Trauma. Stress.* **28**, 489–498 (2015).
40. Weathers, F. et al. The PTSD Checklist for DSM-5 (PCL-5). (2013).
41. World Health Organization. Adverse Childhood Experiences International Questionnaire. (2018).
42. R Core Team. R: A Language and environment for statistical computing. *R Foundation Stat. Computing*. https://www.R-project.org/ (2024).
43. Rosseel, Y. Lavaan: an R package for structural equation modeling. *J Stat. Softw* **48**, 1-36 (2012).
44. Simonsohn, U., Simmons, J. P. & Nelson, L. D. Specification curve analysis. *Nat. Hum. Behav.* **4**, 1208–1214 (2020).
45. Haller, M. & Chassin, L. Risk pathways among traumatic stress, posttraumatic stress disorder symptoms, and alcohol and drug problems: A test of four hypotheses. *Psychol. Addict. Behav.* **28**, 841–851 (2014).
46. Kaufman-Parks, A. M., Longmore, M. A., Manning, W. D. & Giordano, P. C. A prospective study of adverse childhood experiences and illicit drug use in adulthood. *Journal Hum. Services* **43**, 71-96 (2024).
47. Chambers, J. E. et al. Adverse childhood experiences, insecure attachment, and appointment compliance in an outpatient addiction psychiatry treatment population. *Am. J. Addictions.* **34**, 334–341 (2025).
48. Strathearn, L. et al. Pathways Relating the Neurobiology of Attachment to Drug Addiction. *Frontiers in Psychiatry* vol. 10 Preprint at (2019). https://doi.org/10.3389/fpsyt.2019.00737
49. Selected Data from the 2021 Social Survey on Languages. *Israel Central Bureau of Statistics* (2022). www.cbs.gov.il/he/mediarelease/DOcLib/2022/19_22_244b

Acknowledgements

The authors wish to thank Mattan S. Ben-Shachar for his invaluable assistance in statistical analysis.

Author contributions

M.D.L. conceived and designed the study, acquired, analyzed, and interpreted the data, and drafted and revised the manuscript; D.S. contributed to data acquisition, interpretation, and manuscript revision; E.L. contributed to data analysis, interpretation, and manuscript drafting and revision; V.S., S.L., and A.K. contributed to data acquisition. M.M. contributed to the conception and design of the study, data acquisition and interpretation, and manuscript drafting and revision. All authors approved the submitted version and agree to be personally accountable for their own contributions.

Declarations

Competing interests

The authors declare no competing interests.

Additional information

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-026-37883-3.

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