Journal of Dependence, 2026; 27(3):142-154

JOURNAL OF DEPENDENCE

Research Article

Childhood physical neglect and lower peripheral inflammatory indices in hospitalized male patients with substance use disorder: Associations with lifetime suicide attempts

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DOI: 10.51982/bagimli.621

Abstract

Objective: Childhood trauma is a major risk factor for suicidality and long-term biological stress alterations. Although suicidal behavior is commonly linked to systemic inflammation, it may also be associated with attenuated peripheral inflammatory responses. This study examined associations between childhood trauma subtypes, hemogram-derived inflammatory indices, and lifetime suicide attempts in hospitalized male patients with substance use disorder (SUD).

Methods: Childhood trauma, hemogram-derived inflammatory indices—including monocyte-to-lymphocyte ratio (MLR) and systemic inflammation response index (SIRI)—a composite Cumulative Inflammation Score (CIS), and lifetime suicide attempts were assessed in 186 hospitalized men with SUD. Hierarchical multiple linear regression analyses were performed after adjustment for relevant clinical covariates.

Results: Fifty-three patients (28.5%) had a history of suicide attempts and showed lower inflammatory indices (CIS: $p = .006$; MLR: $p = .001$). Physical neglect emerged as the trauma subtype most consistently associated with lower inflammation. In adjusted models, higher physical neglect scores remained independently associated with lower CIS ($\beta = -.258$, $p = .007$), MLR ($\beta = -.248$, $p = .013$), and SIRI ($\beta = -.252$, $p = .009$). These associations persisted after excluding hepatitis-positive participants.

Conclusion: Childhood physical neglect may be associated with relatively lower peripheral inflammatory indices in hospitalized men with SUD, highlighting heterogeneity in inflammatory profiles within this population. Because the underlying mechanisms were not directly examined and the regression models explained only modest variance, these findings should be considered hypothesis-generating. Prospective longitudinal studies are needed to determine whether hemogram-derived inflammatory indices have clinical utility for understanding suicidality in SUD.

Keywords: Childhood Trauma; Physical Neglect; Peripheral Inflammation; Substance-Related Disorders; Suicide Attempt

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Received: 2026-06-11
Accepted: 2026-08-19

e-ISSN: 3108-7973

INTRODUCTION

Systemic inflammation and immune dysregulation are increasingly implicated in the neurobiology of psychiatric disorders and suicidal behavior (Baldini et al., 2025; Zeng et al., 2024). However, the specific trajectory of these immune alterations remains complex, particularly in high-risk clinical populations exposed to chronic allostatic load, such as individuals with Substance Use Disorders (SUD). Current evidence suggests that psychoactive substance use is associated with significant alterations in both central and peripheral inflammatory signaling, involving microglial activation, cytokine release, and neuroinflammatory processes detectable via neuroimaging (Kohno et al., 2019; Namba et al., 2021).

A single biomarker may fail to adequately capture the complexity of systemic inflammation. Recent evidence suggests that hematology-derived composite inflammatory indices— including neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), and platelet-to-lymphocyte ratio (PLR)—may be associated with psychiatric and addiction-related processes (Baykara et al., 2022; Gürbüzer et al., 2024). These indices offer a more comprehensive inflammatory profile by simultaneously capturing multiple cellular components of immune activity. Notably, newer systemic indices such as systemic immune-inflammation index (SII) and systemic inflammation response index (SIRI) may demonstrate greater sensitivity than classical parameters in predicting suicide attempts (Ninla-Aesong et al., 2024). These hematology-derived inflammatory indices may be particularly informative in clinical populations where multiple biological and psychosocial vulnerabilities coexist, such as individuals with SUD and histories of childhood trauma.

Childhood trauma is highly prevalent among individuals with SUD, reflecting a complex bidirectional relationship between early adversity and substance use (Zhang et al., 2020). Childhood trauma may disrupt neurodevelopmental processes and contribute to persistent dysregulation in stress-response systems (Teicher & Samson, 2016) while also substantially increasing the risk of suicidal behavior (Rizk et al., 2021). In the broader literature, childhood adversity has most consistently been associated with immune activation and elevated proinflammatory mediators in adulthood (Baumeister et al., 2016).

Emerging evidence, however, suggests that the inflammatory consequences of childhood adversity may not follow a uniform proinflammatory trajectory, particularly when chronic deprivation rather than acute threat constitutes the predominant form of early stress (McLaughlin et al., 2014; Miller et al., 2007). Consistent with the threat–deprivation framework, deprivation-related adversities such as physical neglect may exert neurobiological effects that differ substantially from those associated with abuse-related trauma (Sheridan & McLaughlin, 2014). Childhood physical neglect has been associated with atypical inflammatory signaling patterns and altered adaptation to repeated psychosocial stress, although the direction of these responses may vary depending on the clinical context and measurement approach (Schreier et al., 2020). Relatedly, blunted hypothalamic-pituitary-adrenal (HPA) axis reactivity has been reported in suicidal populations with chronic stress histories (Melhem et al., 2016; Young et al., 2021), raising the possibility that prolonged adversity may, in some individuals, be associated with attenuated peripheral inflammatory responses over time.

This issue may be particularly relevant in SUD populations, where psychoactive substances themselves exert heterogeneous immunomodulatory effects. While stimulant and opioid exposure have been associated with elevated inflammatory indices in some studies (Lara-Apolinario et al., 2026), chronic opioid dependence has also been linked to reduced monocyte-related inflammatory activity (Quraishi et al., 2022), and cannabis use has been associated with heterogeneous and context-dependent immune effects (Moshfeghinia et al., 2024). These findings suggest that inflammatory patterns in trauma-exposed individuals with SUD may be more heterogeneous than the predominantly proinflammatory profiles commonly reported in general psychiatric populations.

Childhood trauma is among the strongest predictors of suicide attempts in individuals with SUD (Armoon & SoleimanvandiAzar, 2021; Baekkerud et al., 2023). Although suicidal behavior has repeatedly been linked to immune dysregulation (Baldini et al., 2025), the biological pathways through which early trauma—particularly deprivation-related experiences—contributes to suicide vulnerability in SUD populations remain poorly understood (Turecki et al., 2019). While elevated inflammatory indices have been reported in some suicidal populations (Peth□ et al., 2024), it remains unclear whether trauma-exposed individuals with prolonged substance use exhibit predominantly proinflammatory or relatively attenuated inflammatory profiles. Accordingly, the present study aimed to examine the relationship between childhood trauma, systemic inflammatory markers, and suicide attempts in individuals with SUD. We hypothesized that a history of suicide attempts would be associated with alterations in systemic inflammatory markers. Furthermore, we hypothesized that childhood trauma subtypes would show distinct patterns of association with these markers, with deprivation-related adversities demonstrating a different inflammatory profile from threat-related adversities. Finally, we hypothesized that childhood trauma dimensions would remain independently associated with systemic inflammatory indices after adjustment for relevant clinical covariates.

METHODS

Sample and Design

This cross-sectional study was conducted at the addiction center of Adana Dr. Ekrem Tok Mental Health and Diseases Hospital (January 2026–April 2026), including 186 male inpatients (aged 18–65) diagnosed with SUD (Diagnostic and Statistical Manual of Mental Disorders (DSM-5)). Individuals with psychotic, bipolar, organic brain disorders, or comorbid Alcohol Use Disorder (AUD) were excluded. Participants who reported alcohol consumption but did not meet DSM-5 diagnostic criteria for AUD remained eligible for inclusion. Medical histories and eligibility criteria were verified through retrospective medical record review and structured psychiatric interviews. To minimize biological confounding, individuals with malignancy, chronic systemic diseases, acute infection, Body Mass Index > 30 kg/m^2, recent use of inflammation-altering medications (e.g., antibiotics or corticosteroids within the previous three months), or clinically significant hematological abnormalities (e.g., anemia or leukocytosis) were excluded.

The study protocol was reviewed and formally approved by the Adana City Training and Research Hospital Scientific Research Ethics Committee (Date of Approval: December 18, 2025; Meeting No: 20; Decision No: 967). All procedures were conducted in strict accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments.

Sample size estimation was performed using G*Power 3.1 for between-group comparisons with unequal group allocation. Assuming a medium effect size (Cohen's $d = 0.50$), $\alpha = .05$, and 80% statistical power, the minimum required sample size was estimated at 155 participants. The final sample consisted of 186 individuals.

Measurement and Assessment Tools

Sociodemographic and Clinical Data Form

Basic demographic and clinical characteristics were recorded. For patients reporting multiple routes of substance administration, classification was based on the primary route of use. Concomitant alcohol use was defined as self-reported alcohol consumption that did not meet DSM-5 criteria for AUD. Participants with occasional or non-disordered alcohol use were therefore retained, whereas those meeting criteria for current AUD were excluded. To minimize acute inflammatory confounding, only serologically confirmed chronic Hepatitis B/C infection was considered as an infection-related clinical variable. A history of suicide attempt was defined as at least one lifetime suicide attempt requiring emergency medical and/or surgical intervention, as documented in hospital medical records and confirmed during the psychiatric interview. Suicidal ideation and preparatory behaviors in the absence of an actual suicide attempt were not classified as suicide attempts. For statistical analyses, suicide attempt history was coded as a dichotomous lifetime variable (present/absent).

Childhood Trauma Questionnaire (CTQ)

The CTQ, developed by Bernstein et al. (1994) and adapted into Turkish by Şar et al. (2012) (Cronbach α = .93), was used to assess individuals' traumatic experiences prior to the age of 18. High scores on the 28-item 5-point Likert-type scale, consisting of five subscales (emotional, physical, and sexual abuse; emotional and physical neglect), indicate increased trauma severity. In this study, Cronbach α was calculated as .83.

Psychometric assessments, including the Barratt Impulsiveness Scale Short Form (BIS-SF; α = .78) and the Buss-Perry Aggression Questionnaire (BPAQ; α = .90), were conducted on hospitalization day 21 to ensure cognitive stability and exclude acute withdrawal. These scores characterized the clinical profile and supported sample homogeneity.

Cumulative Inflammation Score (CIS) Calculation and Laboratory Analyses

Fasting venous blood samples were collected as part of the routine admission assessment between 08:00 and 10:00 a.m. on the morning of hospitalization, before initiation of pharmacological detoxification or other inpatient treatment procedures. Because this was a naturalistic clinical study, the exact interval since last substance use, withdrawal severity, and acute physiological status were not systematically recorded. Hemogram-derived indices included NLR (neutrophil/lymphocyte count ratio), MLR (monocyte/lymphocyte count ratio), SII (platelet × neutrophil/lymphocyte count), and SIRI (neutrophil × monocyte/lymphocyte count). To comprehensively summarize systemic inflammatory activity while reducing multiple testing, the four hemogram-derived indices (NLR, MLR, SII, and SIRI) were entered into an unrotated principal component analysis (PCA). Sampling adequacy was confirmed using the Kaiser–Meyer–Olkin statistic (KMO = .738) and Bartlett's test of sphericity (χ^2 = 626.741, $p < .001$). A single component with an eigenvalue > 1 was retained, explaining 78.7% of the total variance, with factor loadings ranging from .82 to .93. Individual factor scores were generated using the regression method in SPSS and saved as standardized continuous scores (M = 0, SD = 1), which were termed the CIS.

Statistical Analysis

Data analysis was performed using SPSS 27.0. The primary outcome was systemic inflammation, operationalized using the PCA-derived Cumulative Inflammation Score (CIS). Hierarchical multiple linear regression was the primary analytical approach for evaluating the independent associations between childhood trauma dimensions and systemic inflammation after adjustment for relevant clinical covariates. Analyses of individual inflammatory indices (MLR, SIRI, NLR, and SII), group comparisons, bivariate correlations, and substance-specific subgroup analyses were performed as secondary or sensitivity analyses.

The normality of continuous variables was examined using the Kolmogorov-Smirnov test, supplemented by inspection of skewness and kurtosis values. Depending on distribution characteristics, the Independent Samples t-test, Mann-Whitney U test, and Chi-square test were used for group comparisons. Bivariate relationships were evaluated using Spearman rank correlations with Benjamini-Hochberg False Discovery Rate (FDR) corrections for multiple comparisons. Hierarchical linear regression analyses were conducted to evaluate independent predictors of systemic inflammation, including the CIS and specific inflammatory indices (e.g., MLR, SIRI). Clinical covariates entered in the first step included age, duration of substance use, number of hospitalizations, chronic viral infection (Hepatitis B/C), concomitant alcohol use (present/absent), and suicide attempt history. Childhood trauma subdimensions were entered in the second step. To evaluate whether the observed associations were independent of substance-type heterogeneity, primary substance category (dummy coded) was entered as an additional third block in supplementary hierarchical regression analyses. Multicollinearity was assessed using variance inflation factors (VIF), with all values remaining well below 3. Statistical significance was evaluated at the p < .05 level.

RESULTS

Sociodemographic and Clinical Characteristics

Of the 186 SUD patients included in the study, 53 (28.5%) had a history of suicide attempts. The groups with and without suicide attempts showed similar distributions in terms of age, marital status, and education level (p > .05). However, unemployment was significantly higher among suicide attempters (χ^2 = 6.435, p = .011).

Suicide attempters had longer substance use durations (Z = -2.317, p = .021), more hospitalizations (Z = -3.014, p = .003), and a more prevalent family history of substance use (χ^2 = 7.553, p = .006). Among patients with a history of suicide attempts, chronic viral infection (Hepatitis B/C) positivity (χ^2 = 9.534, p = .002) and concomitant alcohol use (χ^2 = 5.244, p = .022) were found to be significantly higher than in the group without a history of suicide attempts **(Table 1)**.

Childhood Trauma Scores, Behavioral Characteristics, and Hematological Findings

In the comparison of traumatic experiences, CTQ total scores as well as the emotional abuse, emotional neglect, and physical neglect subscale scores were significantly higher among patients

Table 1: Comparison of Sociodemographic and Clinical Characteristics

Variables	Suicide attempt: No (n = 133)		Suicide attempt: Yes (n = 53)		Test Value $t/Z/\chi^2$	p
Age (Years) ***M±SD***	31.14 ± 8.18		31.83 ± 8.27		t = -0.521	.603
	n	%	n	%		
Marital Status						
Married	46	34.6	16	30.2	χ^2 = 1.239	.538
Single	69	51.9	32	60.4		
Divorced	18	13.5	5	9.4		
Education Level						
Elementary School	71	53.4	32	60.4	χ^2 = 1.263	.532
High School	52	39.1	19	35.8		
University	10	7.5	2	3.8		
Employment Status						
Yes	80	60.2	21	39.6	χ^2 = 6.435	.011*
No	53	39.8	32	60.4		
History of incarceration Yes	95	71.4	31	58.5	χ^2 = 2.903	.088
No	38	28.6	22	41.5		
Diagnosis						
Opioid	7	5.3	7	13.2	χ^2 = 7.941	.094
Cannabis	54	40.6	17	32.1		
Sedative	14	10.5	2	3.8		
Amphetamine	34	25.6	12	22.6		
Mixed	24	18.0	15	28.3		
Route of administration						
IV	14	10.5	8	15.1	χ^2 = 3.911	.141
Oral	74	55.6	21	39.6		
Inhaler	45	33.8	24	45.3		
Chronic viral infection (Hepatitis B/C)						
Yes	12	9.0	14	26.4	χ^2 = 9.534	.002**
No	121	91.0	39	73.6		
Substance use in the family						
Yes	112	84.2	35	66.0	χ^2 = 7.553	.006**
No	21	15.8	18	34.0		
Alcohol use						
Yes	96	72.2	29	54.7	χ^2 = 5.244	.022*
No	37	27.8	24	45.3		
Age of first substance use (Years)	18.00 (5.00)		17.00 (4.00)		Z = -1.529	.126
Duration of substance use (Years)	6.00 (9.00)		8.00 (10.00)		Z = -2.317	.021*
Number of hospitalizations	1.00 (1.00)		2.00 (2.00)		Z = -3.014	.003**

Note: Categorical variables are presented as frequencies (n) and percentages (%), and compared using the Chi-square test (χ^2). Continuous variables that met the assumption of normal distribution (Age) are presented as $M \pm SD$ and analyzed using the Independent Samples t-test (t); those that did not show normal distribution are presented as Median (Interquartile Range-IQR) and analyzed using the Mann-Whitney U test (Z). Statistical significance: * p < .05, ** p < .01.

with a history of suicide attempts than among those without such a history (all $p < .05$). Behavioral characteristics assessed by the BIS-SF and BPAQ did not differ significantly between groups (both $p > .05$). Among the raw hematological parameters, only monocyte counts were significantly lower in the suicide attempt group ($p = .038$), whereas white blood cell, neutrophil, lymphocyte, and platelet counts were comparable between groups (all $p > .05$). Regarding hemogram-derived inflammatory indices, NLR ($p = .031$), MLR ($p = .001$), SII ($p = .015$), SIRI ($p = .011$), and the CIS ($p = .006$) were all significantly lower among patients with a history of suicide attempts **(Table 2)**.

Table 2: Comparison of Childhood Trauma, Behavioral Characteristics, and Hemogram-Derived Inflammatory Indices

Variables	Suicide attempt: No (*n* = 133)	Suicide attempt:Yes (*n* = 53)	Test Value	Effect Size	
Childhood Traumas			Z	r	*p*
Emotional Abuse [a]	7.00 (5.00)	9.00 (8.50)	-2.725	.200	.006**
Physical Abuse [a]	5.00 (5.00)	6.00 (8.00)	-1.395	.102	.163
Sexual Abuse [a]	5.00 (2.00)	5.00 (3.50)	-0.939	.069	.348
Emotional Neglect [a]	12.00 (7.00)	16.00 (8.50)	-3.864	.283	< .001**
Physical Neglect [a]	10.00 (5.00)	12.00 (4.00)	-2.351	.172	.019*
CTQ-Total [a]	42.00 (18.50)	52.00 (22.00)	-3.720	.273	< .001**
Behavioral Characteristics			t	*d*	
BIS-SF [b]	29.93 ± 7.40	31.86 ± 5.80	-1.705	0.276	.090
BPAQ [b]	74.92 ± 18.54	80.45 ± 21.96	-1.739	0.283	.084
Raw Hematological Parameters			Z	*r*	
WBC($\times 10^3/\mu L$) [a]	9.30 (3.50)	8.70 (3.20)	−0.994	.073	.321
Neutrophil ($\times 10^3/\mu L$) [a]	6.10 (3.10)	5.40 (2.80)	−1.343	.098	.180
Lymphocyte ($\times 10^3/\mu L$) [a]	2.40 (1.10)	2.55 (0.90)	1.021	.075	.308
Monocyte ($\times 10^3/\mu L$) [a]	0.58 (0.30)	0.52 (0.22)	−2.074	.152	.038*
Platelet ($\times 10^3/\mu L$) [a]	278.00 (95.00)	275.00 (105.00)	−0.431	.032	.667
Inflammatory Markers			Z	*r*	
NLR [a]	2.45 (1.36)	2.13 (1.18)	-2.160	.158	.031*
MLR [a]	0.24 (0.10)	0.20 (0.09)	-3.183	.233	.001**
SII [a]	663.80 (474.56)	560.62 (384.62)	-2.429	.178	.015*
SIRI [a]	1.32 (1.09)	1.08 (0.81)	-2.534	.186	.011*
CIS [a]	-0.06 (1.18)	-0.35 (0.89)	-2.734	.200	.006**

Note: Superscript a indicates variables analyzed using the Mann–Whitney *U* test (reported as median [IQR], *Z* statistic, and effect size *r*). Superscript b indicates variables analyzed using the Independent Samples *t*-test (reported as *M* ± *SD*, *t* statistic, and Cohen's *d*). CTQ: Childhood Trauma Questionnaire; BIS: Barratt Impulsivity Scale; BPAQ: Buss-Perry Aggression Questionnaire; CIS: Cumulative Inflammation Score; NLR: Neutrophil/Lymphocyte Ratio; MLR: Monocyte/Lymphocyte Ratio; SII: Systemic Immune-Inflammatory Index; SIRI: Systemic Inflammation Response Index. Significance: * $p < .05$, ** $p < .01$.

Bivariate Correlations

Bivariate correlation analyses were conducted between childhood trauma dimensions and inflammatory indices **(Table 3)**. Based on FDR-adjusted significance levels, CTQ abuse-related subscales (emotional, physical, and sexual abuse) did not show statistically significant correlations with inflammatory markers (all FDR-adjusted $p > .05$). Initial weak correlations between emotional neglect and inflammatory indices did not remain significant after FDR adjustment. In contrast, higher physical neglect scores remained significantly associated with lower MLR ($r = -.231$, FDR-p = .018), SIRI ($r = -.213$, FDR-p = .029), and CIS ($r = -.239$, FDR-p = .018).

the inclusion of childhood trauma subdimensions significantly improved model fit ($\Delta R^2 = .059$, $p = .043$), and physical neglect showed independent association with lower CIS scores ($\beta = -.258$, $p = .007$).

After adjustment for the same clinical covariates, physical neglect remained negatively associated with MLR ($\beta = -.248$, $p = .013$), SIRI ($\beta = -.252$, $p = .009$), SII ($\beta = -.214$, $p = .028$), and NLR ($\beta = -.197$, $p = .041$). No independent associations were observed between abuse-related trauma dimensions and inflammatory indices (all $p > .05$).

To further evaluate whether the observed

Table 3: Bivariate Spearman Correlation Matrix of Childhood Trauma Dimensions and Inflammatory Indices

Variables	1	2	3	4	5	6	7	8	9	10
1. Emotional Abuse	-									
2. Physical Abuse	.704**	-								
3. Sexual Abuse	.491**	.534**	-							
4. Emotional Neglect	.283**	.269**	.198**	-						
5. Physical Neglect	.433**	.418**	.421**	.532**	-					
6. NLR	.013	.038	.020	-.159	-.143	-				
7. MLR	-.035	-.018	.033	-.188	-.231*	.627**	-			
8. SII	-.025	-.026	-.045	-.150	-.169	.849**	.541**	-		
9. SIRI	-.034	-.021	.027	-.176	-.213*	.796**	.827**	.736**	-	
10. CIS	-.066	-.034	-.018	-.178	-.239*	.845**	.849**	.880**	.913**	-

Note: *Data represent Spearman's rho (ρ) correlation coefficients. CTQ: Childhood Trauma Questionnaire; CIS: Cumulative Inflammation Score; NLR: Neutrophil-to-Lymphocyte Ratio; MLR: Monocyte-to-Lymphocyte Ratio; SII: Systemic Immune-Inflammatory Index; SIRI: Systemic Inflammation Response Index. Statistical significance for correlations between trauma dimensions and inflammatory indices (columns 6-10) was adjusted using the Benjamini-Hochberg False Discovery Rate (FDR) method. * p < .05 (FDR-adjusted), ** p < .01.*

Factors Associated with Systemic Inflammation

Hierarchical multiple linear regression analyses were conducted to identify independent predictors of systemic inflammation **(Table 4)**. Clinical and trauma-related variables were entered hierarchically, with demographic and clinical covariates entered in the first step and childhood trauma dimensions added in the second step. In step one, a history of suicide attempts was significantly associated with lower CIS scores ($\beta = -.187$, $p = .015$). In step two,

associations were independent of substance-type heterogeneity, additional hierarchical regression analyses were performed in which primary substance category (dummy coded) was entered as a third block. The associations remained statistically significant after additional adjustment for primary substance category, with childhood physical neglect continuing to independently predict lower MLR ($\beta = -.175$, $p = .019$) and SIRI ($\beta = -.166$, $p = .022$) (maximum VIF = 1.06).

Table 4: Hierarchical Multiple Linear Regression Analysis of Clinical and Traumatic Factors Predicting Cumulative Inflammation Score

Variables	B	SE	β	95% CI for *B*	t	p	VIF
Step 1: Clinical Covariates							
Age	0.005	0.003	.110	[−0.001, 0.010]	1.326	.187	1.350
Duration of substance use	−0.001	0.003	−.045	[−0.007, 0.006]	−0.528	.598	1.510
Number of hospitalizations	0.006	0.006	.105	[−0.005, 0.018]	1.359	.176	1.201
Chronic viral infection (Hep B/C)	−0.058	0.036	−.161	[−0.130, 0.013]	−1.925	.056	1.416
Alcohol Use	0.005	0.043	.024	[−0.080, 0.090]	0.325	.746	1.118
Suicide attempt history	−0.101	0.046	−.187*	[−0.193, −0.010]	−2.459	.015*	1.225
Step 2: Childhood Trauma Subdimensions							
Age	0.005	0.003	.112	[−0.001, 0.010]	1.361	.175	1.350
Duration of substance use	0.001	0.003	.012	[−0.006, 0.006]	0.137	.892	1.510
Number of hospitalizations	0.005	0.006	.085	[−0.006, 0.017]	1.098	.274	1.201
Chronic viral infection (Hep B/C)	−0.057	0.037	−.145	[−0.130, 0.015]	−1.724	.086	1.416
Alcohol Use	0.002	0.043	.004	[−0.083, 0.087]	0.049	.961	1.118
Suicide attempt history	−0.097	0.047	−.169*	[−0.190, −0.003]	−2.164	.032*	1.225
Emotional Abuse	0.007	0.007	.100	[−0.006, 0.020]	0.875	.383	2.629
Physical Abuse	−0.003	0.007	−.022	[−0.018, 0.012]	−0.201	.841	2.444
Sexual Abuse	0.015	0.008	.137	[−0.001, 0.030]	1.541	.125	1.575
Emotional Neglect	−0.001	0.005	−.027	[−0.009, 0.008]	−0.299	.766	1.644
Physical Neglect	−0.024	0.008	-.258**	[−0.040, −0.009]	−2.714	.007**	1.806

Note: B = unstandardized regression coefficient; SE = standard error; β = standardized regression coefficient; CI = confidence interval; *VIF* = variance inflation factor. Model Summary: Model 1 (Clinical Covariates): $R^2 = .071$, Adjusted $R^2 = .040$, $F(6,179) = 2.291$, $p = .037$. Model 2 (Trauma Subdimensions Added): $R^2 = .130$, $\Delta R^2 = .059$, $\Delta F(5,174) = 2.343$, $p = .043$. Statistical Significance: * $p < .05$, ** $p < .01$.

Sensitivity and Supplementary Analyses

Sensitivity analyses excluding 26 hepatitis-positive participants (n = 160) yielded comparable findings, with physical neglect remaining negatively correlated with CIS (r = −.227, p = .004) and individual inflammatory indices, including MLR (r = −.200, p = .011), SIRI (r = −.200, p = .011), and SII (r = −.165, p = .037).

Supplementary subgroup analyses by primary substance diagnosis revealed significant between-group differences in CIS levels (Kruskal–Wallis H = 11.734, p = .019). Post-hoc analyses indicated that the opioid group had significantly lower CIS levels compared with the cannabis (p = .003), amphetamine (p = .024), and mixed-use (p = .013) groups.

DISCUSSION

This study is one of a limited number of studies examining the possible role of systemic inflammation markers in the relationship between childhood trauma and suicide attempts in SUD patients receiving inpatient treatment. The findings suggest that childhood physical neglect was associated with relatively lower peripheral inflammatory indices among hospitalized male patients with

SUD, a pattern that may reflect heterogeneity in inflammatory responses within this specific clinical population.

Our findings can be interpreted within the framework of the time-dependent stress response model, which proposes that while acute stressors often trigger immune activation, early and chronic stressors such as childhood physical neglect have been proposed to contribute to long-term alterations in stress-response systems, potentially resulting in blunted biological responses in adulthood (Kuhlman et al., 2020; Miller et al., 2007). Consistent with this perspective, blunted hypothalamic-pituitary-adrenal (HPA) axis reactivity has been reported in suicide attempters with chronic stress histories (Melhem et al., 2016; Young et al., 2021), and prolonged adversity may be associated with a non-linear attenuation of peripheral inflammatory activity over time (Danese & McEwen, 2012). Previous theoretical models have suggested that chronic early deprivation may influence glucocorticoid signaling and stress-response regulation, leading to attenuated peripheral immune responsiveness in adulthood (Danese & Lewis, 2017). Because these mechanisms were not directly examined in the present study, this interpretation should be considered speculative. Inflammatory markers were measured at hospital admission rather than during an acute suicidal crisis; therefore, the observed profile is more likely to reflect systemic inflammatory status at admission than acute physiological changes associated with an active suicidal event.

In multiple regression analyses, the finding that physical neglect—rather than abuse-related trauma subtypes—was independently associated with lower inflammatory indices may be interpreted within the threat–deprivation model of childhood adversity (Sheridan & McLaughlin, 2014). According to the threat–deprivation framework, abuse-related adversities have more commonly been linked to acute immune activation, whereas deprivation-related experiences have been hypothesized to be associated with relatively attenuated inflammatory responses. Emerging clinical evidence is broadly consistent with this interpretation, as histories of childhood maltreatment and neglect have been associated with alterations in peripheral inflammatory biomarkers and psychiatric symptom profiles across both clinical and population-based samples (Cuñat et al., 2024; Jiang et al., 2025). In addition, inflammatory alterations related to neglect exposure may be relevant to suicidal behavior specifically (García-Fernández et al., 2025). Although emotional neglect initially demonstrated weak bivariate correlations with inflammatory indices, these associations lost significance after FDR correction. Physical neglect was the only trauma dimension that consistently remained associated with lower inflammatory indices after multivariable adjustment. Moreover, the overall explained variance of the regression models was modest, suggesting that additional biological, behavioral, and environmental factors likely contribute to variability in peripheral inflammatory indices.

Childhood physical neglect encompasses not only psychosocial deprivation but also inadequate fulfillment of basic physical needs, including nutrition. One possible explanation for the present findings is that prolonged deprivation during critical developmental periods may influence the maturation of stress-response and immune regulatory systems. Although this hypothesis is biologically plausible, the present study did not directly assess nutritional history or neuroimmune function. Future longitudinal studies incorporating nutritional, immunological, and neuroendocrine assessments are therefore needed to clarify these pathways.

While suicidal behavior has frequently been associated with elevated inflammatory markers in general psychiatric populations (Elbay et al., 2025; Moshfeghinia et al., 2025), recent evidence suggests that immune alterations in individuals with suicidal behavior are heterogeneous and vary by psychiatric diagnosis and symptom severity (Grendas et al., 2024). Similarly, findings across trauma-exposed and substance-using cohorts remain inconsistent, with both increased and attenuated inflammatory profiles being reported (Velasco et al., 2023). Populations characterized by chronic adversity, prolonged substance exposure, or severe allostatic burden may demonstrate relatively attenuated inflammatory responses over time rather than persistent immune activation—a pattern broadly consistent with the profile observed in the present sample.

The interpretation of these findings should also consider the heterogeneous immunomodulatory effects of chronic substance exposure itself. Opioid dependence has been associated with reduced monocyte activity and lower inflammatory indices, whereas stimulant-related substances may produce more variable inflammatory profiles (Lara-Apolinario et al., 2026; Quraishi et al., 2022). In addition, direct comparisons across SUD diagnoses indicate that NLR and MLR levels may differ substantially by primary substance, with alcohol use disorder associated with higher inflammatory indices than cocaine use disorder (Ruwel et al., 2025). Although patients with primary opioid use demonstrated lower CIS levels in subgroup analyses, the association between childhood physical neglect and inflammatory indices remained evident after adjustment for substance category, suggesting that the observed associations were not fully explained by differences in primary substance category.

The heterogeneity of inflammatory findings across studies may reflect important moderating factors, including the type, timing, and chronicity of trauma exposure, as well as characteristics of the study population. Studies conducted in general population samples or acute clinical settings have predominantly reported elevated inflammatory markers following childhood adversity, with abuse-related adversities showing more consistent associations with proinflammatory profiles than deprivation-related adversities such as neglect (Baumeister et al., 2016; Coelho et al., 2014; Danese et al., 2007). However, attenuated, modest, or inconsistent inflammatory associations have also been documented, particularly in studies examining specific trauma subtypes or populations characterized by chronic allostatic burden (Chiang et al., 2022; Cornman et al., 2023; Velasco et al., 2023). Taken together, these discrepancies underscore the importance of considering trauma subtype, population context, and biological measurement approach when interpreting inflammatory findings in high-risk clinical populations.

The present findings are based on hemogram-derived peripheral inflammatory indices rather than direct cytokine or neuroimmune measurements. Although such peripheral alterations may indirectly relate to neuroimmune processes implicated in stress responsivity and addiction (Hu et al., 2022; Namba et al., 2021; Wohleb et al., 2015), direct mechanistic inferences cannot be drawn from the present data. Therefore, the observed associations should not be interpreted as evidence of neuroinflammatory mechanisms or causal biological pathways.

Several limitations should be considered when interpreting these findings. First, the cross-sectional design precludes causal inference regarding the temporal relationships among childhood trauma, systemic inflammation, and suicide attempt history. Peripheral inflammatory markers were measured only once at hospital admission before treatment initiation, and acute factors such as recent substance use, withdrawal severity, sleep disturbance, dehydration, or nutritional status may have influenced these indices. Second, the heterogeneity of substance use diagnoses represents an important limitation, as different psychoactive substances exert distinct immunomodulatory effects. Although additional analyses adjusting for primary substance category yielded comparable findings, relatively small subgroup sizes limited substance-specific inference. Third, although major biological confounders (e.g., obesity, acute infection, chronic systemic disease, hematological disorders, and recent anti-inflammatory medication use) were excluded, detailed information regarding smoking intensity, psychiatric symptom severity, nutritional status, and medication exposure was unavailable. Although sensitivity analyses excluding hepatitis-positive participants supported the robustness of the findings, detailed virological and liver disease characteristics were unavailable. Fourth, childhood trauma subdimensions frequently co-occur, and the independent contribution of each trauma domain should therefore be interpreted cautiously. Fifth, suicide attempt history was assessed as a lifetime dichotomous variable without information regarding timing, frequency, method, or medical lethality. Although post-hoc power analyses indicated adequate statistical power, the unequal group sizes may have reduced sensitivity for detecting smaller effects. Finally, the single-center sample consisting exclusively of hospitalized male patients limits the generalizability of these findings to female patients, outpatient populations, and other clinical settings.

CONCLUSION

The present findings suggest that childhood physical neglect in hospitalized male patients with SUD is associated with relatively lower hemogram-derived inflammatory indices, highlighting the heterogeneity of peripheral inflammatory profiles in this high-risk clinical population. These findings should not be interpreted as evidence of reduced suicide risk or generalized immune suppression but rather as reflecting one possible pattern of peripheral inflammatory dysregulation. The underlying biological mechanisms remain to be clarified, and these observations should therefore be considered hypothesis-generating. Future prospective longitudinal studies incorporating comprehensive immune profiling are warranted to determine the clinical relevance of hemogram-derived inflammatory indices in suicidality among individuals with SUD.

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Peer-Review

Double blind peer reviewed

Conflict of Interest

The authors declare that they have no conflict of interests regarding content of this article.

Financial Support

The Authors report no financial support regarding content of this article.

Ethical Declaration

The study protocol was approved by the Adana City Training and Research Hospital Scientific Research Ethics Committee (Date: December 18, 2025; Meeting No: 20; Decision No: 967).

Consent to Participate: Written informed consent was obtained from all individual participants included in the study.

Declaration of Generative AI and AI-assisted Technologies in the Writing Process:

During the preparation of this work, the authors used DeepL in order to improve the language quality and readability of the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

Data Availability Statement:

The datasets generated and/or analysed during the current study are not publicly available due to the sensitive nature of the data and patient privacy restrictions but are available from the corresponding author on reasonable request.

Author Contributions

Conceptualization: MK, Methodology: MK, Formal Analysis: MK, SÖ, Resources: MK, AT,MG Writing – Original Draft: MK, Writing – Review, Editing: MK, MG, AT, Supervision: MK, Visualization: SÖ, Software: SÖ, Investigation: AT, MG, Data Curation: AT,MG, All authors read and approved the final manuscript.

Thanks

We would like to express our sincere gratitude to all the patients who voluntarily participated in this study

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