

THE PARADOX OF CHILDHOOD CUMULATIVE ADVERSITY IN ANTISOCIAL PERSONALITY DISORDER: RECONSIDERING TRAUMA-INFORMED APPROACHES IN CARCERAL SETTINGS

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Abstract

Introduction and Purpose: High levels of Adverse Childhood Experiences (ACEs), is often associated with Antisocial Personality Disorder (ASPD) psychopathology. Our study investigates if childhood adversities directly manifest through the dimensions of emotion regulation, psychopathy, impulsivity, and aggression. Dimensions that are commonly addressed in carceral settings.

Methodology: A retrospective study conducted in a carceral setting. We analysed records of male inmates with a clinical diagnosis of ASPD who consulted the prison psychiatric service from January to June 2025. Records with comorbid Borderline Personality Disorder (BPD) or active psychosis were excluded and only included medical records incorporating the relevant psychometric evaluations, the Difficulties in Emotion Regulation Scale (DERS), Psychopathy Checklist-Revised (PCL-R), Levenson Self-Report Psychopathy Scale (LSRP), Barratt Impulsiveness Scale (BIS-11), and Buss-Perry Aggression Questionnaire (BPAQ). Participants were stratified into High (ACE ≥ 4) and Low (ACE < 4) cumulative adversity groups.

Results: The final sample comprised (N=47) participants. No statistically significant differences were found between the High and Low cumulative adversity groups on the total scores of the DERS (p=0.11), PCL-R (p=0.63), LSRP (p=0.55), BIS-11 (p=0.19), and BPAQ (p=0.64). ACE correlation with those psychological dimensions did not reach significance.

Conclusion: Within a pure ASPD sample, a higher load of childhood adversity was not associated with greater deficits in emotion regulation, impulsivity, or overall aggression. This dissociation suggests that the phenotypic expression of ASPD in adulthood may represent a final common etiologic pathway, not necessarily associated with severity of childhood adversities. The findings invite reconsideration of current models and open insights to investigate the utility of integrating trauma-specific, explorative therapies to address deeply seated pathological schemas not addressed by classical Interventions with focus on the measured dimensions.

Keywords

Mental Health, Antisocial Personality Disorder, Childhood Psychotrauma, Adverse Childhood Experiences, Emotion Regulation, Forensic Psychiatry, Trauma-focused

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Publisher: Paradigm (De Gruyter)

DOI: <https://doi.org/10.56508/mhgci.v9i1.338>

Submitted for publication: 02 December 2025

Revised: 05 January 2026

Accepted for publication: 06 February 2026

Introduction

Antisocial Personality Disorder (ASPD) represents a severe and pervasive pattern of disregard for the rights of others, including traits of deceitfulness, impulsivity, irritability, recklessness, and an evident lack of empathy (WHO, 2024). It's a highly prevalent psychopathology in carceral settings, with a prevalence rate ranging 40-60% of male prison inmates, making it a critical mental health concern for forensic psychiatry according to a systemic review analyzing 62 surveys (Fazel and Danesh, 2002). The etiology of ASPD is complex and multifaceted, involving a complexed interplay of genetic predispositions, neurobiological vulnerabilities, and particular environmental triggers in Bio-Psycho-Social model (Jansen, 2022).

Among the documented environmental factors, comes the exposure to childhood Adversity as one of the major and replicated risk factors (Lobbestael et al, 2010). The Adverse Childhood Experiences (ACE) study and subsequent research have compellingly revealed a strong, dose-response relationship model between the number of ACEs, including abuse, neglect, and household dysfunction and subsequent negative outcomes later in adult life. From a forensic perspective, they are more prone to criminal behavior, violence, and the development of ASPD (Felitti et al, 2019; Hughes et al, 2017).

From a neurodevelopmental perspective, chronic early-life stress can induce structural and functional changes in key brain regions, including the prefrontal cortex, dorsolateral and ventromedial aspects, responsible for executive control and impulse regulation, the amygdala, involved in threat detection and fear, and the anterior cingulate cortex, implicated in error processing and empathy (Teicher and Samson, 2016; McCrory et al, 2011). This continuous exposure to stress during early developmental stages can interfere with the development of healthy emotional and behavioral regulation abilities, with tendencies towards externalization behaviors (Van der Kolk, 2014). Given this established link, an intuitive hypothesis would suggest that among individuals who have developed ASPD, those with a cumulative adversity would exhibit a more severe and impairing form of the disorder. This could manifest as more severe deficits in emotion regulation, a core difficulty implicated in reactive aggression and impulsivity (Gratz and Roemer, 2004); higher levels of psychopathy, particularly the secondary variant characterized by emotional dysregulation, impulsivity, reactive anger and expectedly

reflects on the scores of self-reported measures of impulsivity and aggression (Hicks et al, 2010).

We formulate our research hypothesis in easy wording, the null hypothesis H0: Once the diagnostic threshold for ASPD is reached, its phenotypic severity is independent of cumulative adversity; The alternative hypothesis H1: Higher ACE load is associated with proportional greater impairment in emotion regulation, impulsivity, aggression, and psychopathy traits.

However, the clinical reality within forensic populations suggests a more complex pattern of interaction. The psychological architecture of ASPD, which may overlap with but is distinct from psychopathy, can be characterized by significant emotional numbing, detachment, and alexithymia (Hare, 2003; Hemming et al, 2021). It's noteworthy that psychopathy construct is fundamentally based on the existence of two sub-factors affective-interpersonal factor and impulsivity-antisocial life styles. It's a broader dimensional rather than categorical diagnosis and it englobes antisocial personality disorder as well as other diagnosis as narcissistic and borderline personality disorders (Wall et al, 2015; Strickland et al, 2020).

It is a coherent hypothesis that for some individuals, a high adversity burden contributes to the development of ASPD through a process of adaptive emotional blunting, resulting in a personality structure where the subjective experience of emotional dysregulation is markedly attenuated, even if disruptive behavioral mode persists. This observation creates a paradox where the hypothesized etiological agent childhood adversity does not necessarily correlate with the severity of self-reported or clinically rated psychopathy symptoms in adulthood (Wolf, 2025).

Furthermore, the dominant approach of psychological intervention in many prison systems remains, globally, anchored to cognitive-behavioral therapy (CBT) models (Thekkumkara et al, 2022). These programs, such as reasoning and rehabilitation programs, primarily focus on modifying present cognitive distortions, enhancing problem-solving skills, and developing goal-oriented behavioral control. While they show modest efficacy in reducing recidivism (Valizadeh et al, 2020). They largely operate on a present-focused mode, deliberately avoiding deep exploration of past traumatic experiences. This therapeutic gap is particularly salient, in the light of the high prevalence of childhood traumatic adversities in offender population (Beaudry et al, 2021).

Purpose

The present study aims to dissect this complex relationship within a unique and rigorously defined clinical sample: incarcerated men with a primary diagnosis of ASPD, without comorbid disorders that could confound results as active psychosis or Borderline Personality Disorder (BPD).

Methodology

A retrospective cross-sectional study was conducted by reviewing the computerized medical records of the psychiatric service within the Centre Pénitentiaire de Ducos, the principal detention and correctional facility in Martinique, Overseas department of France. The study period spanned from January to June 2025. We screened all clinical records of male inmates who had received a formal diagnosis of ASPD from a staff psychiatrist, based on established ICD-11 diagnostic criteria (ICD11 Code F602) (WHO, 2024). To ensure a homogeneous sample and minimize confounding diagnostic bias in analysis, we applied strict exclusion criteria: any comorbid diagnosis of Borderline Personality Disorder (BPD) (ICD11 Code F603) due to its shared features of impulsivity and emotional dysregulation but different etiological pathways, or any active psychotic disorder (ICD11 Codes F20-29) as schizophrenia and schizoaffective disorder. The final sample comprised 47 participants, who were subsequently categorized into two groups based on their score on the Adverse Childhood Experiences (ACE) questionnaire.

Measures and Psychometric Properties

Data were extracted from the standardized psychological assessments proposed and administered during clinical consultations upon initial evaluation at the psychiatric service.

Cumulative Adversity

The Adverse Childhood Experiences (ACE) Questionnaire is a 10-item self-report tool that assesses exposure to categories of childhood maltreatment and household dysfunction before the age of 18 (Felitti et al, 2019). It covers emotional, physical, and sexual abuse; emotional and physical neglect; and household challenges such as substance abuse, mental illness, and incarceration of a relative. Each affirmative response scores one point, yielding a total score from 0 to 10. The ACE is a widely validated instrument with good test-retest reliability (Hughes et al, 2017). The cut-off score of 4, used to define our High Cumulative Adversity group (ACE \geq 4), is well-established in the literature as a threshold associated with a steep increase in

the risk for multiple poor health and social outcomes (Yu et al, 2022). In our Unit, the validated French version was used (Tarquinio Camille et al, 2023).

Emotion Regulation:

The Difficulties in Emotion Regulation Scale (DERS) is a 36-item self-report questionnaire that provides a comprehensive assessment of emotion dysregulation (Gratz and Roemer, 2004). Participants rate items on a 5-point Likert scale. It yields a total score and six subscale scores: Non-Acceptance of Emotional Responses, Difficulty Engaging in Goal-Directed Behavior, Impulse Control Difficulties, Lack of Emotional Awareness, Limited Access to Emotion Regulation Strategies, and Lack of Emotional Clarity. The DERS has excellent psychometric properties, with a reported Cronbach's alpha of 0.93 for the total score and subscale alphas ranging from 0.80 to 0.89 (Gratz and Roemer, 2004). In our Unit, the validated French version was used (Dan-Glauser and Scherer, 2012). In our study Cronbach's alpha was 0.83.

Clinician rated psychopathy:

The Psychopathy Checklist-Revised (PCL-R) is the gold standard 20-item clinical construct rating scale for the assessment of psychopathy in forensic populations (Hare, 2003). It is completed based on a semi-structured interview and a thorough review of supporting file information. It provides a total score (range 0-40) and two factor scores: Factor 1 (Interpersonal/Affective traits: glibness, grandiosity, deceitfulness, lack of empathy/remorse) and Factor 2 (Lifestyle/Antisocial traits: impulsivity, irresponsibility, need for stimulation, early behavioral problems). The PCL-R has extensive evidence for its reliability and predictive validity, with inter-rater reliability coefficients consistently reported above 0.85 (Hare, 2020). In the present study, the validated French version was used (Cote and Hodgins, 1991).

Self-rated psychopathy:

The Levenson Self-Report Psychopathy Scale (LSRP) is a 26-item self-report measure designed to assess primary and secondary psychopathy traits in community and incarcerated samples (Levenson et al, 1995). It uses a 4-point Likert scale (disagree strongly to agree strongly) and provides a total score, a Primary Psychopathy score (reflecting a selfish, uncaring, and manipulative interpersonal style), and a Secondary Psychopathy score (reflecting impulsivity, poor behavioral control, and self-defeating life strategies). The scale has demonstrated adequate internal consistency,

with Cronbach's alphas reported around 0.82 for the primary scale and 0.63 for the secondary scale (Levenson et al, 1995; Brinkley et al, 2001). In our study, the validated French version was applied (Savard et al, 2014).

Impulsivity:

The Barratt Impulsiveness Scale (BIS-11) is a 30-item self-report questionnaire that is the most widely used measure of trait impulsivity (Patton, 1995). Items are rated on a 4-point scale. It generates a total score and three second-order factor scores: Attentional Impulsiveness (inability to focus attention and cognitive instability), Motor Impulsiveness (tendency to act on the spur of the moment), and Non-Planning Impulsivity (lack of sense of the future and forethought). The BIS-11 has good internal consistency, with a total score Cronbach's alpha typically around 0.83 (Patton, 1995). The validated French version was applied (Gélinas et al, 2015).

Aggressiveness:

The Buss-Perry Aggression Questionnaire (BPAQ) is a 29-item self-report measure that assesses four dimensions of aggression (Buss and Perry, 1992). Items are rated on a 5-point scale. The subscales are: Physical Aggression (e.g., "I get into fights a lot"), Verbal Aggression (e.g., "I often find myself disagreeing with people"), Anger (e.g., "I have trouble controlling my temper"), and Hostility (e.g., "I am suspicious of overly friendly strangers"). The BPAQ has demonstrated good reliability and construct validity, with internal consistency coefficients for its subscales ranging from 0.72 to 0.85 (Buss and Perry, 1992). In our study, the validated French version was used (Bouchard, 2007).

It's worth mentioning that all our sample included in their records at least one event of a violent act or violation of internal carceral regulations leading to a disciplinary measure. This criterion represents an objective support for the dimension of aggressiveness assessed by the above mentioned psychometric tool.

Statistical Analysis

Data analysis was performed using SPSS Statistics version, 26.0. Descriptive statistics (means, standard deviations) were computed for all demographic and clinical variables. Independent samples t-tests were used to compare the High and Low cumulative adversity groups on all continuous outcome variables (DERS, PCL-R, LSRP, BIS-11, and BPAQ total and subscale scores). Pearson bivariate correlation was used to assess the correlation between the quantitative variables as score of all psychometric measures used in the study.

Convergent validity and scale coherence in our sample using the correlation of score of each subscale with the corrected total (sum of other subscales). Intercorrelation between subscales were done to assess discriminant validity. The assumption of homogeneity of variances were verified using Levene's Test. A p-value of < 0.05 was considered statistically significant for all analyses.

Ethical considerations

The study was approved by the Institutional Research Board IRB of Centre Hospitalier Universitaire CHU de Martinique under 2025/045. All patients were signing a non-opposition to use their anonymous data for clinical research at the French University hospitals when registering on the medical records system.

Results

Descriptive statistics of the sample

The final sample consisted of (N=47) male participants with a primary diagnosis of ASPD. The High Cumulative Adversity group (ACE ≥ 4) contained (n=25) individuals, and the Low Cumulative Adversity group (ACE < 4) contained (n=22) individuals. The sample (N=47) showed a mean age of 32.4 ± 8.59 (years). Scores on the diverse psychometric dimensions were as follows: ACE (3.94 ± 2.25), DERS (92.43 ± 26.41), BIS (66.66 ± 9.67), PCL (31.72 ± 3.62), LSRP (61.77 ± 11.26) and BPAQ (94.96 ± 15.92) (Table 1).

Inferential statistics and comparative analysis

Emotion Regulation (DERS), There was no significant difference in the overall emotion dysregulation between the two groups ($p=0.11$). Among the subscales, only Lack of Emotional Clarity approached but didn't reach significance ($p=0.06$). As for Psychopathy (PCL-R and LSRP), The groups did not differ on the total scores of the clinician-rated PCL-R ($p=0.63$) or the self-reported LSRP ($p=0.55$). A significant but mild difference was found only on the LSRP Primary Psychopathy subscale ($p=0.03$).

Lastly both Impulsivity (BIS-11) and Aggression (BPAQ), No significant differences were found for total impulsivity ($p=0.19$) or its subcomponents. Similarly, total aggression scores did not differ ($p=0.64$), this non-significance extended to its four sub-dimensions. (Table 2).

Correlation bivariate analysis

Bivariate Pearson correlation analysis revealed no statistical significant correlation between score of (ACE) and all psychometric measures of

psychopathy, emotional regulation, Impulsivity or Aggressiveness. (Table 3).

Due to the null results in our study, we assessed some psychometric properties of the psychometric measures.

Table 1: Descriptive statistics of the sample for the various psychometric measures (N=47)

Psychometric measure	Mean \pm SD	IC95%
AGE	32.4 \pm 8.59	[29.94 - 34.86]
ACE	3.94 \pm 2.25	[3.3 - 4.58]
DERS	92.43 \pm 26.41	[84.88 - 99.98]
BIS	66.66 \pm 9.67	[63.9 - 69.43]
PCL	31.72 \pm 3.62	[30.69 - 32.76]
LSRP	61.77 \pm 11.26	[58.55 - 65]
LSRP – Primary psychopathy	37.91 \pm 8.06	[35.61 - 40.21]
LSRP – Secondary psychopathy	23.85 \pm 5.11	[22.39 - 25.31]
BPAQ	94.96 \pm 15.92	[90.41 - 99.51]

ACE : Adverse Childhood Experiences ; DERS : Difficulty of Emotions Regulation Scale ; BIS : Barrat Impulsivity Scale ; PCL : Psychopathy Checklist of Hare ; LSRP : Levenson Self-Report Psychopathy Scale ; BPAQ : Buss Perry Aggression Questionnaire

Intercorrelation between subscales

The subscales intercorrelations supported the convergent and discriminant validity of the psychometric measures, with moderate to large positive correlations within each scale. The overall pattern of significant positive correlations within scales, coupled with generally lower or non-significant cross-scale correlations (where reported), aligns with the expected structural validity of each. It's noteworthy that non-significant subscales intercorrelation may be attributed to the small sample size and lower power to show significance (Table 4).

Correlation between subscales and corrected total

Corrected total was calculated as total score minus that subscale's score. For the DERS, the subscales Clarity, Impulse Control, Goals, and Strategies show large correlations (0.75 - 0.76) with the total corrected score, while Non-Acceptance shows a medium correlation and

Awareness is N.S., suggesting that Awareness contributes minimally to the overall DERS total. In the LSRP, both Primary and Secondary Psychopathy subscales show medium correlations with the total corrected score. For the BIS-11, all subscales (Cognitive and Motor Impulsivity, Difficult Planning) demonstrated medium to high positive correlations with the total corrected score, with Motor Impulsivity showing the strongest link. Similarly, for the BPAQ, Anger shows a large correlation with the total corrected score, Physical Aggression and Hostility show medium correlations, and Verbal Aggression was N.S.. Overall, the pattern supports the internal consistency and convergent validity of most subscales, while identifying specific subscales (Awareness in DERS, Verbal Aggression in BPAQ) that are poorly aligned with their respective total scores. This later finding may be explained by the small sample size and low power to detect significance. (Table 5).

Table 2. Comparison between groups with high cumulative adversity (≥ 4) (N=25) and those with low cumulative adversity (< 4) (N=22)

	Patients with score ≥ 4 High cumulative burden (n=25)	Patients with score < 4 low cumulative burden (n=22)	Student t test	P value
Difficulty in Emotions Regulation Scale DERS				
Score total	98.2 \pm 29.32	85.86 \pm 21.46	-1.63	0.11
Awareness	17.88 \pm 5.8	16.18 \pm 4.44	-1.12	0.27
Clarity	13.08 \pm 5.96	10.36 \pm 3.02	-1.93	0.06
Non Acceptance	15.4 \pm 6.37	13.64 \pm 5.01	-1.04	0.3
Impulse control	15.52 \pm 6.84	14.72 \pm 6.04	-0.42	0.67
Goals	15.84 \pm 5.44	13.5 \pm 6.81	-1.31	0.2
Strategies	21.16 \pm 8.02	18.04 \pm 6.22	-1.48	0.15
Psychopathy Check List of Hare PCL				
Score total	31.48 \pm 4.19	32 \pm 2.91	0.49	0.63
Levenson Self Report Psychopathy LSRP				
Score total	60.84 \pm 12.77	62.82 \pm 9.44	0.6	0.55
Primary psychopathy	36.72 \pm 8.89	39.27 \pm 6.96	1.08	0.3
Secondary psychopathy	24.12 \pm 5.59	23.55 \pm 4.63	-0.38	0.71
BARRAT Impulsivity Scale BIS-11				
Score total	68.4 \pm 10.54	64.68 \pm 8.39	-1.33	0.19
Cognitive impulsivity	18.8 \pm 3.99	17.32 \pm 2.83	-1.45	0.15
Motor Impulsivity	23.64 \pm 4.17	22.09 \pm 4.06	-1.29	0.2
Difficult planification	25.96 \pm 5.11	25.27 \pm 4.3	-0.5	0.62
Buss Perry Aggression questionnaire BPAQ				
Score total	93.92 \pm 16.5	96.14 \pm 15.54	0.47	0.64
Physical agressivity	30.36 \pm 6.34	33.6 \pm 6.79	1.69	0.1
Verbal agressivity	18.52 \pm 3.78	17.59 \pm 4.34	-0.79	0.44
Anger	21.08 \pm 6.93	21.09 \pm 4.83	0.01	0.1
Hostility	24.08 \pm 6.32	23.86 \pm 6.21	-0.12	0.9

*p<0.05 ; DERS : Difficulty of Emotions Regulation Scale ; PCL : Psychopathy check list ; LSRP levenson Self Report Psychopathy scale ; BIS : Barrat Impulsivity Scale ; BPAQ : Buss Perry Aggression Questionnaire

Table 3. Correlation between score on Adverse Childhood Experiences (ACE) and other psychological constructs (N=47)

	ACE	DERS	BIS	PCL	LSRP	BPAQ
ACE	1					
DERS	0.26	1				
BIS	0.32	0.37**	1			
PCL	-0.01	0.03	0.37*	1		
LSRP	0.02	0.54***	0.29	0.12	1	
BPAQ	0.01	0.67***	0.43*	0.14	0.63***	1

*p<0.05 ; **p<0.01 ; ***p<0.001 ; ACE : Adverse Childhood Experiences ; DERS : Difficulty of Emotions Regulation Scale ; BIS : Barrat Impulsivity Scale ; PCL : Psychopathy Checklist of Hare ; LSRP : Levenson Self-Report Psychopathy Scale ; BPAQ : Buss Perry Aggression Questionnaire

Table 4. Subscales Intercorrelations for the psychometric measures in the study

	Difficulty in Emotions Regulation Scale (DERS)					
	Awareness	Clarity	Non-Acceptance	Impulse control	Goals	Strategies
Awareness	1					
Clarity	0.33*	1				
Non-Acceptance	-0.05	0.46*	1			
Impulse Control	0.27	0.61*	0.26	1		
Goals	0.2	0.58*	0.51*	0.69*	1	
Strategies	0.19	0.67*	0.43*	0.77*	0.67*	1
	Levenson Self-Report Psychopathy Scale (LSRP)					
	Primary Psychop	Secondary Psychop				
Primary Psychop	1	0.43*				
Secondary Psychop	0.43*	1				
	Barrat Impulsivity Scale (BIS-11)					
	Cognitive impulsivity	Motor impulsivity	Difficult planning			
Cognitive impulsivity	1					
Motor impulsivity	0.35*					
Difficult planning	0.31*	0.53*	1			
	Buss Perry Aggression Questionnaire (BPAQ)					
	Physical	Verbal	Anger	Hostility		
Physical	1					

Verbal	0.02	1		
Anger	0.53*	0.23	1	
Hostility	0.26	0.08	0.39*	1

*p<0.05

Table 5. Correlation between subscales and corrected total score for the psychometric measures used in the study

Subscale	Corrected total	IC95%	Interpretation (per Cohen)
<u>Difficulty in Emotions Regulation Scale (DERS)</u>			
Awareness	0.17	[-0.12, 0.44]	N.S
Clarity	0.76***	[0.6, 0.86]	large
Non-Acceptance	0.41**	[0.14, 0.62]	medium
Impulse control	0.75***	[0.59, 0.85]	large
Goals	0.75***	[0.59, 0.85]	large
Strategies	0.76***	[0.6, 0.86]	large
<u>Levenson Self-Report Psychopathy Scale (LSRP)</u>			
Primary Psychopathy	0.43**	[0.16, 0.64]	medium
Secondary Psychopathy	0.43**	[0.16, 0.64]	medium
<u>Barrat Impulsivity Scale (BIS-11)</u>			
Cognitive Impulsivity	0.38**	[0.1, 0.6]	medium
Motor Impulsivity	0.56***	[0.33, 0.73]	medium
Difficult Planning	0.52***	[0.27, 0.7]	medium
<u>Buss Perry Aggression Questionnaire (BPAQ)</u>			
Physical Aggression	0.47***	[0.21, 0.67]	medium
Verbal Aggression	0.22	[-0.072, 0.48]	N.S.
Anger	0.59***	[0.36, 0.75]	large
Hostility	0.35**	[0.07, 0.58]	medium

*p<0.05; **<0.01; ***p<0.001; Corrected total = The sum of other subscales (calculated as Total score – Subscale score); The interpretation per Cohen for the subscales of “Motor Impulsivity” and “Difficult Planning”, are both large instead of medium, because both have value > 0.5.

Discussion

We hypothesized that if cumulative adversity exerts a direct, dose-dependent influence, on the maladaptive functioning within ASPD, the high load group will demonstrate significantly greater impairment across all measured psychological dimensions. Strikingly, the general lack of significant differences does not support a simple dose-dependent model within this severely affected sample. This could suggest either lack of quantitative association, where high adversity burden generates more severe unhealthy psychological measures, or this association may still exerting an important effect to etiologically induce the psychopathology in a rather present / absence model, where the development of the disorder itself is, hypothetically the potential critical threshold. In this later hypothesized model, additional trauma does not necessarily worsen its specific phenotypic expression. This finding invites consideration of profound implications on the way we conceptualize and treat ASPD in forensic settings. The dissociation between cumulative adversity and ASPD phenotype challenges a straightforward dose-dependent model of trauma in established ASPD.

The development of ASPD may represent a final common pathway or a diagnostic threshold that, once reached, exhibits a ceiling effect on certain psychological measures (Glenn et al, 2013). Our sample, by design, consisted of individuals who had already developed severe and prisoned antisocial behavior. Within this group, the additional dose of childhood adversity may not manifest proportionally with more severe deficits on the assessed psychological dimensions. The psychological machinery of ASPD, the impulsivity, the emotional deficits, the aggressiveness sound to operate at a consistently high pathological level, forming, a more or less, stable personality structure that is somehow dissociated from the quantitative load of its past adversities (DeLisi et al, 2019). This aligns with personality disorder theory, which posits that once maladaptive patterns are generated, they become self-perpetuating, driven more by internal personality dynamics than by their original triggers in a vicious circle model (Smits et al, 2024). Though this theory of final common pathway is a hypothetical possibility, but our cross-sectional design is yet limited with regards the ability to present this theory as established conclusion.

The significant finding of higher self-reported primary psychopathy in the low adversity group provides an interesting finding, subject to debate. This supports longstanding theories of a primary variant of psychopathy with a stronger biological

and temperamental basis, as low fearfulness and blunted affect, which may be less dependent on severe environmental trauma for its expression, so in simple terms, not a particularly trauma-sensitive subtype (Jansen, 2022; Viding and McCrory, 2019). In this model, the core poverty of empathy traits is a pre-existing vulnerability.

Conversely, the individuals in the high trauma group may have developed their antisocial pathology more directly through the internalizing pathways of emotional confusion and dysregulation as suggested by the tendency in the DERS Clarity subscale, even if the final behavioral outcome ASPD and incarceration is phenotypically similar they may still be etiologically heterogeneous (Kimonis et al, 2012).

The assessment tools themselves may be limited in capturing the specific sequelae of trauma in this population. The DERS, for instance, measures conscious awareness and strategies for managing emotion. In high-trauma ASPD, the defensive structure may involve profound emotional numbing, dissociation, or alexithymia that these scales do not fully catch (Hemming et al, 2021; Bach et al, 2022). What shows in results as a lack of clarity, the only significantly associated subscale, could be the reflecting a disconnection from internal states, a survival pathological coping strategy that becomes a personality trait (Wolf, 2025).

Modern neurobiological models offer a framework for understanding this dissociation. Early life adversity can lead to hyper-reactivity of the amygdala and hypoactivity of the prefrontal cortex, creating a brain predisposed to threat hypersensitivity and poor behavioral inhibition (Teicher and Samson, 2016; Herzog et al, 2020). However, in some individuals, particularly those who develop the primary psychopathic variant, the stress response system may be characterized by hypo-reactivity, leading to a lack of anxiety and fearlessness in the face of punishment (Blair, 2013). This biological dichotomy could underpin the different pathways to ASPD, explaining why trauma load does not have systematically the same effect. Furthermore, epigenetic mechanisms, where trauma modifies the level of gene expression without changing the DNA sequence, can create long lasting modulation in stress regulation and social behavior. It is possible that the mere presence of a certain level of trauma triggers these epigenetic changes, and the resulting phenotype then follows its own developmental course, again in a sort of automated pilot mode (Moreira et al, 2022).

Implications for Forensic Mental Health

The clinical implications of these findings are substantial. The current paradigm in many

carceral settings heavily relies on CBT-based programs that target cognitive distortions, problem-solving skills, and future-oriented behavioral control (Thekkumkara et al, 2022; Valizadeh et al, 2020). While these programs have value in managing surface-level behaviors, our results indicate that they are fundamentally incomplete because they systematically fail to address the foundational role of childhood trauma. If cumulative adversity does not directly correlate with current symptom severity, it is a fallacy to believe that treating the symptoms alone will resolve the underlying trauma. Conversely, ignoring the past adversities may mean missing the core etiological wound around which the personality disorder is structured (Levenson et al, 2020).

We therefore suggest a paradigm shift in therapeutic strategy. The high prevalence of CEs in this population is not a historical footnote; it is a central clinical reality that could benefit from direct and specific intervention (Gaber et al, 2025). Forensic mental health services could consider embracing trauma-informed care (TIC) principles, which recognize the pervasive impact of trauma and create environments of safety and empowerment. Beyond this philosophical shift, specific therapeutic modalities could be explored (Seitanidou et al, 2024).

Explorative Modalities like Schema Therapy, developed for personality disorders, are ideally suited to address the early maladaptive schemas, as mistrust/abuse, emotional deprivation and defectiveness, that originate in adverse childhoods (Pilkington et al, 2021). These therapies directly target the emotional and relational patterns formed in response to trauma, aiming to heal them at their root. Evidence-based treatments for trauma, such as Eye Movement Desensitization and Reprocessing (EMDR) (Shapiro, 2014) and Narrative Exposure Therapy (NET) (Elbert et al, 2022), adapted for complex trauma and forensic populations, warrant empirical investigation in this population. These therapies may help the individual process and integrate fragmented and distressing traumatic memories, reducing their power to drive maladaptive behaviors in the present. It's noteworthy that our therapeutic suggestions are based on the high prevalence of childhood adversity among individuals with ASPD, but were not assessed in the present study.

Approaches like Dialectical Behavior Therapy (DBT) (Rostamzadeh et al, 2024) and Acceptance and Commitment Therapy (ACT) (Byrne and Cullen, 2024), while focused on the present time, incorporate mindfulness and distress tolerance skills that are crucial for managing the emotional dysregulation that can emerge when trauma is

processed. They may serve as a vital preparatory and concurrent treatment to explorative work. Implementing such a model in a prison setting is challenging, requiring trained staff, institutional buy-in, and a long-term perspective. However, the potential payoff is a more profound and sustainable change, moving beyond mere recidivism reduction to genuine psychological rehabilitation (Coleman et al, 2024).

Limitations and Future Directions

This study has several limitations. Its retrospective and cross-sectional nature prevents any causal inference. The sample was all-male and from a single prison in a specific cultural context (French Caribbean), which may limit generalizability. While we excluded major comorbidities, we did not control for other factors like substance use disorders or depression, which could influence the results. The reliance on a specific cut-off score for the ACE, while standard, may have obscured more nuanced relationships. Additionally, the study's moderate sample size (N=47) may have limited statistical power to detect small to medium effect sizes. The reliance on retrospective data and standardized assessments, while necessary, may not capture the full complexity of trauma sequelae or the most severe variants of ASPD, as our sample included only inmates who consulted psychiatric services and had complete psychometric data. The high mean of psychopathy scale and the willing to pass an extensive battery of psychometric scales add a selection bias to the profile of patients included in the analysis. The retrospective research model limits control over assessment timing and rater consistency. Future research should employ longitudinal designs, larger and more diverse samples (including females), and more nuanced measures of trauma (e.g., type, timing, perceived impact). Incorporating neurobiological markers (e.g., cortisol levels, fMRI) could also help elucidate the different pathways linking trauma to ASPD. Most importantly, empirical trials are needed to directly test the efficacy of trauma-focused interventions (e.g., Schema Therapy, EMDR, NET) specifically in populations with ASPDs.

Conclusion

In conclusion, within a homogeneous group of incarcerated men with ASPD, a higher load of childhood trauma was not associated with more severe deficits in emotion regulation, impulsivity, or aggression. This dissociation highlights that the psychological presentation of ASPD is not a simple reflection of cumulative adversity but

rather a complex, potentially multi-determined endpoint. The single finding of elevated primary psychopathy in the low trauma group underscores the existence of distinct etiological pathways. The results serve as a powerful reminder that to effectively rehabilitate individuals with ASPD, correctional mental health services must look beyond present behaviors and directly address the ghosts of a traumatic past. This invites consideration of a fundamental shift from purely cognitive-behavioral management to an integrated, trauma-informed model that incorporates explorative, schema-focused, and trauma-processing therapies. Only by confronting the foundational wounds of the past can we hope to foster genuine and lasting change in this challenging population.

Funding statement

The authors declare that this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The publication fee was covered by the authors.

Conflict of interest

The authors have no relevant financial or nonfinancial conflict of interests to disclose.

Acknowledgment

To administrative, nursing and other paramedical staff of the Service Médico-Psychologique Régional SMPR, UF2101, Centre Pénitentiaire de Ducos.

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