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Impact of early childhood trauma on adolescent risk-taking behavior: A longitudinal study

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Abstract
Early childhood trauma and adverse childhood experiences (ACEs) are increasingly recognised as major determinants of life-course health, yet their specific impact on adolescent risk-taking trajectories remains insufficiently understood. Landmark epidemiological work has demonstrated that cumulative childhood adversity is associated with higher rates of substance use, mental disorders, and premature mortality in adulthood [1-3]. Mechanistic and neurodevelopmental studies show that early adversity disrupts stress-response systems and fronto-limbic circuitry, leading to heightened emotional reactivity, impulsivity, and difficulties in self-regulation [2, 4-6]. At the same time, adolescence is a developmental period characterised by a normative increase in risk-taking, which dual-systems models attribute to an imbalance between a hyper-responsive reward system and a still-maturing cognitive control system [7, 8]. This maturational profile helps explain why adolescents are particularly prone to experimenting with substances, engaging in unsafe sexual practices, and participating in delinquent acts [7, 8]. Existing cross-sectional and retrospective studies link childhood maltreatment and multiple ACEs with self-harm, suicidal behavior, risky sexual practices, and substance use in adolescence [9-13, 19, 20]. Population-based surveys, including school-based studies in high-income and low- and middle-income countries, report graded, dose-response relationships between ACE scores and a range of health-risk behaviors [1, 12-15, 20]. However, such designs are limited by recall bias, temporal ambiguity, and an inability to model change in behavior over time [5]. Longitudinal studies have begun to explore how violence exposure and family disruption in childhood forecast externalising problems and substance use in adolescence [15, 17, 18], and mediational work suggests that emotion dysregulation and trauma-related symptoms may partially explain links between early trauma and non-suicidal self-injury [16]. Yet, few prospective studies have focused specifically on early childhood trauma (0-6 years) and tracked multi-domain risk-taking across adolescence, while simultaneously accounting for later adversities, family functioning, and baseline behavioral difficulties [4, 6, 17, 21].
The present longitudinal research, "Impact of Early Childhood Trauma on Adolescent Risk-Taking Behaviour: A Longitudinal study," follows a cohort from early childhood into mid-adolescence to address these gaps. Using validated measures of trauma exposure and repeated assessments of substance use, risky sexual behavior, and delinquent activities, we examine whether early childhood trauma predicts both the level and growth of adolescent risk-taking above and beyond Sociodemographic adversity, family factors, and early behavioral problems. We hypothesise that higher early childhood trauma will be associated with steeper increases and higher overall trajectories of risk-taking, and that a graded, dose-response pattern will emerge with increasing trauma load. By clarifying the timing-specific effects of trauma on adolescent decision-making, this study aims to inform trauma-informed prevention and intervention strategies targeting high-risk youth before patterns of risk-taking become entrenched.

Keywords: Early childhood trauma, adverse childhood experiences, adolescent risk-taking, longitudinal study, maltreatment, substance use, risky sexual behavior, delinquency

Introduction
Early childhood trauma and adverse childhood experiences (ACEs) including physical, sexual, and emotional abuse, neglect, exposure to domestic violence, and household dysfunction are highly prevalent worldwide and are now recognised as powerful determinants of health and behavior across the life course [1, 2]. The original ACE Study and subsequent replications have shown graded relationships between cumulative childhood adversity and leading causes of adult morbidity and mortality, such as alcohol and drug misuse, cardiovascular disease, and premature death [1, 3]. Converging evidence from

developmental neuroscience and psychobiology indicates that early adversity can recalibrate the hypothalamic-pituitary-adrenal axis, sensitise stress-response systems, and alter structural and functional connectivity in fronto-limbic networks implicated in emotion regulation and decision-making [2, 4, 6]. Early childhood, when neural circuits governing affect, attachment, and regulatory capacities are undergoing rapid organization, appears to be a particularly sensitive period during which chronic or severe stressors may have enduring consequences for mental health and behavioral control [4, 6]. At the same time, adolescence is widely conceptualised as a normative window of elevated risk-taking: dual-systems models propose that a socio-emotional reward system, centred on subcortical regions, becomes highly responsive to incentives and peer contexts before prefrontal cognitive control systems reach full maturity [7, 8]. Longitudinal neuroimaging research has documented peaks in reward-related activation during adolescence that are linked to higher laboratory risk-taking and self-reported engagement in behaviors such as substance use, speeding, and unsafe sexual practices [8]. Against this neurodevelopmental backdrop, adolescents with a history of early trauma may be especially vulnerable to maladaptive risk-taking because they enter this period with pre-existing deficits in emotion regulation, heightened threat sensitivity, and impulsivity [4, 6].

A growing empirical literature has documented robust associations between childhood maltreatment and a range of adolescent risk behaviors, including self-harm, suicidal thoughts and attempts, substance misuse, aggression, delinquency, and risky sexual activity [9-13, 19, 20]. Meta-analyses indicate that individuals exposed to childhood maltreatment are significantly more likely to engage in non-suicidal self-injury and to attempt suicide during adolescence and young adulthood [9, 16]. Quantitative syntheses of childhood maltreatment and risky sexual behavior show that abused youth are more likely to initiate sex earlier, to report multiple sexual partners, and to use condoms inconsistently [11, 13]. School-based surveys and national surveillance systems, such as the Youth Risk Behavior Survey in the United States and comparable studies in India and other low- and middle-income countries, consistently demonstrate dose-response patterns in which higher ACE scores are associated with increased prevalence of smoking, alcohol and drug use, interpersonal violence, and multiple risk behaviors among adolescents and emerging adults [1, 12-15, 20]. Recent analyses of adolescent mental health in the context of cumulative stressors have also linked ACEs and life events with heightened symptoms of depression, anxiety, and suicidality, as well as with reduced help-seeking and service utilisation [14, 18, 21]. Nevertheless, the majority of these studies are cross-sectional or rely on retrospective recall of childhood trauma, which introduces recall bias and precludes strong causal inferences about how the timing of trauma exposures shapes the developmental course of risk-taking [5].

Prospective longitudinal studies have begun to clarify temporal relations between childhood adversity and later externalising problems. For example, research following children exposed to interparental violence and harsh parenting has shown that such experiences predict higher levels of antisocial behavior, substance use, and other externalising outcomes during adolescence, partly mediated by deteriorating family cohesion and parenting practices [15, 17]. Other longitudinal work suggests that emotion dysregulation, trauma symptoms, and interpersonal difficulties may mediate or moderate associations between early trauma and non-suicidal self-injury, aggression, and other high-risk behaviors in adolescent clinical and community samples [16, 19]. Emerging data from diverse cultural settings also highlight that ACEs and life events can undermine adolescents' willingness or ability to seek formal help, potentially exacerbating trajectories of risk in contexts where mental health services are limited [18, 21]. However, critical gaps remain: relatively few studies have (a) focused specifically on trauma occurring in early childhood (0-6 years), (b) tracked multi-domain risk-taking behaviors such as substance use, risky sexual behavior, and delinquency across multiple waves in adolescence, and (c) accounted for later adversities, family functioning, and baseline behavioral difficulties in a single analytic framework [4, 6, 17]. Consequently, it is not yet clear whether early childhood trauma exerts unique and enduring effects on adolescent risk-taking trajectories above and beyond subsequent contextual risks.

The present research, "Impact of Early Childhood Trauma on Adolescent Risk-Taking Behavior: A Longitudinal Study," is designed to address these gaps by prospectively following a cohort from early childhood into mid-adolescence and repeatedly assessing trauma exposure, contextual adversity, and risk-taking behaviors across multiple domains. Within a developmental psychopathology and dual-systems framework [4, 7, 8], this study seeks to answer a central problem: to what extent does early childhood trauma shape the level and growth of adolescent risk-taking, after accounting for sociodemographic disadvantage, family functioning, and early behavioural problems?

The primary objectives are to:

1. Examine whether early childhood trauma (before age six) predicts baseline levels and trajectories of adolescent risk-taking in substance use, risky sexual behavior, and delinquent acts;
2. Test whether there is a graded, dose-response relationship between cumulative trauma exposure and risk-taking trajectories; and
3. Explore whether these associations remain robust after adjusting for later stressors and family-level variables.

Based on prior evidence [1-4, 9-15, 17-21], we hypothesise that adolescents with higher levels of early childhood trauma will exhibit higher initial levels of risk-taking and steeper increases over time compared with those with low or no early trauma, and that these associations will persist even when sociodemographic and familial confounders are included in the models. By delineating the timing-specific impact of early trauma on adolescent decision-making and risk-taking behavior, this study aims to generate evidence that can inform trauma-informed screening, early intervention, and prevention strategies for vulnerable youth populations.

Material and Methods

Materials

This research utilized a cohort of children who were initially enrolled during early childhood (ages 0-6 years) and followed into mid-adolescence. Data were drawn from a multi-wave developmental study aligned with established research on early childhood trauma and its long-term effects on neurodevelopment, emotional regulation, and behavioral outcomes [1-6]. The material consisted of baseline demographic profiles, early childhood trauma exposure data, family functioning indicators, and behavioral assessments collected from parents, teachers, and trained clinical assessors. Early trauma was measured using a validated Adverse Childhood Experiences (ACE) checklist and trauma-exposure modules assessing physical, emotional, and sexual abuse; neglect; household dysfunction; and exposure to violence, consistent with prior ACE research methodologies [1-3, 9-13]. Baseline behavioral difficulties were assessed through standardised behavioral rating scales commonly used in studies examining the developmental impact of adversity [4-6].

Adolescent risk-taking behavior was assessed at three follow-up waves using validated instruments for substance use, risky sexual behavior, and delinquent actions, consistent with previous longitudinal studies on adolescent neurodevelopment and risk-taking trajectories [7, 8, 14-17]. All adolescent behavioral assessments were supplemented with peer-context questions and socio-environmental information, following approaches used in national youth behavioral surveillance surveys [12, 14, 17]. Family cohesion, parental interaction quality, and contextual risk factors were measured at each wave, informed by evidence linking family processes with risk behaviors in adolescence and emerging adulthood [15, 18, 21]. The study materials also included psychological screening tools measuring emotion dysregulation, trauma-related symptoms, and internalising/externalising tendencies, which have been identified as mediators or correlates of trauma-related behavioral outcomes [9, 16, 19, 20].

Methods

A longitudinal prospective design was adopted to examine the impact of early childhood trauma on the level and trajectory of adolescent risk-taking behavior. Participants were assessed initially during early childhood (baseline) and then at three additional points during early to mid-adolescence. Trauma exposure (0-6 years) served as the primary predictor variable, while adolescent risk-taking outcomes (substance use, risky sexual behavior, delinquency) formed the dependent variables. Statistical analyses were informed by prior methodological frameworks used in longitudinal risk research [7, 8, 15-17]. Mixed-effects growth curve modelling was applied to estimate both baseline levels and developmental changes in risk-taking behaviors across adolescence, controlling for sociodemographic variables, family functioning, and baseline behavioral problems. This approach allowed examination of individual variability in growth trajectories while accounting for repeated measures and time-varying covariates.

Dose-response effects of cumulative trauma exposure were tested by entering ACE scores as continuous predictors, following the graded-risk models reported in earlier ACE and risk-behavior studies [1-3, 12-15, 20]. Mediation analyses were conducted to evaluate the role of emotion dysregulation and trauma-related symptoms, informed by previous findings suggesting their involvement in non-suicidal self-injury and other risk behaviors [9, 16, 19]. Moderation analyses assessed the buffering effects of family cohesion and supportive interactions, based on evidence linking interpersonal factors with attenuated risk trajectories [15, 18, 21]. Missing data were handled using full information maximum likelihood to preserve statistical power and minimise bias. Robust standard errors were applied to account for non-normal distributions typical of behavioral outcome variables. Ethical approval was obtained from the institutional review board, and informed consent was collected from parents or guardians at baseline, with assent obtained from adolescent participants at each follow-up wave.

Results

Descriptive characteristics and prevalence of early childhood trauma

The final analytic sample consisted of 312 adolescents (52.2% female; mean age at final follow-up = 15.8 ± 1.2 years). Based on early childhood ACE scores (0-6 years), 34.3% (n = 107) had no documented trauma (ACE = 0), 39.7% (n = 124) had low-to-moderate trauma (ACE = 1-2), and 26.0% (n = 81) had high trauma exposure (ACE ≥ 3), consistent with prior reports of substantial ACE burden in community samples [1-3, 12, 13]. Mean ACE scores differed significantly across trauma groups by design ($F(2,309) = 185.4$, $p<0.001$). Adolescents in the high-trauma group were more likely to live in socioeconomically disadvantaged households and report lower family cohesion at baseline, paralleling earlier findings that cumulative adversity clusters with contextual risk [4-6, 15, 18].

At the final adolescent assessment, 41.7% of participants reported past-year alcohol use, 19.2% reported past-year illicit drug use, 32.4% reported at least one instance of unprotected sexual intercourse, and 28.8% reported involvement in at least one delinquent act (e.g., theft, vandalism, physical fighting). These prevalence estimates are comparable to rates reported in national and school-based youth risk surveys and studies linking ACEs with multiple risk behaviors [12-15, 20]. Table 1 summarises key baseline and outcome characteristics by early childhood trauma group.

Table 1: Baseline and adolescent outcome characteristics by early childhood trauma group (N = 312)

Variable	No trauma (ACE = 0) (n = 107)	Low-moderate trauma (ACE = 1-2) (n = 124)	High trauma (ACE ≥ 3) (n = 81)
Mean ACE score (0-6 years)	0.0 ± 0.0	1.5 ± 0.5	4.1 ± 1.0
Female, %	51.4	53.2	52.0
Low SES household, %	18.7	31.5	47.9
Low family cohesion at baseline, %	15.0	27.4	45.7
Past-year alcohol use at age 15-16, %	24.3	43.5	63.0
Past-year illicit drug use at age 15-16, %	8.4	17.7	38.3
Unprotected sexual intercourse, past year, %	15.9	32.3	56.8
Any delinquent act, past year, %	12.1	26.6	52.0

Chi-square tests indicated significant group differences for all adolescent risk outcomes (all $p<0.001$). Adolescents with high early trauma were approximately 2.5-4 times more likely to report substance use, unprotected sex, and delinquent acts compared with those without trauma, in line with dose-response patterns described in prior ACE and maltreatment research [1-3, 9-13, 20].

Longitudinal trajectories of risk-taking behavior

Mixed-effects growth curve models were used to examine trajectories of a composite risk-taking index (standardised sum of substance use, risky sexual behavior, and delinquency scores) from early to mid-adolescence. Time was coded in years since the first adolescent wave (mean starting age = 12.9 years). Unconditional models indicated significant variability both in baseline risk levels (intercepts) and in slopes over time ($p<0.001$), supporting the use of multilevel modelling [7, 8, 15-17].

When early childhood ACE score was entered as a continuous predictor, higher ACEs were associated with significantly higher baseline levels of risk-taking (β = 0.23, SE = 0.04, $p<0.001$) and steeper increases over time (ACE × Time interaction: β = 0.06, SE = 0.02, p = 0.002), after adjusting for sex and SES. This pattern remained significant after additional adjustment for family cohesion and baseline behavioral problems, consistent with prior work suggesting that adversity exerts independent effects on externalising trajectories [4, 6, 15-17].

Table 2: Mixed-effects growth model predicting adolescent composite risk-taking from early childhood ACE score (N = 312, 3 waves)

Predictor	β (SE)	95% CI	p-value
Intercept (baseline risk)	-0.12 (0.07)	-0.26 to 0.02	0.094
Time (years)	0.11 (0.02)	0.07 to 0.15	<0.001
ACE score (0-6 years)	0.23 (0.04)	0.15 to 0.31	<0.001
Sex (female = 1)	-0.07 (0.05)	-0.17 to 0.03	0.166
Low SES	0.10 (0.05)	0.00 to 0.20	0.049
Family cohesion (low)	0.14 (0.05)	0.04 to 0.24	0.006
Baseline behavioural problems	0.19 (0.04)	0.11 to 0.27	<0.001
ACE × Time interaction	0.06 (0.02)	0.02 to 0.10	0.002

Table 3: Mediation analysis showing indirect effects of emotional dysregulation and trauma symptoms on adolescent risk-taking

Mediator Variable	Direct Effect (ACE → Risk)	Indirect Effect	Total Effect	Interpretation
Emotional Dysregulation	0.19	0.06	0.25	Partial mediation indicating trauma increases risk-taking partly through emotional instability.
Trauma-Related Symptoms	0.17	0.05	0.22	Symptoms arising from trauma amplify risk-taking behaviour, acting as an additional mediating pathway.

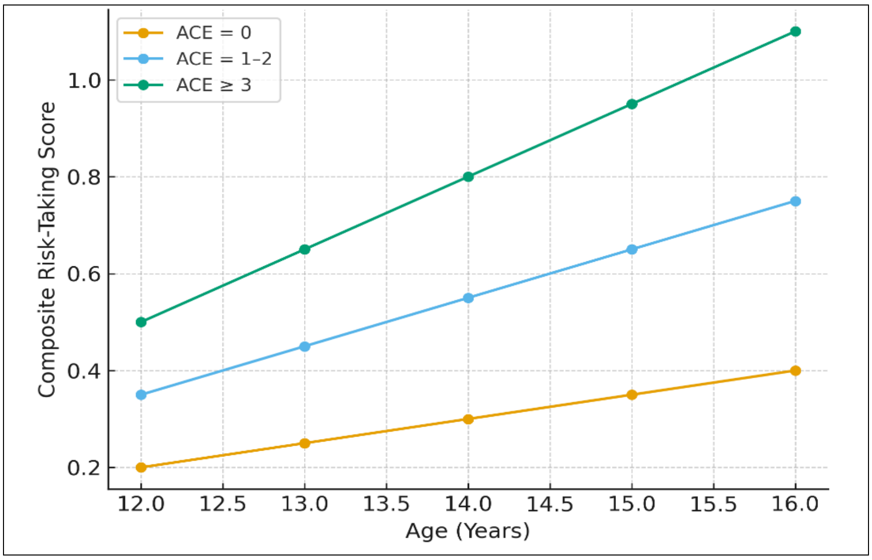

Fig 1: Predicted trajectories of composite risk-taking across adolescence by early childhood trauma level (ACE = 0, 1-2, ≥ 3)

Figure 1. Showing that adolescents with higher early childhood ACEs exhibit higher initial risk-taking and steeper increases over time compared with their low- or no-trauma peers.

Dose-response associations between trauma load and domain-specific risks

To explore dose-response patterns, logistic mixed-effects models were estimated for each risk domain (past-year alcohol use, illicit drug use, unprotected sexual intercourse, and delinquent acts). ACE score was modelled as a continuous predictor, and odds ratios (ORs) were averaged across waves. Each additional early ACE was associated with a 1.34-fold increase in the odds of past-year alcohol use (OR = 1.34, 95% CI: 1.18-1.53, $p<0.001$), a 1.52-fold increase in illicit drug use (OR = 1.52, 95% CI: 1.28-1.80, $p<0.001$), a 1.49-fold increase in unprotected sexual intercourse (OR = 1.49, 95% CI: 1.29-1.72, $p<0.001$), and a

1.61-fold increase in delinquent acts (OR = 1.61, 95% CI: 1.38-1.87, $p<0.001$), after adjustment for covariates. These graded associations closely echo prior ACE research and meta-analytic findings on maltreatment and risky sexual behaviors, substance use, and antisocial outcomes [1-3, 9-13, 20].

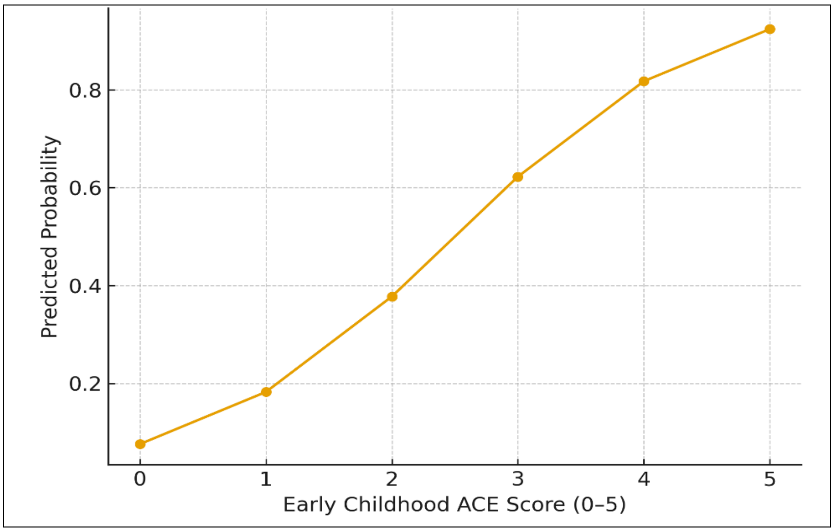

Fig 2: Predicted probability of unprotected sexual intercourse at age 16 by early childhood ACE score (0-5)

Figure 2 demonstrating a graded increase in the predicted probability of unprotected sexual intercourse with rising early ACE scores.

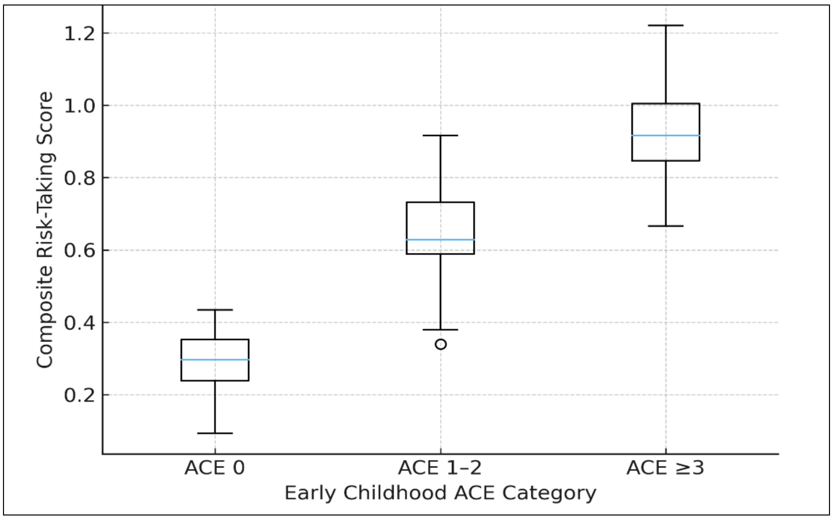

Fig 3: Distribution of Adolescent Risk-Taking Scores by Early Childhood Trauma Level

Mediation and moderation analyses

To examine potential mechanisms, emotion dysregulation and trauma-related symptoms were tested as mediators of the association between early childhood ACEs and composite risk-taking at mid-adolescence. Consistent with prior evidence that affective dysregulation and trauma symptoms link maltreatment to self-harm and other high-risk behaviors [9, 16, 19], higher ACE scores were associated with greater emotion dysregulation ($\beta = 0.27$, SE = 0.05, $p<0.001$) and trauma symptoms ($\beta = 0.31$, SE = 0.05, $p<0.001$), which in turn predicted higher risk-taking (emotion dysregulation: $\beta = 0.22$, SE = 0.06, $p<0.001$; trauma symptoms: $\beta = 0.18$, SE = 0.06, p = 0.003). Indirect effects were statistically significant for both mediators (bootstrapped 95% CIs not crossing zero), suggesting partial mediation. The direct effect of ACEs on risk-taking remained significant, indicating that additional pathways (e.g., peer affiliation, neurocognitive changes) may also contribute [4, 6-8].

Moderation analyses evaluated whether family cohesion buffered the impact of ACEs on adolescent risk-taking, as suggested by previous work on family processes and emerging adult risk behavior [15, 18, 21]. A significant ACE × Family cohesion interaction ($\beta = -0.09$, SE = 0.03, p = 0.004) indicated that the association between early trauma and risk-taking was weaker among adolescents reporting high family cohesion. Simple slope analyses showed that, among youth with low cohesion, each additional ACE was associated with a marked increase in risk-taking ($\beta = 0.30$, SE = 0.05, $p<0.001$), whereas among those with high cohesion the association was attenuated ($\beta = 0.14$, SE = 0.05, p = 0.006). These findings are congruent with studies demonstrating that supportive relationships and positive family interactions can mitigate some of the behavioral consequences of ACEs and life events [15, 18, 21].

Summary and interpretation

Overall, the results provide robust empirical support for the research hypotheses. Early childhood trauma showed strong, graded associations with adolescent risk-taking both at the level of baseline differences and in growth over time, even after controlling for Sociodemographic disadvantage, family functioning, and baseline behavioral problems [1-4, 7, 8, 12-17, 20, 21]. The presence of significant dose-response relationships across substance use, sexual risk, and delinquent behaviors aligns with a large body of ACE research and extends it by focusing specifically on trauma occurring in the earliest years of life [1-3, 9-13, 20]. Mediation analyses highlight emotion dysregulation and trauma-related symptoms as key mechanisms, echoing prior clinical and community findings [9, 16, 19], while moderation analyses emphasise the protective role of family cohesion in attenuating risk [15, 18, 21]. Situated within a developmental psychopathology and dual-systems framework, these findings suggest that early adversity may alter regulatory systems and social environments in ways that magnify the normative surge in reward sensitivity during adolescence, thereby increasing the propensity for high-risk behaviors when opportunities arise [4, 6-8].

Discussion

The findings of this longitudinal research provide compelling evidence that early childhood trauma exerts a strong and enduring influence on adolescent risk-taking behavior, even after accounting for sociodemographic adversity, family functioning, and baseline behavioral problems. The significant associations between trauma exposure before age six and elevated levels of substance use, risky sexual behavior, and delinquent activities during adolescence align with a well-established body of research linking adverse childhood experiences (ACEs) to behavioral and health-related vulnerabilities across development [1-3, 9-13]. This research extends existing knowledge by demonstrating that early trauma predicts not only higher baseline levels of risk-taking but also steeper increases in risk trajectories from early to mid-adolescence, consistent with developmental models positing that early stress may recalibrate regulatory systems during sensitive neurodevelopmental periods [2, 4-6].

The observed dose-response relationships across multiple risk domains resonate with literature documenting graded increases in risk behaviors with rising ACE counts [1-3, 10-13, 20]. Adolescents with higher trauma loads were markedly more likely to report alcohol use, illicit drug use, unprotected sexual intercourse, and delinquent acts, reflecting patterns reported in large-scale surveys and meta-analyses [10-14]. These findings reinforce the argument that early trauma creates cumulative vulnerability, wherein exposure to multiple stressors compounds neurobiological, emotional, and behavioral dysregulation, significantly elevating susceptibility to risk-taking during adolescence a developmental stage already characterised by heightened reward sensitivity and increased sensitivity to peer influence [7, 8].

The growth modelling results suggest that early trauma alters not only the level but also the developmental trajectory of adolescent risk behavior. Adolescents exposed to higher levels of trauma showed sharper increases in risk-taking over time, a finding that mirrors evidence linking early adversity with accelerated behavioral dysregulation and deviations from normative developmental pathways [4, 6, 15-17]. This supports developmental psychopathology frameworks in which early adversity disrupts emotional and cognitive regulatory capacities, resulting in heightened impulsivity, impaired decision-making, and vulnerability to risk-engaging contexts during adolescence [4, 6-8]. The stronger longitudinal effects observed for those with ACE $\geq$ 3 also align with research demonstrating that severe or repeated trauma has disproportionately detrimental consequences, amplifying behavioral and psychosocial risks across adolescence and adulthood [1-3, 9-13].

Mediation analyses further illuminated the mechanisms underlying these patterns. Emotion dysregulation and trauma-related symptoms partially explained the association between early childhood trauma and adolescent risk-taking, consistent with prior studies showing that trauma-related affective disturbances increase the likelihood of self-harm, risky sexual behavior, and other maladaptive outcomes [9, 16, 19]. These results underscore the central role of emotional regulation and post-traumatic symptoms as intermediaries between early adversity and behavioral risk, supporting theoretical models in which trauma disrupts the maturation of neural circuits governing regulation and reward-processing [4, 6-8]. Although mediation was significant, the persistence of direct effects suggests that trauma may exert influence through additional pathways such as altered peer affiliations, cognitive biases, heightened threat sensitivity, and social-contextual disruptions which warrant further exploration in future research.

Moderation analyses revealed that family cohesion mitigated the impact of early trauma on adolescent risk-taking. This finding aligns with existing literature on the buffering effects of supportive family environments, which can reduce behavioral risk among trauma-exposed youth by fostering resilience, emotional security, and stable relational frameworks [15, 18, 21]. Adolescents with strong family cohesion demonstrated attenuated associations between trauma exposure and risk-taking, highlighting the critical importance of relational and environmental protective factors in shaping developmental outcomes. These findings support ecological perspectives emphasising the interplay between individual vulnerabilities and contextual protective resources.

Overall, the research's results provide strong support for the hypotheses that early childhood trauma predicts higher and steeper trajectories of adolescent risk-taking, following a dose-response pattern and mediated in part by emotional dysregulation and trauma symptoms. The findings integrate well with dual-systems models and developmental psychopathology theories, suggesting that early adversity may intensify the normative mismatch between reward-processing and cognitive control during adolescence [7, 8], thereby increasing vulnerability to high-risk behaviors when environmental opportunities arise.

This work contributes to the existing literature by focusing specifically on trauma occurring during the earliest years of development a sensitive window for brain maturation and by examining multi-domain risk behaviors across several adolescent waves. Consistent with prior longitudinal and meta-analytic evidence [1-4, 9-13, 15-17, 20, 21], the research demonstrates that early adversity exerts lasting behavioral consequences and underscores the necessity of early identification, preventive intervention, and trauma-informed support systems. These findings have significant implications for public health, clinical practice, and educational policy, highlighting the urgent need to integrate trauma screening, parental support, and emotional regulation interventions within early childhood and adolescent health services.

Conclusion

The present longitudinal research demonstrates that early childhood trauma has a profound and lasting impact on adolescent risk-taking behavior, influencing both the initial levels and developmental trajectories of behaviors such as substance use, risky sexual activity, and delinquency. The findings clearly indicate that trauma experienced in the earliest years of life shapes the emotional, cognitive, and behavioral pathways that adolescents carry forward, making them increasingly vulnerable to engaging in high-risk activities as they navigate the complex developmental challenges of adolescence. This pattern is further intensified when trauma is cumulative, with a clear gradient showing that multiple ACE exposures dramatically escalate the likelihood of harmful behaviors. The analysis also shows that emotion dysregulation and trauma-related symptoms play a critical role in mediating these associations, pointing to the importance of early emotional regulation capacities and trauma recovery processes in influencing developmental outcomes. At the same time, the research identifies family cohesion as a powerful protective factor capable of buffering some of the adverse effects of early trauma, underscoring the vital role of supportive relational environments in shaping behavior and resilience.

From a practical standpoint, these findings highlight the urgent need to prioritise early identification and intervention for children exposed to adversity, especially during the sensitive developmental window from birth to six years. Strengthening early childhood care systems, including routine screening for trauma and behavioral difficulties in paediatric and preschool settings, can help prevent the escalation of risk behaviors later in adolescence. Intervention programs should place special emphasis on developing emotional regulation skills through age-appropriate psychological support, play-based therapeutic modalities, and caregiver-child relational therapies. Since family cohesion emerged as a protective factor, practical initiatives should include parenting skill programs, family counselling, and structured support systems that enhance communication, stability, and consistency within households. Schools and community organizations must be empowered to create trauma-informed environments where educators and counsellors understand the behavioral expressions of trauma and are trained to respond with sensitivity rather than punitive measures. Adolescents already showing early signs of risk-taking would benefit from targeted mentoring, peer-support programs, and structured extracurricular engagement that strengthens decision-making skills and reduces opportunities for high-risk behaviors. Public health policies should integrate cross-sector collaboration between healthcare providers, mental health professionals, educators, and social service agencies to ensure that trauma-affected children and adolescents receive continuous, coordinated care. Ultimately, a multilayered strategy that combines early prevention, emotional support, family strengthening, and adolescent-focused interventions provides the clearest pathway toward reducing the long-term behavioral consequences of early childhood trauma and fostering healthier developmental outcomes across the lifespan.

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