Association of childhood maltreatment with alcohol use disorder in adulthood and potential mediation by stress regulation

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Abstract

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Background: While childhood maltreatment (CM) is a well-established risk factor for alcohol use disorder (AUD) during adolescence and young adulthood, few longitudinal studies have assessed this association into midlife. In addition, mechanisms of the association as well as the influence of CM exposure timing on AUD risk remain unclear. This study examined the longitudinal association of CM (including the age of initiation) with AUD in adulthood, and whether this association is explained by stress regulation.

Methods: Data were drawn from the Seattle Social Development Project (SSDP), a longitudinal study that recruited students entering the 5th grade from lower income neighborhoods in Seattle, WA. CM was examined both as a binary variable (yes/no experience) and a multinomial categorical variable based on age of initiation (no CM, CM initiation prior to age 10, CM initiation ages 10-17). AUD was defined as ever meeting DSM-IV criteria for alcohol abuse or dependence between ages 27 and 47. The stress regulation variable was examined as a continuous score based on the mean value (range 1-5) of four items measured at age 27. Modified Poisson regression models were used to estimate prevalence ratios (PR) for AUD in

adulthood by CM exposure as well as the timing of initiation of CM. PR from models with and without adjustment for stress regulation were compared to assess whether stress regulation attenuated the association.

Results: The overall prevalence of CM in the analytic sample was 34.6%. Exposure to any CM was associated with a 44% higher prevalence of AUD in adulthood compared to no CM before (aPR=1.44, 95% CI: 1.19, 1.76) and after (aPR=1.44, 95% CI: 1.19, 1.76) adjustment for stress regulation. The association of CM with AUD was pronounced among those who experienced CM initiation prior to age 10 (aPR=1.48, 95% CI: 1.21, 1.81) compared to those who experienced CM initiation between ages 10 to 17 (aPR=1.23, 95% CI: 0.77, 1.98).

Conclusions: CM, particularly when initiated prior to age 10, was associated with increased prevalence of AUD between ages 27 and 47. Stress regulation did not appear to explain this association. These results underscore the need for trauma-informed prevention and treatment approaches for AUD across the life course.

Background

Adverse childhood experiences (ACEs) and childhood maltreatment (CM) are risk factors for the development and persistence of mental and behavioral health (M/BH) problems.[1–4] CM, a component of ACEs, is characterized by physical, emotional, or sexual abuse and physical or emotional neglect prior to age 18.[5] Notably, people with substance use disorders (SUDs) are more likely to have experience with CM.[4,6,7] CM is also a risk factor for increased SUD severity and lower success of treatment.[7,8] SUDs are among the M/BH conditions with the highest mortality rates globally, with alcohol use disorder (AUD) being among the most prevalent.[9,10] An estimated 28.9 million people aged 12 or older in the United States met criteria for AUD within the past year in 2023.[11]

The relationship between CM and alcohol-related outcomes has been widely studied;[4,12–14] however, this research has primarily used retrospective methods and focused on outcomes in late adolescence and young adulthood. There is limited longitudinal research that follows individuals into midlife, restricting the ability to understand how CM influences AUD across the life course. Additionally, this research has primarily focused on exposure to CM at any age before 18. Childhood is a time of rapid neurological development, with distinct changes occurring at various developmental stages that correspond to chronological age.[15,16] Therefore, timing of CM may differentially impact AUD. Prior research has identified that earlier exposure to CM is more strongly associated with depression, anxiety, and post-traumatic stress disorder in adulthood than later CM exposure.[17–20] However, research specifically examining how the timing of CM influences AUD remains limited, suggesting that later CM onset may be more strongly associated with alcohol-related outcomes than earlier CM onset.[20,21]

Mechanisms underlying the association of CM with AUD are still unclear. Some proposed mechanisms include neurobiological alterations such as structural brain changes[22,23] and systemic inflammation, reflected by elevated inflammatory[24] and immune biomarkers.[25]

Other studies apply the self-medication hypothesis,[26] suggesting that people who experience CM are more likely to engage in substance use to manage emotional dysregulation and alleviate stress. These studies suggest that perceived stress, emotional distress, and alcohol-related coping motives may be key mechanisms contributing to the association of CM with AUD, particularly in young adulthood.[27–30] Collectively, these findings highlight the potential utility of targeting stress regulation--the ability to manage, reduce, and adaptively cope with stress--as a point of intervention for AUD prevention and treatment. However, this pathway remains understudied.

This study addressed existing gaps that have hindered our understanding of the relationship of CM with AUD and factors influencing the relationship. Using prospectively collected data from the Seattle Social Development Project, this study examined the longitudinal association of CM with AUD in adulthood. It also examined how age of CM onset influences this association to identify periods of heightened vulnerability. Finally, this study examined whether stress regulation in adulthood explains, at least in part, the association of CM with AUD. Study findings can be used to inform timing and focus of prevention efforts by identifying potential developmental periods during which CM may confer increased risk for developing AUD and may inform future research to guide more targeted public health interventions.

Methods

Study Setting & Study Population

Data for this study were obtained from the Seattle Social Development Project (SSDP).[31] SSDP is a longitudinal study that began in 1985 to assess health behaviors and outcomes across development. All students entering the 5th grade from 18 public schools serving lower income neighborhoods in Seattle, Washington, were eligible for study enrollment. Out of 1053 eligible students, 808 (77%) were enrolled in the study. Data were collected annually between ages 10 through 18, and 8 additional assessments were completed at ages 21, 24, 27, 30, 33, 35, 39 and 47.

Participants with missing responses to all variables associated with the primary exposure, CM, all variables associated with the primary outcome, AUD, or any of the adjustment covariates were excluded from this analysis. The final analytic study sample included 699 (87%) of the original 808 participants within the SSDP study population. Ninety-three individuals were excluded for missing responses to all childhood maltreatment (CM) indicators or all alcohol use disorder (AUD) assessment years. An additional 16 individuals were excluded due to missing responses to the stress regulation or family history of alcohol problems covariates. Informed consent was obtained from all participants, and the study was approved by the Human Subjects Review Committee at the University of Washington.

Data Collection

This analysis included data from constructed variables derived from school records and adulthood assessment at ages 24, 27, 30, 33, 39, and 47. At age 24, study assessments were conducted in-person and interviews were administered by trained study staff. All other adulthood assessments combined in-person surveys and either password-protected web-based surveys or telephone or paper surveys if requested.

Childhood Maltreatment (CM), Alcohol Use Disorder (AUD), and Stress Regulation

CM was assessed at the age 24 study wave using the short-form Childhood Trauma Questionnaire (CTQ-SF).[32] This 25-item questionnaire retrospectively measured experiences with abuse (physical, emotional, and sexual) and neglect (physical and emotional) prior to age 18. Responses were measured on a 5-point Likert scale based on frequency of exposure (1=never, 2=rarely, 3=sometimes, 4=often, 5=always). For any item endorsed as being experienced, participants were subsequently asked whether the experience occurred before age 10. To maintain an adequate sample size, missing responses on individual items were assumed to indicate no exposure rather than being excluded.

To guide the operational definition of exposure to CM, this analysis referenced an approach used in the 10-item Adverse Childhood Experiences (ACE) Questionnaire.[33] This approach includes dichotomous items for different subtypes of CM based on frequency of exposure. Consistent with the ACE questionnaire, sexual abuse was coded as present if reported at any non-zero frequency, whereas physical and emotional maltreatment were coded as present only if reported as occurring often or always. Two CM variables were created for this analysis. A dichotomous variable was created to assess ever experiencing CM prior to age 18 [1=yes, 0=no] and a categorical variable was created to assess timing of initiation to CM [1=no CM, 2=CM initiation prior to age 10, 3=CM initiation between ages 10-17].

To maintain conceptual consistency with ACEs-based definitions and ensure accurate classification of exposure, some CTQ-SF indicators were excluded from the construction of the CM variables. Seven positively worded indicators were removed because they lacked follow-up questions about whether the experience occurred prior to age 10, preventing classification of CM initiation. An additional two indicators were also removed because they did not align with the terminology of ACE questions, and therefore, they could not be reliably interpreted as stand-alone markers of maltreatment when applying a dichotomous coding scheme. This ultimately

resulted in the removal of all emotional neglect indicators from this analysis. Finally, one indicator was removed because of a conceptual overlap with the family history of alcohol problems adjustment variable. Fourteen out of the original 25 CTQ-SF indicators were used to create the constructed CM variables for the purpose of this analysis (**Table 1**).

AUD criteria were assessed using the Diagnostic Interview Schedule (DIS) based on DSM-IV criteria[34] at the age 27, 30, 33, 39, and 47 assessment waves. The DIS is a fully structured diagnostic interview designed for use by trained lay interviewers and has been widely used in epidemiological and clinical research. A dichotomous variable was created to assess ever meeting DSM-IV criteria for alcohol abuse or alcohol dependence at any point across the ages 27, 30, 33, 39, and 47 [1=yes; 0=no]. To maintain an adequate sample size, missing responses for individual survey waves were assumed to indicate not meeting AUD criteria rather than excluding the participant.

Stress regulation was assessed with the following questions: 1) “How often do you control stress in your life?”, 2) “How often do you keep stress levels low?”, 3) “How often do you do something good for yourself each day?”, 4) “How often do you take time for relaxation every day?” Each item was measured on a 5-point Likert scale (1=never, 2=rarely, 3=sometimes, 4=often, 5=always) during the age 27 survey wave. A continuous stress regulation variable was created by calculating the mean score across the items (range 1-5), with higher scores indicating better stress regulation. Internal consistency was high in this sample (alpha=0.77).

Covariates

The following confounders were selected *a priori* for adjustment: sex [1=female, 0=male] as a proxy for gender identity, race [Caucasian, African American, Native American, Asian American], free/reduced school lunch between 5^{th}-7^{th} grade [1=yes, 0=no] as a proxy for childhood household socioeconomic status, and family history of alcohol problems [1=yes,

0=no]. Race was used as a proxy for racism because it may be associated alcohol consumption and with whether someone acknowledges experiencing CM through perceived stigma,[35,36] chronic stress,[36] and institutional discrimination.[35] Sex, race, and free/reduced school lunch status were constructed variables derived from school records and multiple self-reports from study interviews. Family history of alcohol problems was measured during the age 24 assessment based on the interview question: “Has anyone in your family had alcohol problems?”

Statistical Analyses

Selected characteristics were summarized for all study participants stratified by CM exposure. Means and standard deviations were reported for continuous variables, and percentages were reported for categorical variables. Modified Poisson regression models were used to examine the association of CM exposure with AUD in adulthood. Three models were run: an unadjusted model, a fully adjusted model (primary model), and an extended model. The fully adjusted model controlled for sex, race, free/reduced lunch status, and family history of alcohol problems. The extended model additionally included stress regulation to evaluate the extent to which the PR associated with CM was attenuated. Modified Poisson regression models were also used to examine the association of age of CM initiation with AUD in adulthood. CM initiation was included as a categorical variable with three levels: no CM exposure (reference group), CM onset prior to age 10, and CM onset between ages 10 and 17. All estimates reflect comparisons to the no CM exposure group. Two models were run: an unadjusted and fully adjusted model. The fully adjusted model included all the same covariates as described in the previous fully adjusted model. In sensitivity analyses, all analytic models were reproduced using a different operationalization of CM exposure. These analyses used a more inclusive definition of exposure that applied consistent thresholds across CM subtypes.

All analyses were conducted using R version 4.4.2.

Results

The overall prevalence of CM in the study population was 34.6%. The prevalence of experience with individual CM indicators are shown in **Table 1**. Among participants that reported CM exposure, the majority indicated exposure onset prior to age 10 across all CM subtypes. Sexual abuse indicators were endorsed more frequently than physical abuse, emotional abuse, and physical neglect items. Distribution of study sample characteristics overall and by experience of CM is shown in **Table 2**. The analytic sample was 51.0% female, 49.1% Caucasian, 50.4% qualified for free/reduced school lunch, and 45.5% had a family history of alcohol problems. Respondents who experienced CM were more likely to be female, African American, qualify for free/reduced school lunch, and have a family history of alcohol problems compared to respondents who did not experience CM. The mean stress regulation score was lower among those who experienced CM (3.13) than those who did not experience CM (3.33).

The overall prevalence of AUD in adulthood was 44.2% among individuals who experienced CM and 31.3% among individuals who did not experienced CM (**Table 3**). Compared to those who did not experience CM, those who experienced CM had a 41% higher prevalence of AUD in adulthood in the unadjusted model (PR=1.41, 95% CI: 1.16, 1.72) and a 44% higher prevalence of AUD in adulthood after adjusting for covariates (aPR=1.44, 95% CI: 1.19, 1.76). No attenuation of results was observed in the extended model that included stress regulation (aPR=1.44, 95% CI: 1.19, 1.76).

The overall prevalence of AUD in adulthood was 46.5% among individuals whose CM experience was initiated prior to age 10 and 32.5% among individuals whose CM experience was initiated between ages 10 and 17 (**Table 4**). Compared to individuals with no CM experience, those who experienced CM initiation prior to age 10 had a 48% higher prevalence of AUD in adulthood in both the unadjusted and adjusted models (PR=1.48, 95% CI: 1.22, 1.82; aPR= 1.48, 95% CI: 1.21, 1.81). In the unadjusted model, individuals who experienced CM

initiation between ages 10 and 17 had a similar prevalence of AUD in adulthood compared to participants with no CM exposure (PR=1.04, 95% CI: 0.65, 1.66). The PR increased to 1.23 after adjusting for sex, race, free/reduced school lunch status, and family history of alcohol problems, though it remained statistically non-significant (aPR=1.23, 95% CI: 0.77, 1.98).

The results of the sensitivity analyses, which used a more inclusive definition of CM exposure for physical and emotional maltreatment indicators, were largely similar to the main findings (**Tables 5 and 6**).

Discussion

This study found an overall CM prevalence of 34.6% among study participants. Individuals who experienced CM had a 44% higher prevalence of AUD in adulthood compared to those unexposed to CM. This association was particularly pronounced among individuals who first experienced CM prior to age 10 (48% higher prevalence). The association of CM with AUD was not attenuated by stress regulation, suggesting a lack of evidence for stress regulation mediating the relationship.

These findings are consistent with other studies that have identified an association of CM with alcohol-related outcomes.[2–4,12–14] For example, de Waal et al. found that individuals exposed to CM had 1.21 times that odds of problematic alcohol use.[13] Similarly, Kisley et al. found that individuals with substantiated emotional abuse had 1.86 times higher odds of heavy alcohol consumption by age 21.[14] A scoping review by Leza et al. identified that adverse childhood experiences, which encapsulate CM, are positively associated with both the development and severity of AUD.[4] While these studies vary in design and population, findings were consistent in showing a positive association of CM with alcohol-related outcomes. This study extends this literature by contributing longitudinal evidence that supports the association of CM with AUD into midlife. Thus, these results underscore the importance of CM prevention and interventions to support individuals with CM experience across the life course.

Findings regarding CM timing may appear to differ from previous studies suggesting that later CM exposure may be more strongly associated with alcohol-related outcomes in adulthood than earlier CM exposure.[20,21] For example, Kaplow and Widom found that individuals first exposed to CM between ages 9 and 11 were at higher risk of adulthood AUD compared to earlier CM exposure.[20] Similarly, Thornberry et al. found that CM onset between ages 12 and 17 were more strongly associated with problematic alcohol use adulthood compared to CM onset prior to age 12.[21] Several methodological differences may explain these discrepancies. Notably,

both prior studies used substantiated records of CM exposure, whereas the present study used retrospective self-reports. Self-reported data may capture a broader spectrum of CM experiences, including those that are unreported or unrecognized by formal systems, including earlier or less visible forms of maltreatment. This could lead to differential identification of high-risk groups. Additionally, Kaplow and Widom only assessed CM exposure through age 11; therefore, their highest risk age group (ages 9 to 11) partially overlaps with both age groups used in this study, further complicating direct comparability.[20] While the current study assessed age of CM initiation, it did not capture duration of exposure. It is possible that individuals with early-onset CM experienced prolonged maltreatment into later adolescence, which may contribute to the stronger association observed in this age group. Thornberry et al. distinguished between CM that occurred only before age 12 and CM that occurred between ages 12 and 17, the latter of which may have reflected new or prolonged exposure.[21] Notably, both Thornberry et al. and the present study found that the age group operationalized to potentially include prolonged CM exposure had the strongest associations with alcohol-related outcomes in adulthood. While this pattern is suggestive, the current study does not allow clear differentiation between the effects of age of onset and duration. These findings highlight the need for future research that more explicitly disentangles the role of timing and duration of CM exposure.

This study did not find evidence of stress regulation mediating the association of CM with AUD. While stress regulation was significantly associated with CM, it was not significantly associated with AUD, suggesting that the lack of mediation evidence was driven by the absence of a direct pathway between stress regulation and AUD. Although prior studies have not examined stress regulation specifically, they have identified related constructs, such as perceived stress and coping methods, as mechanisms linking CM and AUD.[27–30] It is possible that these constructs may have a larger role on this pathway than stress regulation because they capture experiences more proximally tied to drinking behavior. It is also possible that stress

regulation may be more meaningful earlier in the life course. In this study, stress regulation was assessed at age 27, which may be after key developmental windows when stress-related mechanisms influence risk of AUD. Finally, the absence of mediation evidence may be population-specific, as this analytic cohort was recruited from public schools in low-income neighborhoods in Seattle, WA. In this context, the role of stress regulation in shaping adulthood alcohol outcomes may differ due to shared experiences of chronic adversity during childhood, resulting in alternative resiliency strategies.[37]

Results from the sensitivity analysis further support the robustness of the main findings. The sensitivity analysis used a more inclusive definition of exposure that applied consistent thresholds across CM subtypes. In the main analysis, a stricter definition of CM exposure was used to account for the historical context in which participants were raised. As the study cohort entered fifth grade in 1985, certain experiences were more socially accepted forms of parental discipline, contributing to high endorsement of certain indicators. For instance, over half (52.6%) of participants reported being punished with a belt, board, cord, or other hard object at any non-zero frequency. To improve specificity, higher frequency thresholds were required for physical and emotional maltreatment to be classified as exposed in the main analysis. In contrast, sexual maltreatment indicators did not require the same thresholds, as these behaviors have not been subject to historic normalization. The consistency of findings across both operational definitions strengthens confidence in the results and suggests that the observed association of CM with AUD were not driven by CM operationalization choices.

The burden of CM observed in this sample offers important context to interpret and generalize these findings. The overall prevalence of CM was 34.6%, with particularly high endorsement for sexual abuse indicators, ranging from 6.6% to 18.0%. Emotional abuse indicators endorsement ranged from 7.3% to 9.0% and physical abuse indicators ranged from 1.4% to 13.3%. These results are consistent with population-based studies. For example,

Gilbert et al. conducted a systematic review and reported lifetime prevalence estimates ranging from 15% to 30% for sexual abuse and 5% to 35% for physical abuse across studies, which were primarily derived from retrospective measurements.[38] In contrast, these results are much higher than studies using court-substantiated reports. For instance, research using the National Child Abuse and Neglect Data System found a lifetime CM prevalence of 12.5% in the United States.[39] Notably, the present cohort entered the 5th grade in 1985, a period when substantiated physical and sexual abuse reached a national peak.[40] As such, the observed CM burden may reflect both a historical context and the broader sensitivity of retrospective measurement.

The burden of CM identified in this study also raises broader methodological and equity considerations for how CM is measured in research. Meta-analytic evidence has demonstrated poor agreement between prospective and retrospective measures of CM, indicating that these approaches identify different populations.[41] Retrospective self-report, as used in this study, may capture a broader range of CM experiences that were never formally reported or substantiated. Emerging research has challenged the long-standing view of court-substantiated records as a "gold standard" for CM measurement. Narayan et al. argue that substantiation is shaped by structural racism in the child welfare system, leading to both over and under-identification of CM in marginalized communities.[35] Future research should prioritize multi-method approaches and treat retrospective self-reports as valid and meaningful measurements. This study's use of retrospective reports with carefully defined frequency thresholds represents an effort to capture CM exposure in a way that is both methodologically rigorous and responsive to the cultural and historical context of the cohort.

This study is subject to limitations and should be interpreted within the context of the study design. The most significant limitation was the presence of missing data. Approximately 13% of the original SSDP sample was excluded due to missingness of key variables. Additionally, within the included sample, missingness of individual CM indicators and AUD

assessment waves may have led to misclassification. Missing CM indicators were coded as unexposed and missing AUD assessment waves were coded as not meeting AUD criteria. There may also be residual confounding of parental socioeconomic status (SES). In this study, free/reduced school lunch status was used as a proxy, so this measure may not fully capture the multidimensional nature of SES. More direct parental SES variables were available in the dataset but were excluded due to high missingness. Finally, generalizability may be limited, as the SSDP sample was drawn from youth in lower income neighborhoods in Seattle, Washington.

In sum, this study found increased prevalence of AUD in adulthood among individuals exposed to CM, particularly among those exposed prior to age 10, compared to individuals without CM exposure. These findings contribute to a growing body of literature on the long-term health effects of CM. These results reinforce the importance of using trauma-informed approaches to AUD prevention and treatment, particularly among populations that experience early life adversity. Given that the prevalence of AUD was high into midlife, public health interventions should use a life course perspective and continue to provide support for CM beyond young adulthood.

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Table 1: Prevalence of childhood maltreatment indicators by subtype from the short-form Childhood Trauma Questionnaire.

Childhood Maltreatment Indicator	**Overall childhood maltreatment prevalence*** n (%)	**Maltreatment initiation prior to age 10[ŦŦ]** n (%)	**Maltreatment initiation between ages 10-17[†]** n (%)
Physical Abuse[α]			
I got hit so hard by my mother or father (or other primary caregiver) that I had to see a doctor or go to the hospital.	12 (1.7)	10 (1.4)	0 (0.0)
My mother or father (or other primary caregiver) hit me so hard that it left me with bruises or marks.	26 (3.7)	23 (3.3)	1 (0.1)
I was punished with a belt, a board, a cord, or some other hard object.	93 (13.3)	81 (11.6)	8 (1.1)
I got hit or beaten so badly that it was noticed by someone like a teacher, neighbor, or doctor.	10 (1.4)	6 (0.9)	1 (0.1)
I believe that I was physically abused.	36 (5.2)	32 (4.6)	1 (0.1)
Emotional Abuse[α]			
My mother or father (or other primary caregiver) called me things like “stupid” or “lazy” or “ugly”.	52 (7.4)	38 (5.4)	9 (1.3)
My mother or father (or other primary caregiver) said hurtful or insulting things to me.	51 (7.3)	34 (4.9)	14 (2.0)
I believe that I was emotionally abused.	63 (9.0)	45 (6.4)	12 (1.7)
Sexual Abuse[β]			
My mother or father (or other primary caregiver) tried to touch me in a sexual way, or tried to make me touch them.	126 (18.0)	94 (13.4)	29 (4.1)
My mother or father (or other primary caregiver) threatened to hurt me or tell lies about me unless I did something sexual with them.	46 (6.6)	34 (4.9)	9 (1.3)
My mother or father (or other primary caregiver) tried to make me do sexual things or watch sexual things.	99 (14.2)	83 (11.9)	14 (2.0)

My mother or father (or other primary caregiver) molested me.	112 (16.0)	89 (12.7)	21 (3.0)
I believe that I was sexually abused.	103 (14.7)	77 (11.0)	22 (3.1)
Physical Neglect[α]			
I had to wear dirty clothes.	12 (1.7)	8 (1.1)	1 (0.1)
I didn't have enough to eat.	11 (1.6)	6 (0.9)	3 (0.4)

*Overall prevalence of childhood maltreatment. Prevalence reflects participants who responded to the indicator item (missingness ranges from 0.9% to 3.0%). Age of childhood maltreatment initiation was asked in a separate question; therefore, the sum of the childhood maltreatment initiation categories may not equal the overall prevalence.

[T]Prevalence of childhood maltreatment that was initiated prior to age 10. Prevalence reflects participants who responded to the indicator item (missingness ranges from 1.3% to 3.3%).

[†]Prevalence of childhood maltreatment that was initiated between ages 10-17. Prevalence reflects participants who responded to the indicator item (missingness ranges from 1.3% to 3.3%).

[α]Exposure defined as selecting "Often True" or "Always True" for the indicator item.

[β]Exposure defined as selecting "Rarely True", "Sometimes True", "Often True", or "Always True" for the indicator item.

Table 2: Selected study participant characteristics by experience of childhood maltreatment.

Characteristic	**Overall** (n=699)	**No childhood maltreatment** (n=457)	**Experienced childhood maltreatment** (n=242)
Sex n (%)			
Female	356 (51.0)	217 (47.5)	139 (57.4)
Male	343 (49.0)	240 (52.5)	103 (42.6)
Race[ᵀ] n (%)			
African American	169 (24.2)	97 (21.2)	72 (29.8)
Asian American	147 (21.0)	101 (22.1)	46 (19.0)
Caucasian	343 (49.1)	236 (51.6)	107 (44.2)
Native American	40 (5.7)	23 (5.0)	17 (7.0)
Free/reduced lunch between 5th-7th grade[†] n (%)			
Yes	352 (50.4)	208 (45.5)	144 (59.5)
No	347 (49.6)	249 (54.5)	98 (40.5)
Family history of alcohol problems n (%)			
Yes	318 (45.5)	177 (38.7)	141 (58.3)
No	381 (54.5)	280 (61.3)	101 (41.7)
Stress regulation score*			
Mean score (SD)	3.26 (0.76)	3.33 (0.72)	3.14 (0.80)

[ᵀ]Participants were categorized into listed race categories due to sample size limitations, which prevented the inclusion of more specific categories.

[†]Measures whether participants qualified for free/reduced lunch at any time between 5th-7th grade.

*Score is the mean of four items measuring stress regulation (range 1-5), with higher score indicating better stress regulation.

Table 3: Prevalence ratios for alcohol use disorder in adulthood by exposure to childhood maltreatment.

Childhood maltreatment experience	**Prevalence** n (%)	**Unadjusted model** PR (95% CI)	p-value	**Fully adjusted model*** PR (95% CI)	p-value	**Expanded model**** PR (95% CI)	p-value
No	143 (31.3)	Ref	--	Ref	--	Ref	--
Yes	107 (44.2)	1.41 (1.16, 1.72)	<0.001	1.44 (1.19, 1.76)	<0.001	1.44 (1.19, 1.76)	<0.001

*Adjusted for sex, race, free/reduced lunch status, and family history of alcohol problems.

**Adjusted for sex, race, free/reduced lunch status, family history of alcohol problems, and mean stress regulation score.

Table 4: Prevalence ratios for alcohol use disorder in adulthood by timing of initiation to childhood maltreatment.

Timing of childhood maltreatment	Prevalence	Unadjusted model		Fully adjusted model*	
	n (%)	PR (95% CI)	p-value	PR (95% CI)	p-value
No childhood maltreatment	143 (31.3)	Ref	--	Ref	--
Childhood maltreatment initiation prior to age 10	94 (46.5)	1.48 (1.22, 1.82)	<0.001	1.48 (1.21, 1.81)	<0.001
Childhood maltreatment initiation between ages 10-17	13 (32.5)	1.04 (0.65, 1.66)	0.874	1.23 (0.77, 1.98)	0.388

*Adjusted for sex, race, free/reduced lunch status, and family history of alcohol problems.

Table 5: Sensitivity analysis for alcohol use disorder in adulthood by exposure to childhood maltreatment.

Childhood maltreatment experience	**Prevalence** n (%)	**Unadjusted model** PR (95% CI)	p-value	**Fully adjusted model*** PR (95% CI)	p-value	**Expanded model**** PR (95% CI)	p-value
No	38 (25.2)	Ref	--	Ref	--	Ref	--
Yes	212 (38.7)	1.54 (1.15, 2.06)	0.004	1.51 (1.13, 2.02)	0.005	1.51 (1.13, 2.02)	0.005

Sensitivity analyses used a more inclusive definition of childhood maltreatment exposure.
*Adjusted for sex, race, free/reduced lunch status, and family history of alcohol problems.
**Adjusted for sex, race, free/reduced lunch status, family history of alcohol problems, and mean stress regulation score.

Table 6: Sensitivity analysis for alcohol use disorder in adulthood by timing of initiation to childhood maltreatment.

Timing of childhood maltreatment	Prevalence	Unadjusted model		Fully adjusted model*	
	n (%)	PR (95% CI)	p-value	PR (95% CI)	p-value
No childhood maltreatment	38 (25.2)	Ref	--	Ref	--
Childhood maltreatment initiation prior to age 10	182 (40.0)	1.59 (1.18, 2.14)	0.002	1.56 (1.16, 2.09)	0.003
Childhood maltreatment initiation between ages 10-17	30 (32.3)	1.28 (0.86, 1.92)	0.227	1.28 (0.86, 1.90)	0.224

Sensitivity analyses used a more inclusive definition of childhood maltreatment exposure.
*Adjusted for sex, race, free/reduced lunch status, and family history of alcohol problems.