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Does binge drinking mediate the relationship between four adverse childhood experiences and adult traumatic brain injury? Results from the National Longitudinal Survey of Youth 1979 Cohort

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Abstract

Objective—Adverse childhood experiences (ACEs) are associated with increased risk of sustaining a traumatic brain injury (TBI). Alcohol use may play an important role in this relationship. This study examines whether binge drinking mediates the relationship between four ACEs and TBIs sustained in adulthood.

Methods—Using the National Longitudinal Survey of Youth, 1979 cohort, we conducted longitudinal mediation analyses (n=6317). Interviews occurred annually from 1979 to 1994 and biennially until 2016. We evaluated the direct and indirect effects of individual ACEs (ie, experiencing physical violence, low parental warmth, familial alcoholism and familial mental illness; reported retrospectively) and a cumulative ACEs score on mean level of binge drinking (calculated across waves) and having a TBI in adulthood. To establish temporality, we included binge drinking that was measured at age 18 or older and before any reported TBI.

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Contributors JD, ST-K and NCG designed the project; JD, SM and NCG developed the theoretical framework; ST-K and NCG performed the statistical analysis; JH-K supervised the project; all authors discussed the results and contributed to the final manuscript. JD is the guarantor of the manuscript.

Disclaimer The findings and conclusions in this manuscript are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Ethics approval This study conducted a secondary analysis of data from NLSY79, a publicly available deidentified dataset, as such did not require IRB approval or exemption. Details on the NLYS79 procedures to ensure ethical and confidential data collection can be found here: https://www.nlsinfo.org/content/cohorts/nlsy79/intro-to-the-sample/confidentiality-informed-consent. Participants gave informed consent to participate in the study before taking part.

Results—Cumulative ACEs, familial alcoholism and physical abuse exposure were significantly associated with having a TBI through binge drinking, although this only explained a small part of the association between ACEs and TBI. Other ACEs were not significantly associated with binge drinking or TBI.

Conclusion—The results indicate that while ACEs and adult TBI risk were significantly associated, lifetime binge drinking explains only a small part of the association. Future research could examine alternative social, biological and behavioural mechanisms along the pathway between ACEs and TBI. Determining this mechanism will allow public health practitioners to design and implement effective TBI prevention programmes for those at higher risk of injury due to ACE exposure.

INTRODUCTION

Caused by a bump or jolt to the head or body, traumatic brain injury (TBI) affects how the brain works.[1] Based on recent data, it is estimated that there are over 200 000 TBI-related hospitalisations and over 64 000 TBI-related deaths every year in the USA.[2] Several factors, such as one's sex[3] and risk-taking propensity,[4] increase the risk for sustaining a TBI. Previous evidence suggests a relationship between adverse childhood experiences (ACEs) and TBI.[5] ACEs are potentially traumatic events that occur in childhood (0–17 years), such as experiencing abuse or neglect.[6] ACEs are common and are associated with a wide range of negative mental and physical health outcomes during adulthood, including TBI.[7] For example, Guinn and colleagues, using data from North Carolina, found that after controlling for age, race/ethnicity, sex and income, respondents who reported experiencing sexual abuse, physical abuse, household mental illness, and had incarcerated household members in childhood had a greater odds of sustaining a TBI at some point in their lifetime.[5] Additionally, among a nationally-representative sample of adolescents (aged 12–17), exposure to ACEs was associated with a significant increase in the risk of past and current TBI.[8]

Less is known about *why* the association between ACEs and TBI exists or whether specific ACEs are responsible for the effect. Exposure to ACEs can result in a maladaptive stress response that derails optimal development by producing changes in gene expression, brain architecture, executive function, impulse control, behavioural regulation, psychiatric diagnosis and coping strategies.[6 9-11] These factors may contribute to increased risk of TBI. A related theory is that substance use mediates the relationship between ACEs and TBI. There is a strong evidence that ACE exposure increases risk of substance use,[7] particularly alcohol use, starting as early as high school.[12] Using data from the Behavioral Risk Factor Surveillance System, Fang and McNeil reported that among men, living with a drug misuser as a child and for both men and women, experiencing verbal abuse as a child, put them at increased risk of binge drinking as an adult.[13] The relationship between substance use and ACEs may be related to a combination of developmental processes, low levels of parental monitoring, peer pressure, decision-making skills and high disinhibition during this time.[12 14] For example, life course qualitative research among children in foster care found intersections between exposure to and normalisation of child adversity, and engagement in

alcohol and recreational drug use as a coping strategy, which, in turn, was related with different forms of brain injury sustained throughout their lives.[15]

Heavy or long-term alcohol use is also associated with a higher risk of sustaining a TBI.[16] Heavy or long-term alcohol consumption can change the structure of the brain[17] and produce issues with cognition[18] and executive function,[19] which may increase the lifetime risk of TBI. Furthermore, the prevalence of alcohol intoxication at the time of TBI is estimated to be between 37% and 51%.[20] Excessive alcohol use and intoxication puts one at risk of many of the most common causes of TBI, including motor vehicle crashes[21] and assault.[22] Furthermore, specific types of ACEs (rather than just the number of ACEs) have differential effects on the risk of health sequalae.[23] For example, in a longitudinal study in New Zealand, separate measures of childhood adversity related to maternal parenting and mental health were found to be associated with TBI events.[24] Examining associations between specific ACEs, binge drinking and TBI is important to better elucidate potential causal pathways. Given ACEs' relationship with binge drinking and binge drinking's strong association with TBI, we hypothesise that binge drinking is one important factor that mediates the relationship between ACEs and future TBI.

Objectives

Using data from the National Longitudinal Survey of Youth, 1979 cohort (NLSY79), this study aimed to assess the association between cumulative and individual ACEs and adult TBI and determine if lifetime binge drinking mediates the relationship between ACEs and TBI.

METHODS

Sample

NLSY79 is a nationally representative sample of 12 686 adults born during the years 1957 through 1964. Interviews occurred annually from 1979 to 1994 and on a biennial basis until 2016. The NLSY79 survey is sponsored and directed by the US Bureau of Labor Statistics and managed by the Center for Human Resource Research at The Ohio State University.[25] Interviews are conducted by the National Opinion Research Center at the University of Chicago. To establish temporality between ACEs, alcohol use, and TBI, individuals who experienced TBIs in childhood and individuals with two or more TBIs were excluded (n=651) from the analysis for two reasons: (1) because participants with a TBI were only asked about their age when the *most recent* TBI occurred, it would be impossible to establish temporality between ACEs and TBI if we did not know when the first TBI occurred and (2) because TBIs are a known risk factor for both subsequent TBIs and alcohol use and, therefore, it would be impossible to establish the direction of the association between TBI and binge drinking. Furthermore, the sample was restricted to those respondents who sustained their first TBI at age 18 or older to ensure temporality between ACEs and TBI (ie, the ACEs needed to occur before the TBI). The final analytic sample was n=6317. Patients or the public were not involved in the design, or conduct, or reporting, or dissemination plans of our research.

Measures

In years 2012–2016, participants were asked retrospectively about experiences of childhood adversity. The four questions included: before age 18, (1) did you live with anyone who was depressed, mentally ill, or suicidal? (yes or no); (2) did you live with anyone who was a problem drinker or alcoholic? (yes or no); (3) how often did a parent or adult in your home ever hit, beat, kick or physically harm you in any way? (never, once, or more than once) and (4) how much parental love and affection did you receive growing up? (A great deal, quite a lot, a little, none at all). Each ACE was coded as dichotomous (present or absent). For physical abuse, 'never' was coded as absent, and 'once or more than once' as present. We reverse coded parental love and affection to represent low parental warmth; respondents who reported that the amount of affection they received as a child was 'a little' and 'none at all' were coded as experiencing low parental warmth. We also created a cumulative score by summing the four items.

TBI was assessed retrospectively by asking participants 'Have you ever had a blow to the head or a similar type of head injury that was severe enough to require medical attention, or to lose consciousness or memory loss for a period of time?'

Binge drinking among participants 18 or older was assessed by asking participants to report the number of times they had consumed six or more drinks on one occasion during the past 30 days. We calculated mean level of binge drinking across all waves; data on binge drinking was collected in 1982–1985, 1988–1989, 1992, 1994, 2002 and every 2 years starting in 2006 until 2014. Of note, this definition of binge drinking differs from the Centers for Disease Control and Prevention's definition (four or more drinks for women and five or more drinks for men on one occasion); the implications of which are discussed in the Limitations section.[26]

Analysis

We conducted longitudinal mediation analysis to examine if binge drinking mediates the relationship between ACEs and TBIs sustained in adulthood. We estimated the mediation model five times: one with the cumulative ACE score and once for each ACE subtype. All models controlled for potentially confounding covariates—race/ethnicity, age, lifetime educational attainment and sex. This analysis was conducted using M*plus* V8.6 latent variable modelling software.[27] This software allowed us to estimate the entire mediation model (shown in figure 1) simultaneously. Bootstrapped standard errors were calculated for the indirect effect of ACEs on TBI risk because these effects do not have symmetric sampling distributions. We used the weighted least square mean and variance adjusted (WLSMV) estimator with a probit link function for the TBI outcome.

RESULTS

Table 1 displays the demographic characteristics of the sample. About thirty-four per cent (n=2125) of the sample reported experiencing at least one ACE; 5% reported living with a parent with depression, 12% reported experiencing physical abuse in childhood, 16% reported low parental warmth and 16% reported living with an adult with a drinking

problem. About 1% of the sample reported experiencing all four ACEs. Five per cent reported experiencing a TBI in adulthood (n=346).

Table 2 displays the results of the five mediation models. The 'b' path from lifetime binge drinking to TBI risk was statistically significant and positive for all five models. Because a probit model was used, coefficients are interpretable as the expected change in TBI risk in z-score units. The 'a' path from ACE to mean lifetime level of binge drinking was statistically significant for cumulative ACEs (b=0.02, 95% CI=0.0 to 0.05), familial alcoholism (b=0.06, 95% CI=0.01 to 0.11), and experience of physical abuse (b=0.08, 95% CI=0.02 to 0.14). The 'a' paths were not significant for familial mental illness or low warmth. In turn, the indirect effects (a × b) of ACEs on TBI were small but significant for cumulative ACEs, familial alcoholism and physical abuse (b=0.01, 95% CI=0.0 to 0.01; b=0.01, 95% CI=0.0 to 0.03; and b=0.02, 95% CI=0.0 to 0.03). The effects of lifetime binge drinking explained 6.7% of the total effect of cumulative ACEs on TBI risk, 3.2% of the total effect of familial alcoholism on TBI risk and 6.9% of the total effect of the experience of physical abuse on TBI risk. The direct effect of ACEs on TBI risk after accounting for binge drinking (ie, the c' paths) was uniformly larger than the indirect effects through binge drinking (see table 2).

In sum, these results indicate that lifetime binge drinking explains only a small part of the association between ACEs and TBI. Specifically, binge drinking partially explains the link between familial alcoholism and TBI and between experience of physical abuse and TBI.

DISCUSSION

Millions of Americans sustain a TBI every year in the USA. It is critically important to identify risk and protective factors for the injury in order to design, implement and effectively tailor prevention strategies. Previous research has established that exposure to ACEs increases one's risk of sustaining a TBI in adulthood,[5] but the mechanisms are not well understood. This study sought to determine whether lifetime binge drinking mediated the relationship between ACEs and TBI, in an effort to improve the design and implementation of TBI prevention strategies. The use of alcohol, and binge drinking more specifically, are common behaviours in the USA. A recent report found that during 2018, 16.6% of US adults reported binge drinking in the past 30 days.[26] Given the strong relationship between alcohol use, and particularly alcohol use disorder and alcohol intoxication, and TBI,[20] this puts millions of adults at increased risk of sustaining a TBI. In a nationally representative cohort of American adults, we found that both ACEs (in particular, the cumulative ACE score, family alcoholism and childhood physical abuse) and lifetime binge drinking were significantly associated with subsequent self-reported TBI, but that the lifetime binge drinking is not a strong mediator in the overall relationship between ACEs and TBI.

We found heterogeneity in the direct and mediated effects of some individual ACEs on TBI risk. Low parental warmth had no effect on TBI risk. Familial mental illness had the strongest direct effect on TBI incidence, but the indirect effect through binge drinking was null. On the other hand, childhood physical abuse and familial alcoholism had moderate overall effects on TBI incidence, but those effects were partially mediated by lifetime

binge drinking. There are several possible explanations for the connection between familial alcoholism, physical abuse, binge drinking and TBI. Parents who drink excessively have a higher risk of perpetrating physical abuse on their children,[28] making the co-occurrence of family alcoholism and physical violence in the family quite common. Additionally, having a family history of alcohol use and dependence has long been recognised as one of the best explanatory predictors of later heavy alcohol use and binge drinking.[29 30] Heavy alcohol use, particularly at the time of injury, is strongly associated with one's risk of sustaining a TBI.[16 20] Further, Martin and colleagues reported that boys with a family history of substance abuse have higher levels of aggressivity, inattention and impulsivity than boys without such a history.[31] These externalising behaviours may also put these individuals at a higher risk of sustaining a TBI in their lifetime. There is also some research to suggest that childhood victims of family violence have higher rates of intimate partner violence,[32] which is a common cause of TBI in adulthood.[33] Biological and social responses to physical abuse as a child may differ from other childhood adversities, explaining why alcohol use mediates the relationship between some, but not all, ACEs and TBI.[34] Our results highlight the importance of preventing excessive alcohol consumption, particularly binge drinking, given its strong association between TBI and multiple adverse outcomes. Addressing community-level factors, such as alcohol outlet density, has demonstrated positive impacts on alcohol use.[35] The Community Preventive Services Task Force also recommends healthcare providers use electronic screening and brief intervention to reduce excessive alcohol consumption and alcohol-related problems.[36]

There are several other potential theories for why exposure to ACEs may put one at increased risk for sustaining a TBI in adulthood. Children exposed to ACEs may experience changes in brain structure and functioning, which may increase vulnerability to later injury in adulthood via the disruptive toxic effects of a cumulative stress burden. [10] Risk-taking propensity, which has been found to mediate the relationship with ACEs and other negative outcomes[37] and is associated with TBI,[4] is deserving of further study. And finally, protective adult relationships may help *decrease* the risk of adverse health behaviours and outcomes, including TBI, after ACEs.[10 38] More research is needed to explore other social, biological and behavioural mechanisms that potentially mediate the pathways between ACEs and TBI, including brain development and functioning, socio-emotional functioning,[8] exposure to community or other forms of violence[15] and social and environmental conditions. Despite this, our results underscore the importance of integrating prevention and reduction of childhood adversity as part of comprehensive TBI prevention efforts.[5]

Limitations

There are several limitations to this study. First, the survey only asked respondents about TBIs they sustained that were severe enough to require medical attention, loss of consciousness or the experience of memory loss. However, most people who sustain a TBI do not lose consciousness[3] and do not seek medical care[39] and those who do have more severe injuries. Therefore, our results are biased toward more serious head injuries, and we are likely undercounting the prevalence of TBIs and limiting their association with ACEs and binge drinking. Second, we only included respondents who reported sustaining one TBI in adulthood. Individuals who experienced TBIs in childhood and individuals with two or

more TBIs were excluded from the study because participants with a lifetime history of TBI were only asked about their age when the *most recent* TBI occurred, so it was not possible to infer temporality of alcohol consumption and the first TBI. However, it is possible that individuals who experience multiple TBIs during childhood or adulthood may have specific risk profiles for ACEs and alcohol use. Third, this analysis included a limited set of four ACEs, all of which were self-reported. It is possible that other types of ACEs may have a different relationship to alcohol use and TBI. Additionally, recall of childhood adversities, especially in the early childhood years which are particularly influential to development, may also be limited. Fourth, the alcohol use measures in this study capture general patterns of alcohol use in adulthood, rather than any alcohol use or alcohol intoxication immediately prior to the reported TBI. This likely influences our ability to detect mediation effects of alcohol between ACEs and TBI and might underestimate alcohol's influence. Fifth, the NLSY79 did not collect data on binge drinking at each survey administration, and the definition of binge drinking used (having consumed six or more drinks on one occasion) is different than the standard binge drinking definition (four or more drinks for women and five or more drinks for men on one occasion).[26] This means the study may underestimate the prevalence of binge drinking in the study sample. A more precise measure of binge drinking, such as the standard definition, could potentially explain a larger proportion of the relationship between TBI and ACEs. Finally, all participants who participated in any wave were included in the analysis. Therefore, participants who had an unmeasured TBI after dropout were incorrectly assigned to the non-TBI group. To the extent to which this affected the findings, estimated between-group difference would be too small and significance tests would be conservative.

CONCLUSION

There is a strong relationship between exposure to ACEs and risk of sustaining TBI in adulthood. Our results indicate that while ACEs and adult TBI risk were significantly associated, lifetime binge drinking explains only a small part of the association. Future research could examine alternative patterns of alcohol use, as well as other social, biological and behavioural mechanisms along the pathway between ACEs and TBI. Additionally, researchers may choose to investigate whether the association between ACEs and TBIs varies by sociodemographic characteristics. Determining the mechanisms that connect ACEs to future TBI will allow public health practitioners to design and implement effective TBI prevention programmes for those at higher risk of injury due to ACE exposure.

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Data availability statement

Data are available in a public, open access repository. Data are publicly available.

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WHAT IS ALREADY KNOWN ON THIS TOPIC

- There is an association between adverse childhood experiences (ACEs) and traumatic brain injury (TBI).
- Both ACEs and TBI are also correlated with alcohol use.

WHAT THIS STUDY ADDS

- Binge drinking may partially mediate the relationship between ACEs and adult TBI risk.
- Pathways between ACEs, alcohol use and TBI may be specific to individual ACEs.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE, AND/OR POLICY

- Future research may focus on determining the mechanism connecting experience of ACEs to TBI, which would allow for more tailored TBI prevention efforts.

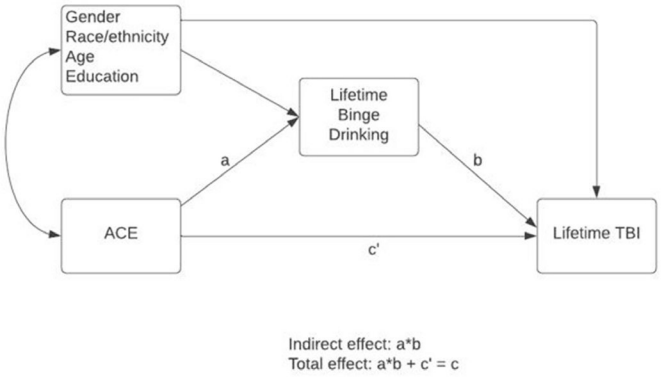

Figure 1.

Hypothesised direct (c′) and indirect (a × b) pathways between adverse childhood experiences (ACEs) and lifetime traumatic brain injury (TBI) via lifetime binge drinking.

Table 1

Demographic characteristics of 1979 Cohort of National Longitudinal Survey of Youth Sample (n=6317), USA, 2016

	Frequency	Per cent
Mean baseline age (range)	17.6 (14, 22)	
Sex		
Male	2919	46.2
Female	3398	53.8
Race/ethnicity		
Non-Hispanic, Non-Black	2964	46.9
Non-Hispanic Black	2122	33.6
Hispanic	1231	19.5
Education		
Less than high school	935	14.8
High school degree	2521	39.9
More than high school	2749	43.5
Number of ACEs		
0	4192	66.4
1	1383	21.9
2	493	7.8
3	197	3.1
4	52	0.8
ACE type		
Physical abuse	758	12.0
Low parental warmth	1010	16.0
Lived with a parent with depression	316	5.0
Lived with an adult with a drinking problem	1010	16.0
Ever TBI		
Yes	346	5.5
No	5971	94.5

ACE, adverse childhood experience; TBI, traumatic brain injury.

Table 2

Effects of binge drinking on the relationship between adverse childhood experiences (ACEs) and traumatic brain injury (TBI) (coefficient and 95% CIs), 1979 Cohort of National Longitudinal Survey of Youth Sample, USA, 2016

	Total effect of ACE on TBI (*c path*)	**Direct effect of ACE on binge drinking (*a path*)**	**Direct effect of binge drinking on TBI (*b path*)**	**Direct effect of ACE on TBI (*c' path*)**	**Indirect (mediated) effect of ACE on TBI via binge drinking (*a*b*)**	**Per cent of total effect mediated by binge drinking**
Cumulative ACEs	**0.15 (0.08 to 0.21)**	**0.02 (0 to 0.05)**	**0.23 (0.16 to 0.30)**	**0.14 (0.08 to 0.20)**	**0.01 (0 to 0.01)**	**6.7**
Familial mental illness	**0.48 (0.25 to 0.69)**	−0.04 (−0.11 to 0.04)	**0.23 (0.17 to 0.30)**	**0.49 (0.25 to 0.70)**	−0.01 (−0.03 to 0.01)	0
Familial alcoholism	**0.31 (0.16 to 0.44)**	**0.06 (0.01 to 0.11)**	**0.23 (0.16 to 0.30)**	**0.10 (0.15 to 0.41)**	**0.01 (0 to 0.03)**	**3.2**
Low warmth	−0.01 (−0.18 to 0.14)	0.01 (−0.04 to 0.06)	**0.23 (0.16 to 0.30)**	−0.02 (−0.19 to 0.14)	0.00 (−0.01 to 0.01)	0
Physical abuse	**0.29 (0.13 to 0.44)**	**0.08 (0.02 to 0.14)**	**0.23 (0.16 to 0.30)**	**0.27 (0.11 to 0.42)**	**0.02 (0 to 0.03)**	**6.9**

All models controlled for gender, age, race/ethnicity and education. Bolded cells are statistically significant with p<0.05.