

Trauma's distinctive and combined effects on subsequent substance use, mental health, and neurocognitive functioning with the NCANDA sample

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ABSTRACT

Purpose: Traumatic brain injury (TBI) and potentially traumatic events (PTEs) contribute to increased substance use, mental health issues, and cognitive impairments. However, there's not enough research on how TBI and PTEs combined impact mental health, substance use, and neurocognition.

Methods: This study leverages a subset of The National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA) multi-site dataset with 551 adolescents to assess the combined and distinctive impacts of TBI, PTEs, and TBI+PTEs (prior to age 18) on substance use, mental health, and neurocognitive outcomes at age 18.

Results: TBI, PTEs, and TBI+PTEs predicted greater lifetime substance use and past-year alcohol and cannabis use. PTEs predicted greater internalizing symptoms, while TBI+PTEs predicted greater externalizing symptoms. Varying effects on neurocognitive outcomes included PTEs influencing attention accuracy and TBI+PTEs predicting faster speed in emotion tasks. PTEs predicted greater accuracy in abstraction-related tasks. Associations with working memory were not detected.

Conclusion: This exploratory study contributes to the growing literature on the complex interplay between TBI, PTEs, and adolescent mental health, substance use, and neurocognition. The developmental implications of trauma via TBIs and/or PTEs during adolescence are considerable and worthy of further investigation.

1. Introduction

Traumatic brain injury (TBI) and traumatic potentially traumatic events (PTEs) frequently co-occur, with 1 in 5 youth who experience a TBI endorsing 4+ instances of PTEs (Bright and Thompson, 2018; Jackson et al., 2022). Trauma exposure, whether it be TBI or PTEs, during adolescence can impact brain development and functioning, which increases the risk of lifelong consequences, including psychiatric disorder diagnosis up to 30 years after TBI exposure (Fleminger, 2008; Koponen et al., 2002) and psychopathology following PTEs (McLaughlin et al., 2020; Patel and Oremus, 2022).

Alongside TBI and PTE exposure, the onset of mental health problems, initiation of regular substance use, and neurocognitive

development typically occur through adolescence and emerging adulthood (Uhlhaas et al., 2023). TBI and PTE exposure can impact mental health, substance use, and neurocognition. The overlap in these exposures (TBI and PTEs) and outcomes (mental health, substance use, and neurocognition) creates developmental junctions, highlighting a critical need to examine the distinctive and combined effects of TBI and PTEs on mental health, substance use, and neurocognitive development at the culmination of adolescence. As we elaborate subsequently, TBI and PTEs have been shown to impact mental health, substance use, and neurocognition independently during adolescence. However, less is known about their combined impacts on these outcomes.

TBI exposure has been extensively and independently linked to subsequent mental health problems (Alway et al., 2016; Max et al.,

Abbreviations: TBI, traumatic brain injury; PTEs, potentially traumatic events; NCANDA, National Consortium on Alcohol and Neurodevelopment in Adolescence.

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2013; Perry et al., 2016; Whelan-Goodinson et al., 2009; Zgaljardic et al., 2015), development of substance use disorders (McHugo et al., 2017; Pagulayan et al., 2016; West, 2011), and greater neurocognitive difficulties (Babikian and Asarnow, 2009; Goh et al., 2021). The extent to which impairment is observed following a TBI depends on the specificity and severity of the injury. Specificity concerns the extent of the damage (i.e., limited or broad), while severity determines the duration of challenges (i.e., acute versus chronic). TBIs can lead to structural damage within the brain, impacting functional outcomes. Broad and severe TBIs tend to have impacts that last longer than more specific and less severe TBIs.

Similarly, PTEs have been extensively and independently linked to mental health problems (Gardner et al., 2019; Kessler et al., 2010; R.-Mercier et al., 2018), substance use disorders (Puetz and McCrory, 2015; Sebalo et al., 2023), and neurocognitive difficulties (Hawkins et al., 2021; Kavanaugh et al., 2017; Lund et al., 2020; Puetz and McCrory, 2015; R.-Mercier et al., 2018). Many PTEs do not result in direct structural brain damage (e.g., emotional neglect will not lesion the brain) but can help rewire brain circuitry, leading to challenges in mental health, substance use, and neurocognition.

For similar impacts, TBIs and PTEs may lead to changes on a global brain systems level, contributing to poor mental health, increased substance use, and neurocognitive difficulties. However, they may also differentially impact mental health, substance use, and neurocognition. The extent to which TBI and PTEs operate on similar or disparate mechanisms is unknown as there is a dearth of evidence comparing combined (TBI+PTEs) and distinctive effects (TBI or PTEs) on these outcomes.

Determining the combined and distinctive effects of TBIs and PTEs on mental health, substance use, and neurocognition is further complicated by trait-level factors such as impulsivity. Impulsivity has been associated with TBI (Dimoska-Di Marco et al., 2011; Fusi et al., 2023; Rochat et al., 2013), potentially traumatic events (Lovallo, 2013), mental health (Berg et al., 2015), neurocognition (Nigg, 2017; Wilhelm et al., 2016), and substance use (Lee et al., 2019). More impulsive adolescents may be more likely to get themselves into dangerous situations that result in TBIs and PTEs. Impulsivity has also been linked to mental health, substance use, and neurocognition. As such, controlling for the bias that impulsivity may create an association between TBIs/PTEs and these outcomes is important.

In summary, the developmental trajectory of the impact of TBI and PTEs has not been well characterized. The National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA; (Brown et al., 2015) is a multi-site study of adolescents and young adults followed longitudinally, which makes it an ideal data source for the central aim of this study.

The present study offers an exploratory examination of the distinctive and combined effects of childhood TBI and PTEs on mental health, substance use, and neurocognitive functioning among adolescents at age 18 (see Fig. 1 for a conceptual model of the research question). Here, we operationalize TBI as instances of head trauma with and without a loss of consciousness and PTEs as the endorsement of criterion A traumatic events for posttraumatic stress disorder using a validated clinical interview. We hypothesize experiencing both TBI and PTEs, relative to neither or one alone, before age 18 will contribute to increased substance use, worse mental health, and impaired neurocognitive

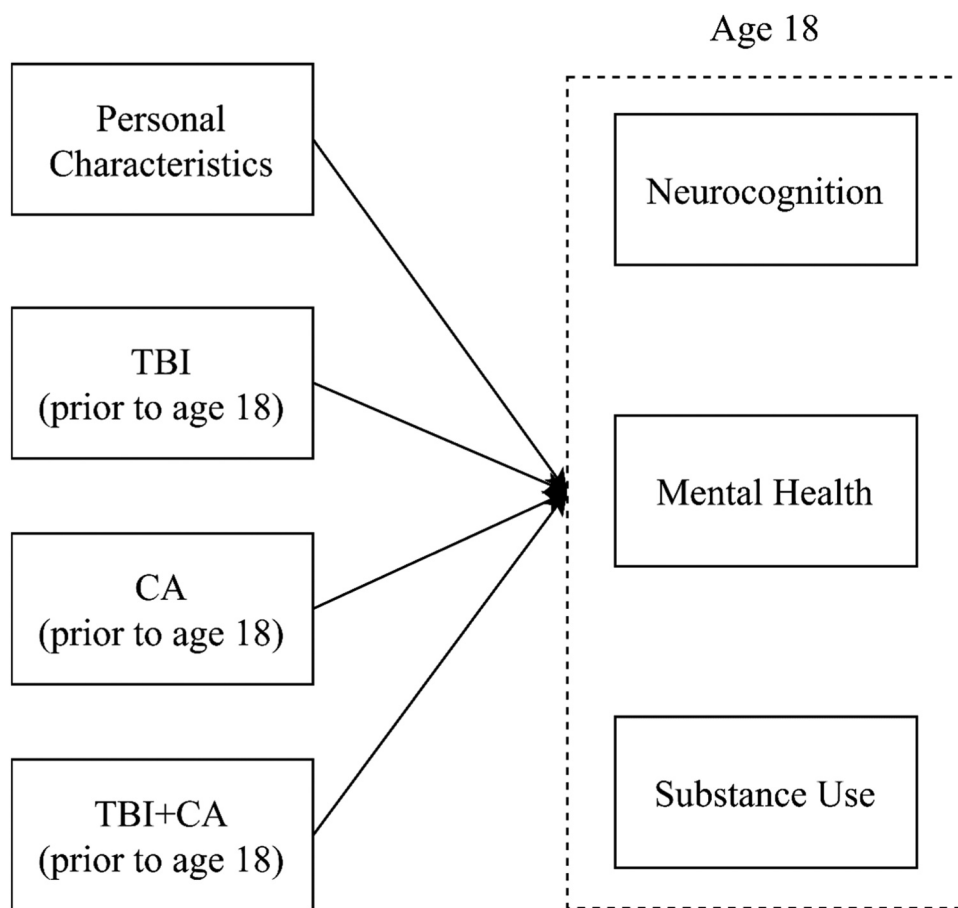


Fig. 1. Conceptual Model Depicting the Research Question of the Study. This study will examine the impact of lifetime traumatic brain injury and potentially traumatic events exposure on substance use, mental health, and neurocognition at age 18. TBI = traumatic brain injury; PTEs = potentially traumatic events. Personal Characteristics include sex at birth, socioeconomic status, and impulsivity.

functioning at age 18.

2. Methods

2.1. Participants

Participant data were drawn from the National Consortium on Alcohol and Neurodevelopment in Adolescence (NCANDA), a study that recruited youth between the ages of 12 and 21 and assessed them annually for 7 years (including baseline), using an accelerated cohort design (Brown et al., 2015). Refer to Brown et al. (2015) for further recruitment and demographic details. A sample of 831 youth were recruited through school mailers, community fliers and advertisements, and announcements at local universities at five sites: University of California, San Diego, SRI International, Duke University Medical Center, University of Pittsburgh, and Oregon Health & Science University.

For the current study, we draw upon data from all participants until 18 years old. As such, we exclude participants who started the study at age 18 or older ($n = 276$). Given some participants completed two visits while they were 18 years old ($n = 22$), the visit closest to the average age of participants in our study ($M = 18.49$, $SD = 0.39$) was used as their age 18 visit. Thus, we have a sample of 555 adolescents belonging to 464 families. Of the included sample in the current study, 52 % were female (based on sex at birth), 77 % identified as White, 12 % identified as Black/African American, 7 % as Asian/Pacific Islander, and 5 % as biracial. Moreover, 11 % identified as Hispanic/Latinx.

2.2. Procedure

A standardized protocol was followed at every site in which data was first collected at the baseline visit, and subsequent follow-up data were collected in annual appointments for up to 7 years (Brown et al., 2015). Individuals completed self-reports of behavior, psychiatric symptoms, substance use, and a comprehensive battery of neuropsychological assessments. Refer to Brown et al. (2015) for further procedural details. To enhance the accuracy of self-report, youth were assured that their information would remain confidential and would not be revealed to parents except in the case of serious risk to self or others (e.g., suicidal/homicidal ideation or child abuse). Each site provided independent IRB approval with parent approval and assent for youth participants under age 18 and participant consent for those over age 18. As of 20th December 2022, ethics approval was centralized to the UCSD site (#120915).

2.3. Measures

The data were part of the public data release NCANDA_PUBLIC_7Y_REDCAP_V02 (Pohl et al., 2022), distributed according to the NCANDA Data Distribution agreement (<https://www.niaaa.nih.gov/ncanda-data-distribution-agreement>). Demographic data included age, sex at birth, education, and household income. Brief descriptions of each measure are provided below, for more details see Brown et al. (2015).

Traumatic Brain Injury. TBI was assessed using the Ohio State TBI Inventory (adapted from Corrigan & Bogner, 2007). The measure assesses an individual's self-reported lifetime history of TBI. Individuals who endorsed a head injury regardless of daze or loss of consciousness were considered to have experienced a TBI before age 18.

Potentially Traumatic Events. PTEs were assessed using the Semi-structured Assessment for the Genetics of Alcoholism (SSAGA), a semi-structured clinical interview that assesses mental disorders according to DSM-IV (baseline SSAGA) and DSM-V (follow-up years 1-7) criteria (American Psychiatric Association, 2013, 1994; Buchholz et al., 1994). The lifetime PTSD section of the SSAGA was used to determine whether participants experienced childhood trauma based on their endorsement of Criterion A for PTSD before age 18. Both retrospective endorsement of

childhood trauma and reporting trauma as an adolescent qualified as childhood trauma in the current study. The SSAGA was administered annually to all participants during the first four years of the study and at participant ages 24, 27, 30, and 33 for the remaining timepoints included in the study. PTE exposure was coded dichotomously (0 = no, 5 = yes) for each year the SSAGA was administered. Participants who met criteria for PTEs before age 18 were considered to have experienced PTEs.

Mental Health. Mental health symptoms were assessed using the Achenbach rating system (Achenbach and Rescorla, 2003, 2001). The Achenbach rating system is a well-validated and widely used self-report measure of problem behaviors among adolescents and adults. During adolescence, participants completed the Youth Self-Report (YSR) form; during young adulthood, they completed the corresponding Adult Self-Report (ASR) form. Participants responded to how applicable a series of statements were to them using a Likert scale ("0 = Not True" to "2 = Very True or Often True"). Raw scores from the ASR at age 18 were used for analysis.

Substance Use. A wide range of substance use metrics were obtained using the Customary Drinking and Drug Use Record (CDDR; (Brown et al., 1998)). The CDDR is a well-validated and widely used self-report measure of substance use among adolescents and adults. From the CDDR, we obtained metrics of: lifetime use any substance, including alcohol (dichotomous score for any use in lifetime); indiscriminate past year use of any substance, including alcohol, (dichotomous score for any use in past year); frequency of alcohol use (days of use in past year); frequency of cannabis use (days of use in past year); frequency of nicotine use (number of cigarettes used in past year). Nicotine use frequency was dichotomized into whether youth used cigarettes in the past year (score of 1) or did not (score of 0) as prevalence of use was low ($n = 75$), but use varied quite a bit, impairing model fit. Data obtained at age 18 years old was used for analyses.

Neurocognition. Neurocognitive functioning in eight functional domains was assessed using performance on a common web-based neuropsychological battery (PennCNP; <https://webcnp.med.upenn.edu/>). The following domains and respective tasks assessing those domains were used as outcomes: (1) attention, assessed by Continuous Performance Test - Number Letter Version; (2) abstraction, assessed by Conditional Exclusion Task, Matrix Analysis Test, and Logical Reasoning; (3) emotion, assessed by Emotion Recognition Test, and Measured Emotion Differentiation; and (4) working memory, assessed by the Short Fractal N-Back Test-2 Back Version. General ability speed and accuracy scores (assessed by the Vocabulary Test, WRAT-4 Math Calculations, and WRAT-4 Word Reading) were included as covariates in all models with neurocognition outcomes. Only neurocognition measures assessed at age 18 years old were extracted for all participants.

2.4. Analytic strategy

All analyses were conducted using R v2023.06.1+524 (R Core Team, 2022). We created four binary indicator variables based on TBI and PTE endorsement before age 18. The four variables corresponded to no exposure to TBI and PTEs (neither group), exposure to TBIs only (TBI only), exposure to PTEs only (PTEs only), and exposure to both (TBI+PTEs). Exposure to neither TBI nor PTEs was used as the reference in all models. This stratification and using no exposure as a reference in all models allowed us to examine the combined and distinctive effects of TBI and PTEs on outcomes of mental health, substance use, and neurocognition. Models with an interaction term (TBI x PTEs) were fitted to assess for synergistic effects of TBI and PTEs. There was only one statistically significant interaction effect predicting lifetime indiscriminate substance use ($b = 0.40$, $SE = 0.17$, $z = 2.31$, $p = .02$). Henceforth, we refer to TBI+PTEs as a combined effect rather than synergistic. All outcomes were assessed at the age of 18. All the independent variables in our sample are equivalent except for sex at birth and household income, which were included in our models as covariates alongside impulsivity.

Given all outcomes were assessed at 18 years old, age was not included as a covariate. See Table S1 for zero-order correlations between all variables of interest.

A series of linear regressions were fit to mental health, substance use, and neurocognition outcomes with our four binary indicators of exposure type as predictors for a total of 21 models. Tobit and logistic regressions were fit to models with residual distributions that deviated greatly from normality to improve model fit (specifics are listed below).

All outcomes were assessed at age 18 and the 4-level indicator variable for TBI and PTE exposure were used in analyses. For mental health outcomes, two linear regressions were fit for internalizing and externalizing symptoms, respectively. For substance use outcomes, four negative binomial regressions were fit, and a logistic regression for past year nicotine use. For neurocognition outcomes, regressions were fit for speed and accuracy scores on abstraction, attention (Tobit regression for accuracy on Continuous Performance Task), emotion, and working

Table 1
Sample Characteristics.

| | Total Sample (N = 555) | TBI Only Group (n = 84) | PTEs Only Group (n = 171) | TBI PTEs Group (n = 143) | Neither Group (n = 157) |
|--|---------------------------|----------------------------|------------------------------|-----------------------------|----------------------------|
| Demographics | | | | | |
| Age | 18.48 (0.27) | 18.48 (0.25) | 18.48 (0.28) | 18.50 (0.27) | 18.48 (0.30) |
| % Female | 52 % | 42 % | 61 % | 49 % | 49 % |
| Race | | | | | |
| White | 77 % | 80 % | 67 % | 77 % | 85 % |
| Black/African American | 12 % | 3 % | 23 % | 9 % | 6 % |
| Asian/Pacific Islander | 7 % | 5 % | 6 % | 9 % | 6 % |
| Biracial | 5 % | 12 % | 4 % | 5 % | 3 % |
| Household Income | \$100,000 - \$199,999 | \$100,000 - \$199,999 | \$75,000 - \$99,999 | \$100,000 - \$199,999 | \$100,000 - \$199,999 |
| High school diploma or equivalency (GED) | 77 % | 77 % | 77 % | 76 % | 75 % |
| Employment Status: Student | 77 % | 73 % | 77 % | 78 % | 77 % |
| Impulsivity | 1.94 (0.37) | 1.94 (0.38) | 1.92 (0.38) | 2.01 (0.37) | 1.89 (0.36) |
| Mental Health (n = 502) | | | | | |
| | n = 502 | n = 78 | n = 156 | n = 128 | n = 140 |
| Internalizing Symptoms | 10.38 (9.21) | 10.15 (8.82) | 11.81 (10.42) | 11.45 (10.06) | 7.93 (6.33) |
| Externalizing Symptoms | 7.33 (6.30) | 7.58 (6.75) | 6.88 (5.88) | 9.24 (7.19) | 5.94 (5.14) |
| Substance Use (n = 555) | | | | | |
| Lifetime Use | 1.73 (1.77) | 2.11 (2.34) | 1.56 (1.45) | 2.10 (1.79) | 1.38 (1.64) |
| Past Year Use | 0.28 (0.56) | 0.26 (0.54) | 0.29 (0.53) | 0.37 (0.68) | 0.21 (0.47) |
| Alcohol Use Frequency | 11.37 (21.86) | 18.00 (33.08) | 7.89 (16.36) | 15.14 (24.89) | 8.17 (14.12) |
| Cannabis Use Frequency | 20.96 (62.83) | 27.20 (59.94) | 18.52 (59.63) | 27.76 (75.71) | 14.08 (53.81) |
| Nicotine Use Frequency | 30.24 (247.90) | 92.57 (428.42) | 10.14 (68.16) | 43.12 (340.52) | 7.06 (82.19) |
| Neurocognition (n = 327 501) | | | | | |
| Attention | | | | | |
| | n = 501 | n = 77 | n = 153 | n = 130 | n = 141 |
| CPT-NL Speed | 528.96 (48.92) | 521.90 (50.74) | 521.59 (51.15) | 518.17 (47.99) | 515.21 (46.46) |
| CPT-NL Accuracy | 57.57 (4.75) | 57.31 (5.96) | 57.67 (3.62) | 58.00 (3.46) | 57.21 (5.97) |
| Abstraction | | | | | |
| | n = 501 | n = 77 | n = 153 | n = 130 | n = 141 |
| CET Speed | 1797.08 (647.10) | 1758.81 (574.53) | 1885.57 (864.57) | 1748.25 (510.15) | 1766.96 (502.83) |
| CET Accuracy | 36.71 (8.64) | 35.77 (6.96) | 36.91 (9.06) | 36.21 (7.44) | 37.49 (9.94) |
| MAT Speed | 9935.25 (5211.04) | 10667.76 (5803.94) | 9512.55 (4767.99) | 9790.60 (5666.18) | 10127.26 (4887.04) |
| MAT Accuracy | 17.33 (4.62) | 18.06 (4.40) | 16.95 (4.65) | 17.22 (4.48) | 17.43 (4.83) |
| | n = 327 | n = 44 | n = 97 | n = 73 | n = 113 |
| LRT Speed | 6646.33 (2608.82) | 7472.74 (3098.05) | 6626.94 (2651.38) | 6354.15 (2334.12) | 6529.92 (2501.88) |
| LRT Accuracy | 20.20 (4.29) | 20.86 (3.67) | 19.43 (5.17) | 19.59 (4.36) | 20.99 (3.41) |
| Emotion | | | | | |
| | n = 501 | n = 77 | n = 153 | n = 130 | n = 141 |
| ERT Speed | 1747.37 (289.31) | 1771.16 (298.00) | 1769.23 (335.42) | 1680.87 (2228.88) | 1771.97 (273.40) |
| ERT Accuracy | 36.95 (2.16) | 37.05 (2.13) | 36.88 (2.43) | 37.08 (2.13) | 36.84 (1.91) |
| | n = 497 | n = 76 | n = 152 | n = 130 | n = 139 |
| MED Speed | 2327.78 (598.75) | 2322.29 (649.81) | 2377.77 (665.59) | 2239.46 (466.18) | 2358.73 (600.06) |
| MED Accuracy | 28.76 (3.12) | 28.92 (3.33) | 28.81 (3.36) | 28.78 (2.74) | 28.59 (3.09) |
| Working Memory | | | | | |
| | n = 501 | n = 77 | n = 152 | n = 130 | n = 141 |
| SFNB 2B Speed | 549.32 (82.09) | 547.08 (80.80) | 554.76 (83.81) | 546.53 (81.96) | 547.25 (81.64) |
| SFNB 2B Accuracy | 28.40 (2.84) | 28.79 (1.66) | 28.07 (4.24) | 28.26 (2.41) | 28.65 (1.47) |

Note. All values presented are means and standard deviations except for the percentages of the sample for demographic characteristics and the median household income range. CPT-NL = Continuous Performance Test - Number Letter Version; CET = Conditional Exclusion Task; MAT = Matrix Analysis Test; LRT = Logical Reasoning; ERT = Emotion Recognition Test; MED = Measured Emotion Differentiation; SFNB-2B = Short Fractal N-Back Test-2 Back.

memory (Tobit regression for accuracy on Short Fractal N-back Task). Here, models included general ability speed and accuracy scores as covariates to account for general functioning irrespective of exposure to TBI and PTEs. Results for models fitted without general ability as a covariate are presented in [Supplemental Material](#). For example, it is possible that someone without TBI or PTEs exposure could have poor performance on general ability tasks. Alternatively, TBI and PTEs could have led to poor general ability performance and not including it as a covariate would bias our estimates of the effect of TBI and PTEs on specific neurocognitive outcomes. Since we had 91 sibling pairs in our sample, a random effect for families capturing whether youth belonged to the same family (also indicating sibling status) was included in each model to account for the non-independence of observations. As such, restricted maximum likelihood estimation (REML) was used for all models.

Statistical significance was evaluated using a neo-Fisherian framework (Hurlbert et al., 2019; Hurlbert and Lombardi, 2009), which considers p-values as providing a continuum of evidence (Amrhein et al., 2019) in an exploratory setting. The interpretation of the results is couched in recognition that the study is exploratory rather than confirmatory (Wagenmakers et al., 2012). As such, we do not correct for multiple comparisons (Rothman, 1990; Sullivan and Feinn, 2021). Identified patterns should be considered suggestive and used to guide confirmatory studies (Wagenmakers et al., 2012).

3. Results

3.1. Mental health

The PTEs group predicted greater internalizing symptoms ($b = 2.70, p = .02$), and the TBI+PTEs group predicted greater externalizing symptoms ($b = 2.17, p = .002$). Associations between the TBI group ($b = 1.22, p = .34$) and TBI+PTEs group ($b = 1.89, p = .10$) and internalizing

symptoms were not detected. The TBI group ($b = 1.56, p = .05$) and PTEs group ($b = 0.73, p = .29$) did not predict externalizing symptoms. For full results, refer to [Table 2](#).

3.2. Substance use

The TBI group ($b = 0.46, p = .001$), the PTEs group ($b = 0.36, p = .003$) and the TBI+PTEs group ($b = 0.42, p = .001$) predicted more lifetime substance use, greater past-year cannabis use frequency (TBI: $b = 1.60, p = .001$; PTEs: $b = 1.44, p = .001$; TBI+PTEs: $b = 1.19, p = .003$) and greater past-year alcohol use frequency (TBI: $b = 0.83, p = .001$; PTEs: $b = 0.50, p = .04$; TBI+PTEs: $b = 0.99, p = .001$). However, none of them predicted past year substance use (TBI: $b = 0.18, p = .57$; PTEs: $b = 0.40, p = .16$; TBI+PTEs: $b = 0.43, p = .12$). Only the TBI group increased the odds for past year nicotine use ($OR = 2.92, p = .03$). The PTEs and TBI+PTEs groups did not predict the odds for past year nicotine use (PTEs: $OR = 1.00, p = .99$; TBI+PTEs: $OR = 2.07, p = .10$). For full results, refer to [Table 2](#).

3.3. Neurocognition

Attention. For the attention domain, there was one task (Continuous Performance Task - Number Letter Version) with a speed and accuracy score. Here, the PTEs group predicted accuracy ($b = 1.82, p = .02$), but the TBI group ($b = 0.03, p = .97$) or the TBI+PTEs group ($b = 1.18, p = .12$) did not significantly predict accuracy. The TBI group ($b = 4.97, p = .53$), the PTEs group ($b = 2.36, p = .73$), and the TBI+PTEs group ($b = 1.44, p = .83$) did not significantly predict speed. For full results, refer to [Table 2](#).

Abstraction. For the abstraction domain, there were three tasks, and each had a speed and accuracy score. For the Conditional Exclusion Task, neither the TBI, PTEs, or TBI+PTEs groups predicted accuracy (TBI: $b = 1.19, p = .38$; PTEs: $b = 2.20, p = .06$; TBI+PTEs: $b = 2.18,$

Table 2
Results of Multiple Linear and Tobit Regressions.

| | TBI | | | PTEs | | | TBI PTEs | | |
|------------------------|-----------------|-------|------|-----------------|-------|------|-----------------|-------|------|
| | Est. (SE) | t / z | p | Est. (SE) | t / z | p | Est. (SE) | t / z | p |
| Mental Health | | | | | | | | | |
| Internalizing Symptoms | 1.22 (1.28) | .95 | .34 | 2.70 (1.13) | 2.40 | .02 | 1.89 (1.15) | 1.64 | .10 |
| Externalizing Symptoms | 1.56 (.87) | 2.00 | .05 | .73 (.68) | 1.07 | .29 | 2.17 (.70) | 3.11 | .002 |
| Substance Use | | | | | | | | | |
| Lifetime Use | .46 (.13) | 3.45 | .001 | .36 (.12) | 2.93 | .003 | .42 (.12) | 3.49 | .001 |
| Past Year Use | .18 (.32) | .57 | .57 | .40 (.28) | 1.42 | .16 | .43 (.27) | 1.56 | .12 |
| Alcohol Use Frequency | .83 (.25) | 3.30 | .001 | .50 (.24) | 2.05 | .04 | .99 (.24) | 4.15 | .001 |
| Cannabis Use Frequency | 1.60 (.44) | 3.67 | .001 | 1.44 (.40) | 3.62 | .001 | 1.19 (.40) | 3.00 | .003 |
| Nicotine Use* | 2.92 (1.41) | 2.22 | .03 | 1.00 (.49) | .01 | .99 | 2.07 (.90) | 1.67 | .10 |
| Neurocognition | | | | | | | | | |
| Attention | | | | | | | | | |
| CPT-NL Speed | 4.97 (7.82) | .64 | .53 | 2.36 (6.81) | .35 | .73 | 1.44 (6.80) | .21 | .83 |
| CPT-NL Accuracy | .03 (.88) | .03 | .97 | 1.82 (.77) | 2.37 | .02 | 1.18 (.76) | 1.55 | .12 |
| Abstraction | | | | | | | | | |
| CET Speed | 74.26 (94.75) | .78 | .43 | 31.22 (82.96) | .38 | .71 | 129.29 (83.44) | 1.55 | .12 |
| CET Accuracy | 1.19 (1.36) | .88 | .38 | 2.20 (1.18) | 1.86 | .06 | 2.18 (1.18) | 1.85 | .07 |
| MAT Speed | 696.61 (728.21) | .96 | .34 | 67.53 (633.72) | .11 | .92 | 748.02 (633.39) | 1.18 | .24 |
| MAT Accuracy | .38 (.59) | .65 | .51 | 1.08 (.51) | 2.10 | .04 | .22 (.51) | .44 | .66 |
| LRT Speed | 282.25 (467.75) | .60 | .55 | 179.67 (399.53) | .45 | .65 | 496.13 (408.26) | 1.22 | .23 |
| LRT Accuracy | .19 (.66) | .29 | .77 | .07 (.56) | .12 | .90 | .12 (.57) | .22 | .83 |
| Emotion | | | | | | | | | |
| ERT Speed | 27.53 (41.24) | .67 | .50 | 53.32 (35.93) | 1.48 | .14 | 116.10 (35.94) | 3.23 | .001 |
| ERT Accuracy | .27 (.33) | .83 | .41 | .56 (.28) | 1.98 | .05 | .50 (.28) | 1.78 | .08 |
| MED Speed | 124.88 (75.06) | 1.51 | .13 | 83.36 (72.06) | 1.16 | .25 | 196.68 (72.02) | 2.73 | .007 |
| MED Accuracy | .19 (.44) | .43 | .67 | .42 (.38) | 1.11 | .27 | .44 (.38) | 1.15 | .25 |
| Working Memory | | | | | | | | | |
| SFNB 2B Speed | 7.36 (13.06) | .56 | .57 | 5.01 (11.37) | .44 | .66 | 8.28 (11.36) | .73 | .47 |
| SFNB 2B Accuracy | .40 (.64) | .62 | .53 | .26 (.56) | .47 | .64 | .01 (.55) | .01 | .99 |

Note. CPT-NL = Continuous Performance Test - Number Letter Version; CET = Conditional Exclusion Task; MAT = Matrix Analysis Test; LRT = Logical Reasoning; ERT = Emotion Recognition Test; MED = Measured Emotion Differentiation; SFNB-2B = Short Fractal N-Back Test-2 Back.

* odds ratio and standard error from a logistic regression model are provided

$p = .07$), or speed (TBI: $b = 74.26, p = .43$; PTEs: $b = 31.22, p = .81$; TBI+PTEs: $129.29, p = .12$). For the Matrix Analysis Test, neither the TBI or TBI+PTEs groups predicted accuracy (TBI: $b = -0.38, p = .51$; TBI+PTEs: $0.22, p = .66$), but the PTEs group did ($b = 1.08, p = .04$). Neither the TBI, PTEs, or TBI+PTEs groups predicted speed (TBI: $b = 696.61, p = .34$; PTEs: $b = 67.53, p = .92$; TBI+PTEs: $b = 748.02, p = .24$). Lastly for the Logical Reasoning Task, neither the TBI, PTEs, or TBI+PTEs groups predicted accuracy (TBI: $b = 0.19, p = .77$; PTEs: $b = 0.07, p = .90$; TBI+PTEs: $b = 0.12, p = .83$) or speed (TBI: $b = 282.25, p = .55$; PTEs: $b = 179.67, p = .65$; TBI+PTEs: $b = 496.13, p = .23$). For full results, refer to [Table 2](#).

Emotion. For the emotion domain, there were two tasks, and each had a speed and accuracy score. For the Emotion Recognition Test, the PTEs group did not predict accuracy ($b = 0.56, p = .05$), but the TBI+PTEs group significantly predicted faster speed on the task ($b = 116.10, p = .001$). For the Measured Emotion Differentiation task, neither the TBI, PTEs, or TBI+PTEs groups predicted accuracy (TBI: $b = 0.19, p = .67$; PTEs: $b = 0.42, p = .27$; TBI+PTEs: $b = 0.44, p = .25$) but the TBI+PTEs group significantly predicted faster speed on the task ($b = 196.68, p = .007$). For full results, refer to [Table 2](#).

Working Memory. For the working memory domain, one task was used (Short Fractal N-Back Test-2 Back Version) which had a speed and accuracy score. Neither the TBI, PTEs, or TBI+PTEs groups predicted speed (TBI: $b = 7.36, p = .57$; PTEs: $b = 5.01, p = .66$; TBI+PTEs: $b = 8.28, p = .47$) or accuracy (TBI: $b = 0.40, p = .53$; PTEs: $b = 0.26, p = .64$; TBI+PTEs: $b = 0.01, p = .99$). For full results, refer to [Table 2](#).

4. Discussion

In this study, the combined and distinctive impacts of TBI and PTEs were examined on mental health, substance use, and neurocognition outcomes among a large sample of adolescents up to age 18. The current exploratory findings shed light on the nuanced relationships between these traumatic exposures and various domains of functioning during late adolescence/emerging adulthood. Specifically, TBI, PTEs, and TBI+PTEs predicted greater lifetime substance use, including alcohol and cannabis use. Differences were also found for internalizing and externalizing symptoms and neurocognitive outcomes, which are discussed as follows.

Consistent with previous research, PTEs emerged as a predictor of internalizing symptoms, aligning with the well-established link between PTEs and subsequent psychological distress ([Gardner et al., 2019](#); [Kessler et al., 2010](#); [Teicher et al., 2006](#)). Interestingly, the combination of TBI and PTEs (TBI+PTEs) specifically predicted externalizing symptoms, emphasizing the potential combined effects of these two risk factors on behavioral outcomes during adolescence ([Emery et al., 2016](#); [Jackson et al., 2022](#)). However, the same pattern was not observed for internalizing symptoms. With TBI, there are strong associations with externalizing symptoms, given TBI's associations with impulsivity ([Fusi et al., 2023](#); [Lovallo, 2013](#); [Rochat et al., 2013](#)). Within the current models, impulsivity was covaried and the combination of TBI+PTEs predicted greater externalizing symptom severity but not internalizing symptoms. It is likely that individuals exposed to TBI+PTEs have a greater risk for externalizing symptoms rather than the prototypical internalizing symptom presentation following PTEs. Greater externalizing symptoms as a function of TBI+PTEs could be a result of shared mechanisms of action between TBI and PTEs whereby global disruption of brain functioning is leading to more externalizing behaviors rather than exposure to TBI and PTEs alone. Whereas internalizing symptoms are more a function of PTEs than TBI, indicating differential mechanisms of action for internalizing symptoms whereby specificity and severity of TBI may need to overlap with functional changes due to PTEs associated with internalizing problems.

The current substance use findings highlight the distinct roles of TBI and PTEs. Nicotine use was the only substance with a positive association with TBI but not PTEs or their combination, suggesting that TBI and

nicotine use may be specifically related through a TBI-specific mechanism indicative of TBI's impact on decision-making and reward processing. Lifetime substance use, past-year alcohol use, and past-year cannabis use were all associated with TBI alone, PTEs alone, and TBI+PTEs. As indicated by coefficient magnitudes, on average, TBIs confer the greatest risk for substance use, whereas PTEs only confer the lowest risk, and TBI+PTEs fall somewhere in between. These results suggest that there is a differential experience and mechanism following TBI and PTE exposure on substance use outcomes whereby PTEs are potentially mitigating some of the risks from TBI exposure. One potential reason could include types of PTEs experienced by youth directly influencing expectancies ([Kosted et al., 2023](#); [Lavigne et al., 2017](#); [Weil et al., 2018](#)). For example, youth experiencing physical or emotional abuse from a perpetrator under the influence of substances would directly impact the youth's expectancies for substance use. Youth may have more positive expectancies about cannabis following PTEs to self-medicate for the stress and negative effects of adverse experiences ([Grummitt et al., 2021](#); [Sebalo et al., 2023](#)). For TBI, positive expectancies about alcohol may be due to the social facilitation of alcohol during development, where youth with TBI may be more likely to use alcohol to facilitate social connection ([Weil et al., 2018](#)). Furthermore, TBIs may confer more acute damage to the brain for most youth ([Arciniegas et al., 2005](#)), whereas PTEs confer more broad (i.e., non-specific to reward processes) and longer-term brain changes ([McLaughlin et al., 2020](#); [McLaughlin and Sheridan, 2016](#)), resulting in more heterogeneity following PTEs that may or may not influence youth's substance use, thus lower risk for PTEs compared to TBIs.

The examination of neurocognitive domains revealed distinctive patterns of association between TBI, PTEs, and specific cognitive functions. Notably, PTEs independently predicted greater accuracy in the attention and abstraction domains. This finding suggests that the impact of PTEs on attentional processes may be more pronounced among those where it is their only type of trauma exposure ([Walker et al., 2021](#)). While the broader literature shows deficits in attention and abstract domains due to PTEs ([Lund et al., 2022, 2020](#)), it is possible that the current study observed greater accuracy in attention and abstraction domains due to posttraumatic growth following PTEs whereby the youth has processed their trauma and has begun recovery ([Kilmer et al., 2014](#)). Recovery would be possible within the current sample as PTEs were categorized as exposure prior to age 18, not their last PTE exposure, so a significant amount of time may have passed since their last PTE exposure, allowing for posttraumatic growth. Conversely, TBI+PTEs predicted slower speed in the abstraction and emotion domains, indicating a combined influence on processing speed in tasks among those domains. Here, TBI+PTEs exposure was associated with quicker reaction times (faster or less processing of information), indicating that the combination of TBI+PTEs may make youth more impulsive in their decision-making, where they may not be thinking before making decisions, but this is not necessarily associated with a decrease in accuracy in our results. However, it is possible that in other samples, both speed and accuracy would be implicated following TBI+PTEs. Faster speed following TBI+PTEs could be due to a combined impact of TBI and PTEs whereby TBI conferred alterations in processing time that are sustained by broader and long-term alterations in brain functioning by PTEs. Further research is needed to parse out the independent and combined effects of TBI and PTEs on neurocognitive outcomes related to abstraction and emotion.

The lack of significant associations between TBI, PTEs, and working memory outcomes suggests that these exposures may not, independently or combined, contribute to alterations in working memory during adolescence in our sample. Given that working memory is known to fluctuate ([Adam and dedeBettencourt, 2019](#)), it is possible that TBI and PTEs exposure was sufficiently robust in the past, and the acute impacts of TBI and PTEs were not observed among our sample. For working memory, other factors may contribute to the maintenance of this cognitive capacity among the current sample, such as posttraumatic

growth (Kilmer et al., 2014). Further research may elucidate potential interactions with other variables or long-term effects beyond the assessed age range.

Conclusions drawn in the current study should be interpreted in the light of the limitations. First, to conserve power for analyses, the current study did not disaggregate specific types of PTEs or TBIs to examine the differential effects of PTEs and TBIs on outcomes. However, it is important to note that the current research question sought to examine the combined and distinctive effects of TBI and PTEs on these outcomes, which has not been explored within the literature. Future research can focus on the effect of specific types of PTEs in conjunction with specific types of TBIs on relevant outcomes. Second, there is a lack of specificity in the timing of TBI and PTE exposure to elucidate the cross-lagged relationships between TBI and PTEs on the outcomes. For example, does exposure to TBI first, followed by PTEs or vice versa, lead to differential outcomes for adolescents? Data on TBI and PTE timing was not available in the current study. Third, while the current study did control for covariates related to TBI, PTEs, and outcomes (e.g., sex at birth, SES, impulsivity, and general neurocognitive ability), it did not control for all potential covariates. Future research should explore potential mediating and moderating factors, such as posttraumatic growth, that may influence the observed relationships.

In conclusion, the current exploratory study contributes to the growing body of literature by presenting differential clustering of effects for mental health and neurocognition while observing combined clustering of effects for substance use highlights the complex nature, specificity, and interactions between TBIs and PTEs on adolescent neurodevelopment. The developmental implications of trauma via TBIs and/or PTEs during adolescence are considerable and worthy of further investigation.

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CRediT authorship contribution statement

Kevin Cummins: Writing review & editing, Supervision, Methodology, Formal analysis, Data curation, Conceptualization. **Sandra A. Brown:** Writing review & editing, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Jessica C. Reich:** Writing review & editing, Software, Formal analysis, Data curation. **Mary Milo O. Woodley:** Writing review & editing, Methodology, Formal analysis, Data curation. **Herry Patel:** Writing original draft, Visualization, Methodology, Investigation, Conceptualization. **Kate Brody Noonan:** Writing review & editing, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.dcn.2024.101427.

References

- Achenbach, T.M., Rescorla, L.A., 2001. Manual for the ASEBA School-Age Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families.
- Achenbach, T.M., Rescorla, L.A., 2003. Manual for the ASEBA Adult Forms & Profiles. University of Vermont, Research Center for Children, Youth, & Families.
- Adam, K.C.S., deBettencourt, M.T., 2019. Fluctuations of attention and working memory. *J. Cogn.* 2, 33. <https://doi.org/10.5334/joc.70>.
- Alway, Y., Gould, K.R., Johnston, L., McKenzie, D., Ponsford, J., 2016. A prospective examination of Axis I psychiatric disorders in the first 5 years following moderate to severe traumatic brain injury. *Psychol. Med.* 46, 1331–1341. <https://doi.org/10.1017/S0033291715002986>.
- American Psychiatric Association, 2013. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*, 5th ed. American Psychiatric Association, Washington, D.C.
- American Psychiatric Association, 1994. *Diagnostic and statistical manual of mental disorders: DSM-IV*, 4th ed. American Psychiatric Association.
- Amrhein, V., Trafimow, D., Greenland, S., 2019. Inferential statistics as descriptive statistics: there is no replication crisis if we don't expect replication. *Am. Stat.* 73, 262–270. <https://doi.org/10.1080/00031305.2018.1543137>.
- Arciniegas, D.B., Anderson, C.A., Topkoff, J., McAllister, T.W., 2005. Mild traumatic brain injury: a neuropsychiatric approach to diagnosis, evaluation, and treatment. *Neuropsychiatr. Dis. Treat.* 1, 311–327.
- Babikian, T., Asarnow, R., 2009. Neurocognitive outcomes and recovery after pediatric TBI: Meta-analytic review of the literature. *Neuropsychology* 23, 283–296. <https://doi.org/10.1037/a0015268>.
- Berg, J.M., Litzman, R.D., Bliwise, N.G., Lilienfeld, S.O., 2015. Parsing the heterogeneity of impulsivity: a meta-analytic review of the behavioral implications of the UPPS for psychopathology. *Psychol. Assess.* 27, 1129–1146. <https://doi.org/10.1037/pas0000111>.
- Bright, M.A., Thompson, L.A., 2018. Association of adverse childhood experiences with co-occurring health conditions in early childhood. *J. Dev. Behav. Pediatr.* 39, 37. <https://doi.org/10.1097/DBP.0000000000000514>.
- Brown, S.A., Brumback, T., Tomlinson, K., Cummins, K., Thompson, W.K., Nagel, B.J., Bellis, M.D.D., Hooper, S.R., Clark, D.B., Chung, T., Hasler, B.P., Colrain, I.M., Baker, F.C., Prouty, D., Pfefferbaum, A., Sullivan, E.V., Pohl, K.M., Rohlfing, T., Nichols, B.N., Chu, W., Tapert, S.F., 2015. The national consortium on alcohol and neurodevelopment in adolescence (NCANDA): a multisite study of adolescent development and substance use. *J. Stud. Alcohol Drugs* 76, 895. <https://doi.org/10.15288/JSAD.2015.76.895>.
- Brown, S.A., Myers, M.G., Lippke, L., Tapert, S.F., Stewart, D.G., Vik, P.W., 1998. Psychometric evaluation of the customary drinking and drug use record (CDDR): a measure of adolescent alcohol and drug involvement. *J. Stud. Alcohol* 59, 427–438. <https://doi.org/10.15288/jsa.1998.59.427>.
- Bucholz, K.K., Cadoret, R., Cloninger, C.R., Dinwiddie, S.H., Hesselbrock, V.M., Nurnberger, J.I., Reich, T., Schmidt, L., Schuckit, M.A., 1994. A new, semi-structured psychiatric interview for use in genetic linkage studies: a report on the reliability of the SSAGA. *J. Stud. Alcohol* 55, 149–158. <https://doi.org/10.15288/jsa.1994.55.149>.
- Corrigan, J.D., Bogner, J., 2007. Initial Reliability and Validity of the Ohio State University TBI Identification Method. *J. Head Trauma Rehabil.* 22 (6), 318. <https://doi.org/10.1097/01.HTR.0000300227.67748.77>.
- Dimoska-Di Marco, A., McDonald, S., Kelly, M., Tate, R., Johnstone, S., 2011. A meta-analysis of response inhibition and Stroop interference control deficits in adults with traumatic brain injury (TBI). *J. Clin. Exp. Neuropsychol.* 33, 471–485. <https://doi.org/10.1080/13803395.2010.533158>.
- Emery, C.A., Barlow, K.M., Brooks, B.L., Max, J.E., Villavicencio-Requis, A., Gnanakumar, V., Robertson, H.L., Schneider, K., Yeates, K.O., 2016. A systematic review of psychiatric, psychological, and behavioural outcomes following mild traumatic brain injury in children and adolescents. *Can. J. Psychiatry* 61, 259–269. <https://doi.org/10.1177/0706743716643741>.
- Fleminger, S., 2008. Long-term psychiatric disorders after traumatic brain injury. *Eur. J. Anaesthesiol.* 25, 123–130. <https://doi.org/10.1017/S0265021507003250>.
- Fusi, G., Crepaldi, M., Palena, N., Segatta, C., Bariselli, M., Cerrano, C., Rusconi, M.L., Vascello, M.G.F., 2023. Decision-making abilities under risk and ambiguity in adults with traumatic brain injury: what do we know so far? A systematic review and meta-analysis. *J. Clin. Exp. Neuropsychol.* 45, 389–410. <https://doi.org/10.1080/13803395.2023.2245107>.
- Gardner, M.J., Thomas, H.J., Erskine, H.E., 2019. The association between five forms of child maltreatment and depressive and anxiety disorders: a systematic review and meta-analysis. *Child Abus. Negl.* 96, 104082. <https://doi.org/10.1016/j.chiabu.2019.104082>.
- Goh, M.S.L., Looi, D.S.H., Goh, J.L., Sultana, R., Goh, S.S.M., Lee, J.H., Chong, S.-L., 2021. The impact of traumatic brain injury on neurocognitive outcomes in children: a systematic review and meta-analysis. *J. Neurol. Neurosurg. Psychiatry* 92, 847–853. <https://doi.org/10.1136/jnnp-2020-325066>.

- Grummitt, L., Kelly, E., Barrett, E., Keyes, K., Newton, N., 2021. Targets for intervention to prevent substance use in young people exposed to childhood adversity: a systematic review. *PLOS ONE* 16, e0252815. <https://doi.org/10.1371/journal.pone.0252815>.
- Hawkins, M.A.W., Layman, H.M., Ganson, K.T., Tabler, J., Ciciolla, L., Tsotsoros, C.E., Nagata, J.M., 2021. Adverse childhood events and cognitive function among young adults: prospective results from the national longitudinal study of adolescent to adult health. *Child Abuse Negl.* 115, 105008 <https://doi.org/10.1016/j.chiabu.2021.105008>.
- Hurlbert, S.H., Levine, R.A., Utts, J., 2019. Coup de Grâce for a Tough Old Bull: “Statistically Significant” Expires. *Am. Stat.* 73, 352–357. <https://doi.org/10.1080/00031305.2018.1543616>.
- Hurlbert, S.H., Lombardi, C.M., 2009. Final Collapse of the Neyman-Pearson Decision Theoretic Framework and Rise of the neoFisherian. *Ann. Zool. Fenn.* 46, 311–349. <https://doi.org/10.5735/086.046.0501>.
- Jackson, D.B., Posick, C., Vaughn, M.G., Testa, A., 2022. Adverse childhood experiences and traumatic brain injury among adolescents: findings from the 2016–2018 National Survey of Children’s Health. *Eur. Child Adolesc. Psychiatry* 31, 289–297. <https://doi.org/10.1007/s00787-020-01687-1>.
- Kavanaugh, B.C., Dupont-Frechette, J.A., Jerskey, B.A., Holler, K.A., 2017. Neurocognitive deficits in children and adolescents following maltreatment: Neurodevelopmental consequences and neuropsychological implications of traumatic stress. *Appl. Neuropsychol. Child* 6, 64–78. <https://doi.org/10.1080/21622965.2015.1079712>.
- Kessler, R.C., McLaughlin, K.A., Green, J.G., Gruber, M.J., Sampson, N.A., Zaslavsky, A.M., Aguilar-Gaxiola, S., Alhamzawi, A.O., Alonso, J., Angermeyer, M., Benjet, C., Bromet, E., Chatterji, S., Girolamo, G., de, Demyttenaere, K., Fayyad, J., Florescu, S., Gal, G., Gureje, O., Haro, J.M., Hu, C., Karam, E.G., Kawakami, N., Lee, S., Lépine, J.-P., Ormel, J., Posada-Villa, J., Sagar, R., Tsang, A., Üstün, T.B., Vassilev, S., Viana, M.C., Williams, D.R., 2010. Childhood adversities and adult psychopathology in the WHO World Mental Health Surveys. *Br. J. Psychiatry* 197, 378–385. <https://doi.org/10.1192/bjp.bp.110.080499>.
- Kilmer, R.P., Gil-Rivas, V., Griese, B., Hardy, S.J., Hafstad, G.S., Alisic, E., 2014. Posttraumatic growth in children and youth: clinical implications of an emerging research literature. *Am. J. Orthopsychiatry* 84, 506–518. <https://doi.org/10.1037/ort0000016>.
- Koponen, S., Taiminen, T., Portin, R., Himanen, L., Isoniemi, H., Heinonen, H., Hinkka, S., Tenovu, O., 2002. Axis I and II psychiatric disorders after traumatic brain injury: a 30-year follow-up study. *Am. J. Psychiatry* 159, 1315–1321. <https://doi.org/10.1176/appi.ajp.159.8.1315>.
- Kosted, R., Kirsch, D.E., Le, V., Fromme, K., Lippard, E.T.C., 2023. Subjective response to alcohol: interactive effects of early life stress, parental risk for mood and substance use disorders, and drinking context. *Pharmacol. Biochem. Behav.* 229, 173591 <https://doi.org/10.1016/j.pbb.2023.173591>.
- Lavigne, A.M., Wood, M.D., Janssen, T., Wiers, R.W., 2017. Implicit and explicit alcohol cognitions: the moderating effect of executive functions. *Alcohol Alcohol* 52, 256–262. <https://doi.org/10.1093/alcac/agw066>.
- Lee, R.S.C., Hoppenbrouwers, S., Franken, I., 2019. A systematic meta-review of impulsivity and compulsivity in addictive behaviors. *Neuropsychol. Rev.* 29, 14–26. <https://doi.org/10.1007/s11065-019-09402-x>.
- Lovallo, W.R., 2013. Early life adversity reduces stress reactivity and enhances impulsive behavior: Implications for health behaviors. *Int. J. Psychophysiol., Blunted Cardiovasc. React. - What Does It Mean?* 90, 8–16. <https://doi.org/10.1016/j.ijpsycho.2012.10.006>.
- Lund, J.I., Boles, K., Radford, A., Toombs, E., Mushquash, C.J., 2022. A Systematic review of childhood adversity and executive functions outcomes among adults. *Arch. Clin. Neuropsychol.* 37, 1118–1132. <https://doi.org/10.1093/arclin/acac013>.
- Lund, J.I., Toombs, E., Radford, A., Boles, K., Mushquash, C., 2020. Adverse childhood experiences and executive function difficulties in children: a systematic review. *Child Abuse Negl.* 106, 104485 <https://doi.org/10.1016/j.chiabu.2020.104485>.
- Max, J.E., Pardo, D., Hanten, G., Schachar, R.J., Saunders, A.E., Ewing-Cobbs, L., Chapman, S.B., Dennis, M., Wilde, E.A., Bigler, E.D., Thompson, W.K., Yang, T.T., Levin, H.S., 2013. Psychiatric disorders in children and adolescents six-to-twelve months after mild traumatic brain injury. *J. Neuropsychiatry Clin. Neurosci.* 25, 272–282. <https://doi.org/10.1176/appi.neuropsych.12040078>.
- McHugo, G.J., Krassenbaum, S., Donley, S., Corrigan, J.D., Bogner, J., Drake, R.E., 2017. The prevalence of traumatic brain injury among people with co-occurring mental health and substance use disorders. *J. Head. Trauma Rehabil.* 32, E65 <https://doi.org/10.1097/HTR.0000000000000249>.
- McLaughlin, K.A., Colich, N.L., Rodman, A.M., Weissman, D.G., 2020. Mechanisms linking childhood trauma exposure and psychopathology: a transdiagnostic model of risk and resilience. *BMC Med.* 18, 96. <https://doi.org/10.1186/s12916-020-01561-6>.
- McLaughlin, K.A., Sheridan, M.A., 2016. Beyond cumulative risk: a dimensional approach to childhood adversity. *Curr. Dir. Psychol. Sci.* 25, 239–245. <https://doi.org/10.1177/0963721416655883>.
- Nigg, J.T., 2017. Annual Research Review: On the relations among self-regulation, self-control, executive functioning, effortful control, cognitive control, impulsivity, risk-taking, and inhibition for developmental psychopathology. *J. Child Psychol. Psychiatry* 58, 361–383. <https://doi.org/10.1111/jcpp.12675>.
- Pagulayan, K.F., Temkin, N.R., Machamer, J.E., Dikmen, S.S., 2016. Patterns of Alcohol Use after Traumatic Brain Injury. *J. Neurotrauma* 33, 1390–1396. <https://doi.org/10.1089/neu.2015.4071>.
- Patel, P., Oremus, M., 2022. The association between adverse childhood experiences and late-life cognition: a systematic review of cross-sectional and case-control studies. *Gerontol. gnac041*. <https://doi.org/10.1093/geront/gnac041>.
- Perry, D.C., Sturm, V.E., Peterson, M.J., Pieper, C.F., Bullock, T., Boeve, B.F., Miller, B.L., Guskiewicz, K.M., Berger, M.S., Kramer, J.H., Welsh-Bohmer, K.A., 2016. Association of traumatic brain injury with subsequent neurological and psychiatric disease: a meta-analysis. *J. Neurosurg.* 124, 511–526. <https://doi.org/10.3171/2015.2.JNS14503>.
- Pohl, K.M., Sullivan, E.V., Podhajsky, S., Baker, F.C., Brown, S.A., Clark, D.B., Colrain, I.M., DeBellis, M., Goldston, D., Nagel, B.J., Nooner, K.B., Tapert, S.F., Pfefferbaum, A., 2022. “NCANDA PUBLIC 6Y REDCAP V04” Data Release of the National Consortium on Alcohol and NeuroDevelopment in Adolescence (NCANDA). Sage Bionetworks Synapse. <https://doi.org/10.7303/syn26951066>.
- Puetz, V.B., McCrory, E., 2015. Exploring the relationship between childhood maltreatment and addiction: a review of the neurocognitive evidence. *Curr. Addict. Rep.* 2, 318–325. <https://doi.org/10.1007/s40429-015-0073-8>.
- R Core Team, 2022. R: A language and environment for statistical computing.
- R-Mercier, A., Masson, M., Bussières, E.-L., Cellard, C., 2018. Common transdiagnostic cognitive deficits among people with psychiatric disorders exposed to childhood maltreatment: a meta-analysis. *Cogn. Neuropsychiatry* 23, 180–197. <https://doi.org/10.1080/13546805.2018.1461617>.
- Rochat, L., Beni, C., Annoni, J.-M., Vuadens, P., Linden, M.V., der, 2013. How inhibition relates to impulsivity after moderate to severe traumatic brain injury. *J. Int. Neuropsychol. Soc.* 19, 890–898. <https://doi.org/10.1017/S1535617713000672>.
- Rothman, K.J., 1990. No adjustments are needed for multiple comparisons. *Epidemiol. Camb. Mass* 1, 43–46.
- Sebalo, I., Königová, M.P., Sebalo Vňuková, M., Anders, M., Ptáček, R., 2023. The associations of adverse childhood experiences (ACEs) with substance use in young adults: a systematic review. *Subst. Abuse Res. Treat.* 17, 11782218231193914 <https://doi.org/10.1177/11782218231193914>.
- Sullivan, G.M., Feinn, R.S., 2021. Facts and fictions about handling multiple comparisons. *J. Grad. Med. Educ.* 13, 457–460. <https://doi.org/10.4300/JGME-D-21-00599.1>.
- Teicher, M.H., Samson, J.A., Polcari, A., McGreenery, C.E., 2006. Sticks, Stones, and hurtful words: relative effects of various forms of childhood maltreatment. *Am. J. Psychiatry* 163, 993–1000. <https://doi.org/10.1176/ajp.2006.163.6.993>.
- Uhlhaas, P.J., Davey, C.G., Mehta, U.M., Shah, J., Torous, J., Allen, N.B., Avenevoli, S., Bella-Awusah, T., Chanan, A., Chen, E.Y.H., Correll, C.U., Do, K.Q., Fisher, H.L., Frangou, S., Hickie, I.B., Keshavan, M.S., Konrad, K., Lee, F.S., Liu, C.H., Luna, B., McGorry, P.D., Meyer-Lindenberg, A., Nordentoft, M., Öngür, D., Patton, G.C., Paus, T., Reininghaus, U., Sawa, A., Schoenbaum, M., Schumann, G., Srihari, V.H., Susser, E., Verma, S.K., Woo, T.W., Yang, L.H., Yung, A.R., Wood, S.J., 2023. Towards a youth mental health paradigm: a perspective and roadmap. *Mol. Psychiatry* 28, 3171–3181. <https://doi.org/10.1038/s41380-023-02202-z>.
- Wagenmakers, E.-J., Wetzels, R., Borsboom, D., van der Maas, H.L.J., Kievit, R.A., 2012. An agenda for purely confirmatory research. *Perspect. Psychol. Sci.* 7, 632–638. <https://doi.org/10.1177/1745691612463078>.
- Walker, C.S., Walker, B.H., Brown, D.C., Buttross, S., Sarver, D.E., 2021. Defining the role of exposure to ACEs in ADHD: Examination in a national sample of US children. *Child Abuse Negl.* 112, 104884 <https://doi.org/10.1016/j.chiabu.2020.104884>.
- Weil, Z.M., Corrigan, J.D., Karelina, K., 2018. Alcohol use disorder and traumatic brain injury. *Alcohol Res. Curr. Rev.* 39, 171–180.
- West, S.L., 2011. Substance use among persons with traumatic brain injury: a review. *NeuroRehabilitation* 29, 1–8. <https://doi.org/10.3233/nre-2011-0671>.
- Whelan-Goodinson, R., Ponsford, J., Johnston, L., Grant, F., 2009. Psychiatric disorders following traumatic brain injury: their nature and frequency. *J. Head. Trauma Rehabil.* 24, 324. <https://doi.org/10.1097/HTR.0b013e3181a712aa>.
- Willhelm, A.R., Fortes, P.M., Czermainski, F.R., Rates, A.S.A., Almeida, R.M.M. de, 2016. Neuropsychological and behavioral assessment of impulsivity in adolescents: a systematic review. *Trends Psychiatry Psychother.* 38, 128–135. <https://doi.org/10.1590/2237-6089-2015-0019>.
- Zgaljardic, D.J., Seale, G.S., Schaefer, L.A., Temple, R.O., Foreman, J., Elliott, T.R., 2015. Psychiatric disease and post-acute traumatic brain injury. *J. Neurotrauma* 32, 1911–1925. <https://doi.org/10.1089/neu.2014.3569>.