# The Relationship Between Trauma Exposure and Memory Impairment: The Mediating Effect of Anxiety, Depression, and Sleep Disturbance

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**Abstract**

The impact of trauma on individuals can have a profound and lasting effect on wellbeing. Ongoing research in the field is interested in the neurocognitive impact of trauma, including its effect on memory. However, there are some discrepancies regarding the direction of various effects found. While some studies find that trauma does not impact memory at all or they attribute the effects to mediators, others have found there to be an independent effect. Furthermore, few studies have examined the relationship between trauma exposure and memory impairment in youth. The current study aims to provide further clarification to the literature by analyzing longitudinal data from the Adolescent Brain Cognitive Development (ABCD) Study and hypothesizes that trauma exposure is associated with episodic memory impairment, and that anxious-depressive symptoms and sleep disturbances mediate this relationship.

## Introduction

Posttraumatic stress disorder (PTSD) is a mental health disorder that may develop after exposure to a potentially traumatic event that is beyond a typical stressor (American Psychiatric Association (APA), 2022). According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5-TR), symptoms of PTSD include recurrent, involuntary, and intrusive distressing memories of the traumatic event, avoidance of or efforts to avoid associated distressing memories, alterations in arousal and reactivity, and changes in cognition and mood such as the inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia) (APA, 2022). Thus, memory impairment can be associated with the development of this disorder, although not all PTSD patients need to show memory impairment to receive a diagnosis of PTSD. Importantly, the DSM-5-TR criteria target the memory of the traumatic event itself. This paper aims to supplement ongoing research about the impact of trauma on memory deficits in not just the traumatic memory, but also everyday memory.

To ensure clarity, the terms trauma and memory impairment are defined below. According to the American Psychological Association, trauma is “any disturbing experience that results in significant fear, helplessness, dissociation, confusion, or other disruptive feelings intense enough to have a long-lasting negative effect on a person’s attitudes, behavior, and other aspects of functioning” (American Psychological Association, n.d.). The DSM-5-TR has a more specific definition of trauma as “any event (or events) that may cause or threaten death, serious injury, or sexual violence to an individual, a close family member, or a close friend” (APA, 2022). Examples of traumatic events include natural disasters, accidents, sexual assaults, physical assaults, combat, childhood sexual abuse, torture, or life-threatening illness (APA, 2022). While childhood neglect is form of maltreatment or adverse childhood experiences (ACEs), there is no consensus regarding whether neglect should be

considered a trauma or not. Therefore, for the purposes of this review, findings involving neglect are also included. Memory impairment is defined as “the loss of memory associated with a memory disorder”—a memory disorder is “any impairment in the ability to encode, retain, or retrieve information or representations of experiences” and one of the possible causes listed for memory impairment is psychological trauma (American Psychological Association, n.d.). Thus, there appears to be a possible association between trauma and memory impairment.

In support of this, PTSD patients have been found to report deficits in memory that are not limited to the traumatic event but also involve everyday working memory, verbal memory, explicit memory, etc. For example, a study that looked at Vietnam veterans found cognitive deficits in tasks of working memory, sustained attention, and initial learning (Vasterling et al., 2002). Adult survivors of childhood sexual abuse show deficits in verbal (not visual) memory as measured by the Wechsler Memory Scale (WMS) in another study (Bremner et al., 1995). An additional study found poorer explicit, but not implicit memory in Holocaust survivors (Golier et al., 2002). The effects of trauma on memory, while not thoroughly researched in the field, suggest that the scope of memory impairment extends beyond the traumatic event itself.

Beyond these individual studies, meta-analyses have been conducted to examine the effects of trauma on memory by combining data across studies. For example, Brewin et al. (2007) compared data from 27 studies that investigated memory in samples with PTSD and healthy controls. The study aimed to summarize existing literature objectively and differentiate between different types of episodic memory (looking at visual and verbal memory) and different trauma types (military trauma, state persecution–terror, and interpersonal trauma). Despite variability within studies, the meta-analysis found a decrement in memory performance in PTSD survivors with a small to moderate effect size and the

authors also found this effect was not specific to the type of trauma experienced (Brewin et al., 2007). A stronger effect was seen for verbal memory over visual memory in Brewin et al. (2007), which could be attributed to a functional separation between verbal and nonverbal processing in PTSD (Constans, 2005).

Another meta-analysis investigated trauma and episodic memory in 47 studies and 3,062 subjects and found that PTSD patients show episodic memory deficits compared to all controls (Petzold & Bunzeck, 2022). The authors examined a general relationship between PTSD and episodic memory as well as possible effects of trauma history (traumatized control vs. healthy control) and stimulus material (verbal vs. nonverbal tasks). All analyses reached statistical significance and showed poorer performance in the PTSD group versus the control group, except for the comparison between PTSD and traumatized control when they measured verbal versus non-verbal memory (Petzold & Bunzeck, 2022). While the authors concluded that there is a link between increased trauma and deficits in memory performance (especially verbal—consistent with previous studies), the study acknowledged that the causal relationship between PTSD and memory was less clear (Petzold & Bunzeck, 2022). They note that further research is needed to determine whether PTSD caused memory impairments or whether a memory deficit increased the likelihood of developing PTSD, or both. In support of the second point (memory deficits increasing the likelihood of PTSD), Parslow and Jorm (2007) conducted a study with 2,097 young adults using pre-and post-trauma neurocognitive measures before and after a natural disaster. While the PTSD symptoms of reexperiencing and arousal were inversely associated with word recall among other neurocognitive abilities, the authors also found worse performance on all neurocognitive measures prior to the trauma, suggesting that memory deficits are a risk factor for developing PTSD rather than an outcome of having PTSD (Parslow & Jorm, 2007).

In contrast, other studies have not supported the association between abuse and memory impairments but have found a relationship between neglect and memory deficits. Terock et al. (2020) conducted a study to investigate the association of childhood trauma with verbal declarative memory performance and the mediating role of alexithymia. They concluded that while childhood abuse was not associated with a reduced memory capacity, childhood emotional neglect was particularly detrimental to memory functioning in adulthood (Terock et al., 2020). This association was also significantly mediated by alexithymia, which refers to the inability to identify and describe emotions experienced by oneself (Cherney, 2021). This mediating effect emphasizes that emotional processing capabilities play an important role in memory. This study provides an important contribution to the literature by focusing on the direct relationship between trauma and memory, both with and without accounting for mediating variables. While they did not find a relationship between abuse and memory deficits, their findings do support a significant relationship between neglect and reduced memory capacity, suggesting that memory impairments extend to the broader category of maltreatment.

Some studies, on the other hand, find a lack of relationship between memory and trauma. Pursuing the overall idea that different trauma populations could account for variability in results when it comes to neurocognitive decline, Jelinek et al. (2013) looked at World War II survivors and explored the effects of PTSD on older adults and their offspring following forced displacement. They found there to be no clear evidence for mnemonic dysfunction in displaced individuals with PTSD. Performance of the cognitive tests also neither correlated with PTSD nor depression severity, which contradicts the existing literature (Herrmann et al., 2007). This study, however, was limited by a small sample size of 20 PTSD-displaced participants and 11 nondisplaced individuals. Considering the mean age of the participants was 70 years, the authors acknowledged that there could have been memory

deficits for decades in the past. Another study that does not support a relationship between trauma and memory deficits was conducted by Rubin and colleagues. Rubin et al. (2008) examined whether memory in trauma survivors was impaired or fragmented while offering a new perspective that memories in participants with higher PTSD symptoms may be more emotionally intense. In a series of studies, the authors found that voluntary and involuntary memories in participants with high PTSD symptom severity showed greater emotional intensity, higher frequency of voluntary and involuntary retrieval, but not more narrative fragmentation (Rubin et al., 2008). The lack of narrative fragmentation brings into question whether there is a relationship between memory deficits and trauma. Given the inconsistencies in the literature, greater clarity regarding the relationship and direction between trauma and memory is needed including whether this directionality changes with a lack of diagnosis but the presence of trauma. It is also possible that there are other mediating variables that account for the relationship between trauma and memory.

Not only emotion, but also several other factors such as sleep, anxiety, and depression have acted as mediators in studies investigating the relationship between trauma and memory impairments. In the attempt to better understand the potential causes and directionality of the relationship between trauma and memory, it is important to consider these mediators. Sleep disturbance is an important symptom that is involved in the clinical picture of PTSD and is also associated with neurocognitive deficits. Per the DSM-5-TR criteria for PTSD, the individual may experience intrusive symptoms in the form of “recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s)” as well as “sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep)” (APA, 2022). These symptoms have the potential to hamper the individual’s cognitive function in both the short- and long-term.

McCarthy et al. (2019) analyzed the responses of 714 veterans (from a population-based sample of 3,157 U.S. military veterans who participated in the National Health and Resilience in Veterans Study) who self-reported poor sleep quality. They found poor sleep to be higher for individuals who screened positive for potential PTSD compared to those who did not screen positive (84.2% vs. 24.7%, respectively) and revealed significant associations between greater severity of PTSD symptoms and poorer performance on measures of cognitive functioning with sleep acting as a partial mediator (McCarthy et al., 2019). Likewise, an unpublished dissertation by Montry (2018) specifically looked at memory performance in veterans with PTSD and noted that while individuals with PTSD exhibited worse memory performance (especially verbal memory), the group differences were no longer significant when sleep disturbance was added to the model. As this study was cross-sectional, temporal precedence (that one event led to another) cannot be inferred–disrupted sleep may also exacerbate PTSD symptoms, suggesting the relationship between PTSD and sleep may be reciprocal. Moreover, the study did not look at trauma exposure, only PTSD as a clinical diagnosis, limiting inferences about the effect of trauma severity on memory performance, and it looked at only veterans, not civilians, which could affect the generalizability of the results. Nonetheless, the research on sleep as a mediator of the relationship between trauma and cognitive functioning including memory is sufficient to warrant further clarification.

Two other variables that tend to be considered not only as mediators but also as potential comorbidities are anxiety and depression. A comparatively well-researched subject, anxiety and depression are closely related to both trauma and memory (among other cognitive functions) and have also been examined as mediators when analyzing the relationship between trauma and neurocognitive abilities. Specifically, anxious symptoms in

PTSD include sleep disturbance, negative emotional state including fear, irritability, difficulties with concentration, and perceived imminent threat (APA, 2022). Depressive symptoms that overlap with PTSD include sleep disturbance, impaired ability to concentrate, tiredness and fatigue, anhedonia (loss of interest in activities), feelings of detachment and estrangement from others, and impaired ability to feel positive emotions (APA, 2022). Anxiety and depressive disorders are also highly comorbid with PTSD (Price et al., 2019).

Furthermore, anxiety and depression are also associated with neurocognitive functioning. Anxiety has been found to be a risk factor for impaired memory and neurocognitive decline. Sinoff and Werner (2003) analyzed a sample of 137 elderly individuals and found that anxiety acted as a predictor for cognitive decline. Anxiety has also been found to compromise performance on cognitively demanding tasks by decreasing working memory resources and reducing accuracy in working memory tasks (Maloney et al., 2014; Lisica et al., 2022). There is also a substantial amount of research relating anxiety to deficits in episodic memory, specifically. For example, a study examining anxiety and related disorders across panic with and without agoraphobia, social anxiety disorder, generalized anxiety disorder, obsessive-compulsive disorder, and specific phobia showed impairments in episodic memory performance but no deficits in verbal fluency or psychomotor speed (Airaksinen et al., 2005). Thus, it is possible that greater anxiety symptoms associated with trauma may lead to impaired memory performance.

Depressive symptoms also have a close link with memory impairments. Looking at the bidirectional temporal relationship between cognitive decline and depression, one study found that baseline depressive symptoms did not predict cognitive decline; instead, they found that an accelerated annual increase in depressive symptoms predicted memory impairment (among other functions) at baseline (Vinkers et al., 2004). Other studies have found depressive symptoms to significantly predict cognitive decline in older populations

(Wilson et al., 2004; Yaffe et al., 1999). Beyond older populations, depressed individuals of all ages have also been shown to have poor memory for positive events and impaired recollection, and chronic stress was found to be one contributor to this effect (Dillon & Pizzagalli, 2018). Thus, the clear relationship between depressive disorders and memory deficits suggests that depressive symptoms provide another potential mediator of the association between trauma and memory impairments.

Thus far, this review has focused on adult populations. However, to further understand the impact of trauma on episodic memory (or lack thereof), we also need to examine these effects in younger populations with an early onset of trauma. Previous studies have examined memory impairments associated with anxiety (Sbicigo et al., 2020) and depression (Barch et al., 2019) in childhood and found there to be lower memory performance in anxious and depressed youth, where severity predicted the extent of impairment in both studies. Barch and colleagues in particular showed specific deficits in episodic memory in anxious children, suggesting that long-term memory for previous experiences may be especially impaired in those with depressive symptoms (Barch et al., 2019). However, few studies have tested depression as a potential mediator of the relationship between trauma and memory performance in youth. It is important to examine a younger population to understand whether there is a relationship between trauma and memory performance in children and whether sleep, anxiety, or depressive symptoms mediate this relationship using a longitudinal design that can account for the temporal precedence between variables.

Based on this review of the literature, the current study aims to investigate the association between trauma exposure and episodic memory performance while considering anxiety, depression, and sleep as potential mediating variables in a pediatric population. Research thus far has focused on either the association between PTSD and memory or the relationship between anxiety, depression, or sleep and memory. This limits the ability to

analyze these variables simultaneously in the same population. There are also some inconsistencies in whether the relationship between trauma and memory exists at all, thus this study aims to provide additional research for clarification. There have also been few studies that look at the combination of these variables during development, which is a critical time for intervention to prevent long-term negative effects. Moreover, much of the research so far has only examined a clinical diagnosis of PTSD, when there have been studies that show that symptom severity is more strongly associated with cognitive performance than the diagnosis itself (Pitts et al., 2022; Scott et al., 2015). Thus, the current study hypothesizes that trauma exposure will significantly predict episodic memory impairment in a pediatric population. It also hypothesizes that this effect will still be apparent even when anxious-depressive symptoms and sleep disturbance are included as potential mediators. While it is predated that these variables will result in partial mediation, the lack of a full mediating effect would suggest that anxious-depressive symptoms and sleep difficulties do not fully account for the relationship between trauma and memory.

## Methods

### Participants

This study used the dataset from release 5.1 of the Adolescent Brain Cognitive Development (ABCD) Study, which is the largest longitudinal study designed that follows the environmental, genetic, neurobiological, and behavioral correlates of children for at least ten years (Barch et al., 2018). The participants in this study were 11,876 children aged 9-10 years who completed several assessments that gauged their physical, cognitive, and academic functioning. These assessments are done annually. They also went through neuroimaging and biospecimen collection across 21 sites in the United States (USA)—this is done biannually. These participants will be continuously assessed until they turn 19-20 years of age, and the present study will analyze three years' worth of data (including baseline, first-year follow-up,

and second-year follow-up). The primary sociodemographic factors considered in sample recruitment were age, gender, race/ethnicity, socio-economic status, and urbanicity. The lead investigators collaborated with private, public, and chartered schools to obtain as much of a representative sample as feasible. Data was securely kept separate from identifying information. The researchers also obtained parental consent and children's assent, and the families were compensated for their participation. The sample was predominantly non-Hispanic White and balanced between males and females. Both household income and education had a skewed distribution with about two-thirds of the parents earning over $50000 a year and the majority with a bachelor's degree or higher. The use of this publicly available, de-identified dataset was approved by the Institutional Review Board at Vanderbilt University.

Table 1 shows the basic demographic information of the ABCD Study sample. The mean age of the participants was 9.92 with a standard deviation of 0.63. Approximately 52% of the sample identified as White, while the others identified as Hispanic, African American, or Other. In terms of sex, approximately 47% identified as female and 52% as male. The most common income group (27.91%) had an annual household income between $100,000 and $199,999, and more than half of the sample had parents with a bachelor's degree or higher.

**Table 1**

*Summary of demographic characteristics of the sample (N = 11,876)*

| | *Mean* | *SD* |
|---|---|---|
| Age (years) | 9.92 | 0.63 |
| Episodic Memory | 105.32 | 16.87 |
| Trauma Total | 0.51 | 0.93 |
| Anxious-Depression | 0.18 | 0.23 |

| | | |
|---|---|---|
| Sleep disturbance | 0.13 | 0.27 |
| | *N* | *%* |
| Sex | | |
| Female | 5,682 | 47.84 |
| Male | 6,194 | 52.16 |
| Race-Ethnicity | | |
| White | 6,180 | 52.04 |
| Hispanic | 1,784 | 15.02 |
| African American | 2,411 | 20.3 |
| Other | 1,499 | 12.62 |
| Missing | 2 | 0.02 |
| Household Annual Income | | |
| < $5,000 | 417 | 3.51 |
| $5,000-$11,999 | 421 | 3.54 |
| $12,000-$15,999 | 273 | 2.3 |
| $16,000-$24,999 | 524 | 4.41 |
| $25,000-$34,999 | 654 | 5.51 |
| $35,000-$49,999 | 934 | 7.86 |
| $50,000-$74,999 | 1,499 | 12.62 |
| $75,000-$99,999 | 1,572 | 13.24 |
| $100,000-$199,999 | 3,314 | 27.91 |
| ≥ $200,000 | 1,250 | 10.53 |
| Missing | 1,018 | 8.57 |
| Parental Education | | |

| | | |
|---|---|---|
| No degree (1-11) | 604 | 5.09 |
| Highschool degree/GED | 1,442 | 12.14 |
| Some college | 1,949 | 16.41 |
| Associate degree | 1,538 | 12.95 |
| Bachelor's degree | 3,333 | 28.07 |
| Master's degree | 2,279 | 19.19 |
| Professional/Doctoral degree | 714 | 6.01 |
| Missing | 17 | 0.14 |

*Note.* The "Other" Race/Ethnicity category includes those who were identified by their parent as American Indian/Native American, Alaska Native, Native Hawaiian, Guamanian, Samoan, Other Pacific Islander, Asian Indian, Chinese, Filipino, Japanese, Korean, Vietnamese, Other Asian, or Other Race. SD, Standard Deviation; GED, General Education Development degree.

**Measures**

For the present analyses, we examined four main variables from the study: trauma exposure, episodic memory, anxious-depressive symptoms, and sleep disturbance. Trauma exposure was assessed using the PTSD criterion A traumatic events checklist from the Kiddie Schedule for Affective Disorders and Schizophrenia (K-SADS) (Kaufman et al., 1997), which was administered to a parent or guardian and contained 17 items assessing the occurrence of traumatic events. A sum of these 17 items will be taken to make a single "trauma_total" variable that will have a score ranging from 1-17, with a higher score demonstrating increased trauma exposure.

The episodic memory variable was measured with the Picture Sequence Memory Test from the NIH Toolbox, which requires participants to reproduce the order of an arbitrarily ordered sequence of pictures presented on a computer. The sequence length varies from 6 to

15 pictures to adjust for ability and takes about 7 minutes. The level of difficulty of the task for different age ranges was determined during pilot testing. For the test, color images appear one by one on the computer screen in a fixed sequence. Each picture initially appears in the middle of the screen as a 3- by 5-inch image. As each picture appears, a short recording is played that briefly describes the contents of each image. After 3 seconds of displaying the entire sequence, the images are placed in a random spatial array in the middle of the screen. Participants used the touch screen to move each picture to the correct position in a sequence. The participant's score on the Picture Sequence Memory Test is derived from the cumulative number of adjacent pairs of pictures remembered correctly over three learning trials. So, if the participant placed the images in the sequence 1-3-4-2-5-6, a score of 1 would be given as the only correct adjacent pair is 5-6.

The Anxious-Depressed variable comes from the Child Behavior Checklist (CBCL) questionnaire for school-aged children. This checklist includes 113 items rated on a scale of 0-2 (not true, sometimes true, always true), where all items target different behavioral or socioemotional problems. The depression items on the Anxious-Depressed scale are as follows: complains of loneliness; cries a lot; feels or complains that no one loves him/her; feels worthless or inferior; feels too guilty; and unhappy, sad, or depressed. The anxiety items on the Anxious-Depressed scale are as follows: fears he/she might think or do something bad; feels he/she has to be perfect; feels others are out to get him/her; nervous, high-strung, or tense; too fearful or anxious; self-conscious or easily embarrassed; suspicious; and worries.

The sleep disturbance variable also comes from the CBCL questionnaire and has the same 0-2 scale. The sleep disturbance items are as follows: sleeps less than most kids, sleeps more than most kids, and trouble sleeping.

### Data Analysis

First, we examined Pearson correlations between 1) trauma exposure and Anxious-Depressed scores, 2) trauma exposure and sleep disturbance scores, 3) trauma exposure and episodic memory, 4) Anxious-Depressed scores and episodic memory, 5) sleep disturbance and episodic memory, and 6) Anxious-Depressed scores and sleep disturbance at baseline in RStudio. Then we performed mediation analysis in R to examine a multiple mediation model that looks at the direct effect of baseline KSADS trauma exposure on year 2 episodic memory and the indirect effects of year 1 anxious-depression and year 1 sleep disturbances on episodic memory. We used bootstrapping for the mediation model. We also included a contrast in the model to see if the two indirect effects are equal or not.

## Results

### Trauma, Sleep, Anxious-Depression, and Episodic Memory

First, we examined the relationships between trauma exposure, sleep disturbance, anxious-depressive symptoms, and episodic memory. Descriptive statistics for these variables are depicted in Table 2. Trauma exposure was negatively correlated with episodic performance, suggesting that increased trauma exposure was associated with decreased episodic memory performance. Sleep disturbance and anxious-depressive scores were also negatively correlated with episodic memory performance, aligning with the findings in the literature. There was a significant positive correlation between sleep disturbance and trauma exposure. Anxious depressive scores were significantly positively correlated to both trauma exposure and sleep disturbance.

**Table 2**

*Means, standard deviations, and correlations with confidence intervals.*

| Variable | *M* | *SD* | 1 | 2 | 3 |
|---|---|---|---|---|---|
| 1. Trauma Total | 0.51 | 0.93 | | | |
| 2. Sleep | 0.13 | 0.27 | .11** [.09, .12] | | |
| 3. Anxious-Depressed | 0.18 | 0.23 | .13** [.11, .14] | .41** [.39, .42] | |
| 4. Episodic Memory | 105.31 | 16.87 | -.03** [-.05, -.01] | -.03** [-.05, -.01] | -.02* [-.04, -.00] |

*Note. M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation. The confidence interval is a plausible range of population correlations that could have caused the sample correlation (Cumming, 2014). * indicates $p < .05$. ** indicates $p < .01$.

**Mediating Effects of Sleep Disturbance and Anxious-Depression**

To further understand the temporal precedence between our variables of interest and evaluate the mediating effects of sleep disturbance and anxious-depression, we executed a multiple mediation model in R. Table 3 shows the statistics from the mediation analysis and Figure 1 depicts the results of the model. We found a significant total effect between trauma exposure and episodic memory and found that the indirect effects of trauma exposure on episodic memory through anxious depression and through sleep disturbance were both not significant. The effect of anxious-depression on episodic memory and sleep disturbance on episodic memory were also not significant. Trauma exposure on episodic memory was found to be significant. We also found that the covariance between the two mediators (anxious depression and sleep) was significant. We also included a contrast in the model to see if the indirect effects were equal or not and found the test to be not significant, thus giving no evidence that the indirect effects were different (B=.02, p=.698).

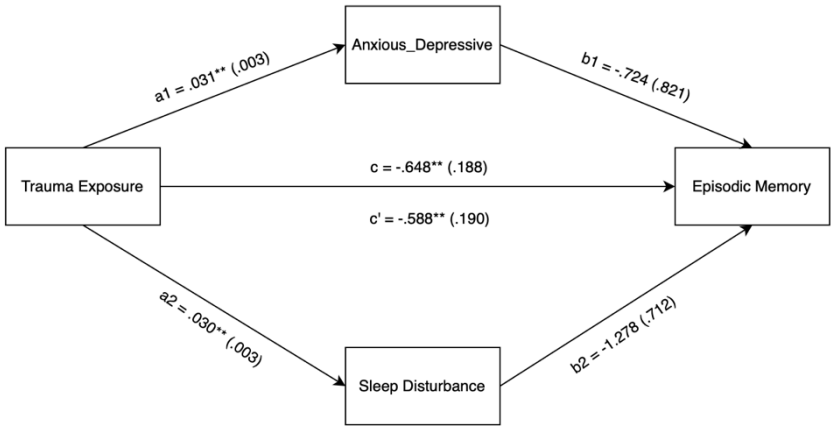
**Table 3**

*Multiple mediation model with beta coefficients, standard errors, z scores, and p values.*

| Effect | *β* | *SE* | *z* | *p* |
|---|---|---|---|---|
| Regressions | | | | |
| Trauma → Anxious-Depression | 0.03 | 0.00 | 7.54 | <.001 |
| Trauma → Sleep Disturbance | 0.03 | 0.01 | 6.50 | <.001 |
| Anxious-Depression → Episodic Memory | -0.72 | 0.82 | -0.89 | .375 |
| Sleep → Episodic Memory | -1.28 | 0.73 | -1.76 | .079 |
| Trauma → Episodic Memory | -0.59 | 0.20 | -2.88 | .004 |
| Covariances | | | | |
| Anxious-Depression and Sleep | 0.02 | 0.00 | 21.96 | <.001 |
| Variances | | | | |
| Episodic | 283.64 | 3.97 | 71.46 | <.001 |
| Anxious-Depression | 0.05 | 0.00 | 35.15 | <.001 |
| Sleep | 0.07 | 0.00 | 31.99 | <.001 |
| Indirect and total effects | | | | |
| Indirect Trauma → Anxious-Depression → Episodic | -0.02 | 0.03 | -0.87 | .383 |
| Indirect Trauma → Sleep → Episodic | -0.04 | 0.02 | -1.68 | .093 |
| Total effect | -0.65 | 0.20 | -3.17 | .002 |

**Figure 1**

*Trauma Exposure on Episodic Memory, mediated by Anxious-Depression and Sleep Disturbance*




*Note.* a1, a2, b1, b2, c, and c' are unstandardized regression coefficients and standard errors (in parenthesis) obtained by bootstrapping. The c path coefficient represents the total (direct and indirect) effect of trauma exposure on episodic memory. The c-prime path refers to the direct effect of the trauma exposure on episodic memory. Paths a1, a2, c, and c' were significant (**p<.01).

## Discussion

The purpose of the current study was to investigate how baseline trauma exposure impacts episodic memory performance two years later, while examining whether anxiety, depression, or sleep disturbance at year 1 mediate this relationship. The results show a significant total effect which is a summation of the total direct effect and total indirect effects; in other words, an increase in baseline trauma exposure has a significant direct association with a decrease in episodic memory performance two years later after accounting for both mediators. Additionally, we see that the indirect mediation effect of both anxious-depression and sleep disturbance was not significant, thus showing that memory impairment caused by increased trauma exposure exists despite accounting for anxiety, depression, and sleep

disturbance in year 1. The study also found the associations between baseline trauma and year 1 anxious-depression and sleep disturbance to be significant. In other words, while trauma may lead to anxiety, depression, and sleep disturbances, it is possible that its effect on memory is not due to any of these variables. Overall, the study's results support the hypothesis that trauma exposure significantly predicts episodic memory impairment and that this effect is apparent even after accounting for mediators. There may be some evidence of partial mediation as the effect of trauma on memory is slightly smaller when the mediators are accounted for, but it is still significant.

Consistent with previous studies (Claxton et al., 2021; Kascakova et al., 2020), we found a significant effect of trauma on anxious depression and a significant effect of trauma on sleep disturbances (Brindle et al., 2018). Considering the significant direct effect of trauma exposure on episodic memory, our study also allows room for further research to challenge some of the preexisting notions that trauma is not directly associated with memory (Terock et al., 2020; Jelinek et al., 2013; Rubin et al., 2008). As in previous work (Brewin et al., 2007), our study not only found there to be a relationship between trauma and memory but also established the temporal precedence of trauma exposure before episodic memory which was a limitation in some studies (Petzold & Bunzeck, 2022).

***Limitations***

While this study has several strengths including a large sample size, establishing temporal precedence, and comprehensive measures of constructs, we must also account for its limitations. This dataset includes twins, triplets, and siblings, which introduces non-independence between participants since relatives share genetic material. Such dependence between subjects needs to be accounted for in future research. Second, participants are nested within the 21 data collection sites, which also introduces a form of dependence since children from the same site might be more similar than children from a different site. Ideally, to

address this, we would account for site in our analyses. Third, the sample is not perfectly representative of the United States population since the sites were chosen out of convenience. The ideal way to address this is to use the post-stratification weights provided by the ABCD Study, which can be used to make the sample more representative of the U.S. population. Fourth, in the ABCD sample, we only have data available for three years, which is not only a narrow age range but also might not be enough time for memory impairment symptoms to develop. Fifth and finally, the dataset is also very skewed in terms of there being a much smaller proportion of trauma-exposed individuals, and thus, it might be helpful to conduct a similar study in a clinical population.

***Future Directions***

Based on the limitations, we can identify several future directions. It would be beneficial to examine a longer duration between time points in a clinical population to address the issues of few participants with trauma and a short duration to test for effects on memory. In future work, to target non-independence in the samples, more refined analyses could aim to cluster based on family ID and stratify based on site. To make the sample more representative, post-stratification weights can be applied using software like Mplus. If the direct effect of trauma on memory impairment is still significant, further research could explore whether this effect exists for other types of memory such as working memory or semantic memory, and if it changes based on the type of trauma endured. Research could also explore what features of trauma exposure are most closely related to memory impairment and if this impairment is long-term. Neuroimaging data could be integrated into future analyses to understand the pathways and brain regions that are related to memory and affected by trauma. Furthermore, experiments can be conducted to stimulate these brain regions to further understand trauma-related memory impairments. Finally, future work could design and test

interventions to target trauma symptoms to reduce memory impairment in the hope of preventing the long-term negative effects of memory deficits on functioning and wellbeing.

***Conclusion***

The literature so far has suggested that trauma predicts memory impairments to a certain extent, but why this happens has not been understood. By examining the mediating effect of anxiety, depression, and sleep disturbance, this study aimed to move the field one step closer to examining the specificity of the trauma-memory relationship, thus providing a basis to understand why memory is impacted after trauma. Another importance of this endeavor that makes it worth pursuing is its implications for intervention and diagnosis. There is much ambiguity about the consequences of trauma on memory, and while some of this has to do with the impact of trauma on the individual's brain and the variety of effects different kinds of trauma can have on memory, it is still worth exploring more thoroughly. A thorough analysis of trauma exposure and its impact on memory, while accounting for mediating variables, allows us to fill in a gap in the literature and potentially tailor treatments to survivors experiencing memory impairment symptoms. Such work may also have implications for the justice system, could aid in patient diagnosis, and has the potential to help improve pharmaceutical endeavors.

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