Associative Learning Difficulties following Childhood Trauma Exposure: A Transdiagnostic Risk Factor for Psychopathology

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Abstract

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Childhood trauma is associated with greater transdiagnostic risk for psychopathology. Smaller hippocampal volume is associated with both childhood trauma and multiples forms of psychopathology. We propose that childhood trauma may lead to impairments in associative learning, a basic function of the hippocampus, which in turn may increase general vulnerability for psychopathology. Children (n=84, 9-19 years, 55 exposed to trauma involving interpersonal violence) completed four behavioral tasks assessing associative learning of visual, auditory, contextual, and temporal information. The tasks involved facial stimuli with neutral, happy, and angry expressions, with the exception of temporal encoding. Participants also completed a structural MRI scan. A latent factor for general psychopathology ("p-factor")—representing co-occurrence of psychopathology symptoms across multiple internalizing and externalizing

domains—was estimated using confirmatory factor analysis. Childhood trauma exposure was associated with worse associative memory, and smaller hippocampal volume mediated this association. Difficulties with associative learning were observed in trauma-exposed children across different information types and irrespective of the emotional nature of the stimuli, suggesting that poor associative learning is not specific to threat-related cues. Associative memory improved with age for participants without trauma exposure, but not for participants exposed to trauma. Finally, lower associative learning performance was associated with higher scores on the general psychopathology factor and mediated the association of trauma with the p-factor. Broad hippocampus-dependent associative learning difficulties may be one transdiagnostic mechanism linking childhood trauma to psychopathology.

Keywords: childhood adversity, trauma, associative learning, hippocampus, p-factor

Associative Learning Difficulties following Childhood Trauma Exposure: A Transdiagnostic Risk Factor for Psychopathology

Exposure to childhood trauma is a transdiagnostic risk factor for psychopathology. Children exposed to trauma are more likely to develop anxiety, mood, behavior, and substance use disorders than children without trauma exposure, and there is little variation in the strength of these associations across different disorders (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012). Recent work suggests that childhood trauma is associated with higher scores on a transdiagnostic general psychopathology factor (or p-factor), which is a latent variable that represents the shared variance across symptoms spanning multiple disorder types (Caspi et al., 2014; Lahey, Krueger, Rathouz, Waldman, & Zald, 2017), with little residual associations with specific factors for internalizing or externalizing types of psychopathology (Caspi et al., 2014; Schaefer et al., 2018). Here we investigate a novel mechanism linking childhood trauma exposure to transdiagnostic psychopathology. Specifically, we hypothesize that exposure to trauma in childhood is associated with reduced hippocampal volume and impairments in basic functions of the hippocampus involving associative learning, which, in turn, may increase general vulnerability for psychopathology.

The negative consequences of trauma on the hippocampus have been observed in both animal models and human studies, with effects that are particularly dramatic following exposure early in life. In rodents, chronic early-life stress is associated with reduced dendritic branching and synaptic plasticity in hippocampal neurons and deficits in forms of memory that rely on the hippocampus that persist into adulthood (Brunson et al., 2005; Eiland, Ramroop, Hill, Manley, & McEwen, 2012; Ivy et al., 2010). These effects are mediated by excess levels of corticotropin-releasing hormone (CRH) and activation of CRH receptors in the hippocampus (Brunson,

Eghbal-Ahmadi, Bender, Chen, & Baram, 2001; Ivy et al., 2010). Consistent with these patterns in animal models, childhood trauma exposure is associated with smaller hippocampal volume in both children and adults in human studies (Hanson et al., 2015; Lambert, Sheridan, et al., 2017; McLaughlin, Weissman, & Bitran, 2019; Teicher, Anderson, & Polcari, 2012). Despite this well-replicated effect, the precise cognitive functions that are disrupted as a result of these changes in hippocampal structure following childhood trauma exposure are largely unknown.

One of the most well-established functions of the hippocampus is associative learning, a process that is necessary for forming coherent and detailed memories of personal events (for reviews, see Davachi, 2006; Eichenbaum, Yonelinas, & Ranganath, 2007; Lavenex & Amaral, 2000; Squire, 1992). Learning who and what were present during an event, where an event occurred, or the sequence of events over time are all examples of hippocampus-mediated associative learning. Specifically, the sensory cortex sends information from different sensory domains (e.g., visual, auditory, olfactory) to medial temporal lobe cortical regions, which integrate sensory information into representations of individual items (e.g., objects in perirhinal cortex, contexts in posterior parahippocampal cortex). Item information is then projected through the entorhinal cortex to the hippocampus, which binds co-occurring items or sequences of items together, across sensory domains (e.g., visual, auditory) and category membership (e.g., objects, contexts, people). Childhood trauma exposure may influence hippocampal function broadly in ways that contribute to associative learning difficulties across diverse forms of learning involving different types of information (e.g., visual, auditory, contextual, temporal). Given that episodic memory—such as memory of scenes and stimulus pairings—improves dramatically across childhood and adolescence (DeMaster & Ghetti, 2013; Ghetti, DeMaster, Yonelinas, & Bunge, 2010; Ofen, Chai, Schuil, Whitfield-Gabrieli, & Gabrieli, 2012; Rosen et al., 2018;

Selmeczy, Fandakova, Grimm, Bunge, & Ghetti, 2018), alterations in the structure and function of the hippocampus following childhood trauma exposure may lead to associative learning difficulties that become more pronounced with increasing age (Lambert et al., 2019).

However, an alternative possibility is that these deficits in associative learning are not general but instead occur primarily in the presence of threat cues. Enhanced threat-related processing has been consistently observed in children exposed to trauma (McLaughlin & Lambert, 2017). This is reflected in greater perceptual sensitivity, faster attention orienting, and longer sustained attention to threatening faces among violence-exposed children than those who have never experienced violence (Pollak, Cicchetti, Hornung, & Reed, 2000; Pollak & Kistler, 2002; Pollak & Sinha, 2002; Pollak & Tolley-Schell, 2003; Shackman, Shackman, & Pollak, 2007). These information processing patterns could limit visual processing of surrounding episodic details in situations that involve the potential for danger and interfere with associative encoding. Consistent with this possibility, violence-exposed children exhibited poor memory for contextual information presented behind angry faces, but not behind happy or neutral faces (Lambert, Sheridan, et al., 2017). These reductions in contextual memory were associated with lower hippocampal activation and greater hippocampus-ventrolateral prefrontal cortex (vlPFC) functional connectivity during encoding in the presence of angry faces (Lambert, Sheridan, et al., 2017). Given the role of vlPFC in attention orienting and selective attention (Bishop, 2008; Shiba, Santangelo, & Roberts, 2016), this pattern suggests that attentional narrowing on the threat cue may have occurred at the expense of hippocampus-dependent processing of broader contextual information. If this pattern generalizes to other forms of associative learning, difficulties with associative memory among children exposed to trauma would be expected to emerge specifically in the presence of threat cues but not otherwise.

Much of the theoretical and empirical research on the role that the hippocampus and associative learning play in psychopathology has focused on post-traumatic stress disorder (PTSD). Difficulty recalling where and when a trauma occurred could lead to fear to trauma cues in safe places and at safe times, and difficulty recalling who and what were present during the trauma could lead to fear to non-threatening cues that resemble those present during the trauma (Brewin, 2001; Lambert & McLaughlin, 2019; Liberzon & Abelson, 2016). Meta-analyses have found that adults with PTSD have smaller hippocampal volume, worse associative memory, and overly general autobiographical memory compared to those exposed to trauma without PTSD or without trauma exposure (Karl et al., 2006; Kitayama, Vaccarino, Kutner, Weiss, & Bremner, 2005; Lambert & McLaughlin, 2019; Logue et al., 2018; Moore & Zoellner, 2007; Ono, Devilly, & Shum, 2016). We argue that difficulties with associative learning could contribute to the emergence of multiple forms of internalizing and externalizing problems, not just PTSD. Problems with associative learning could cause an inability to draw on specific memories in appraising different situations, thus leading to inappropriate or ineffective responses characteristic of multiple disorders (Kashdan & Rottenberg, 2010). In depression, difficulty recalling successful problem-solving strategies used in similar situations in the past could make it more difficult to generate effective problem solving strategies in current stressful situations (Evans, Williams, O'loughlin, & Howells, 1992; Goddard, Dritschel, & Burton, 1996). Meta-analyses have found that adults with depression have smaller hippocampal volume, difficulty remembering locations of stimuli, and overly general autobiographical memory compared to adults without depression (Campbell, Marriott, Nahmias, & MacQueen, 2004; Cole, Costafreda, McGuffin, & Fu, 2011; Rock, Roiser, Riedel, & Blackwell, 2014; Sumner, Griffith, & Mineka, 2010; Videbech & Ravnkilde, 2004). In externalizing disorders, difficulty recalling where, when,

or around whom a behavior was inappropriate or maladaptive could make it more difficult to select appropriate behaviors in a current situation. Lower IQ and executive functioning difficulties have been consistently observed among youth with externalizing disorders (Keyes, Platt, Kaufman, & McLaughlin, 2017; Schoemaker, Mulder, Deković, & Matthys, 2013; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). However, we argue that externalizing disorders could relate to a broader set of cognitive impairments that also involve basic associative learning processes.

In this study, we predicted that difficulties in hippocampus-dependent associative learning may be a transdiagnostic mechanism linking childhood trauma to internalizing and externalizing psychopathology. Specifically, we expected that children exposed to violence would perform worse on associative memory tasks compared to children without violence exposure, and smaller hippocampal volume would explain the relationship between childhood violence exposure and worse associative memory. We also expected that associative memory difficulties would occur broadly for different domains of information (visual, auditory, contextual, and temporal) and regardless of the emotional nature of the stimuli being learned (neutral, happy, and angry faces) but may be most pronounced in the presence of threat. Regarding age-related variation, violence-related differences in associative memory may become more pronounced with age. Given that the hippocampus is sensitive to stress early in life, we expected that childhood violence exposure would have specific impacts on hippocampus-based functions like associative learning independent from other cognitive domains that are not supported by the hippocampus but could influence task performance in a general way. Thus, we expected that this pattern of result would remain even after adjustment for inhibitory control, a type of executive function supported by the fronto-parietal network (Munakata et al., 2011;

Zhang, Geng, & Lee, 2017) that has broad influences on learning and behavior but does not rely on the hippocampus. Finally, we expected that worse associative memory would be associated with higher p-factor and would mediate the association of childhood violence exposure with this transdiagnostic risk.

Methods

Sample

Children and a parent/guardian were recruited from a larger longitudinal study examining violence exposure, neural systems underlying emotion regulation, and psychopathology (Weissman, Bitran, et al., 2019). The sample was recruited in Seattle, WA between January 2015 and June 2017. Children were recruited from schools, after-school and prevention programs, adoption programs, food banks, shelters, parenting programs, medical clinics, and the general community. To ensure variation in exposure to violence, recruitment targeted neighborhoods with high levels of violent crime, clinics that serve a predominantly low socioeconomic status area, and agencies that support families exposed to violence (e.g., domestic violence shelters and programs for parents mandated to receive intervention by Child Protective Services). Children were excluded if they had an IQ < 80, a pervasive developmental disorder, active psychotic symptoms or mania, active substance abuse, or the presence of safety concerns. This study focused on a sub-set of the 130 children who completed a follow-up visit approximately two years after the baseline visits.

For the present study, we recruited children from this larger sample to participate in an additional study, with a particular focus on recruiting children who had experienced violence. Based on the grant funding for this study and time constraints related to our lab moving to another university, we stopped recruitment at 84 participants, 55 children with violence exposure

and 29 children without violence exposure. The sample was 9-19 years (M = 14.10 years, SD = 2.79 years; 49 male). See Table 1 for demographic characteristics of the final sample according to violence exposure; participants with violence exposure were matched to control participants on sex and age. The Institutional Review Board at the University of Washington approved all procedures. Written informed consent was obtained from legal guardians, and children provided written assent.

Violence Exposure

We focus on childhood trauma exposure involving interpersonal violence, which has particularly strong associations with psychopathology (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012). We defined violence exposure as child- or parent-reported physical or sexual abuse or child-reported direct witnessing of domestic violence (Weissman, Jenness, et al., 2019). We used a multi-method, multi-reporter approach to assess violence exposure. Children completed two interviews with a trained member of our research team. The Childhood Experiences of Care and Abuse (CECA) Interview assesses experiences with caregivers, including physical and sexual abuse (Bifulco, Brown, & Harris, 1994). We modified the interview to ask parallel questions about witnessing domestic violence (i.e., directly observing violence directed at a caregiver). Inter-rater reliability for maltreatment reports is excellent, and validation studies suggest high agreement between siblings on maltreatment reports (Bifulco, Brown, Lillie, & Jarvis, 1997). The Violence Exposure Scale for Children-Revised (VEX-R) assesses frequency of exposure to and witnessing violence in the home, school, and neighborhood (Raviv et al., 2001; Raviv, Raviv, Shimoni, Fox, & Leavitt, 1999). The VEX-R demonstrates good reliability and has been validated with children as young as second grade (Raviv et al., 2001, 1999).

Children also completed two self-report measures. The Childhood Trauma Questionnaire (CTQ) assesses the frequency of maltreatment during childhood, including physical and sexual abuse (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997). The CTQ has excellent psychometric properties including internal consistency, test-retest reliability, and convergent and discriminant validity with interviews and clinician reports of maltreatment (Bernstein et al., 1997, 1994). The UCLA Posttraumatic Stress Disorder Reaction Index (UCLA PTSD-RI) includes a trauma screen that assesses exposure to numerous traumatic events, including physical and sexual abuse and domestic violence (Steinberg, Brymer, Decker, & Pynoos, 2004). The UCLA PTSD-RI has good internal consistency and convergent validity (Steinberg et al., 2013).

Caregivers completed two self-report measures. The Juvenile Victimization Questionnaire (JVQ) lifetime caregiver report assesses multiple forms of victimization, including physical and sexual abuse and witnessing violence (Finkelhor, Hamby, Ormrod, & Turner, 2005). The JVQ has excellent psychometric properties, including test-retest reliability and construct validity (Finkelhor et al., 2005). Caregivers also completed the trauma screen of the UCLA PTSD-RI.

Children were classified as experiencing physical or sexual abuse if abuse was endorsed by the child on the CECA interview, UCLA PTSD-RI trauma screen, or above the validated threshold on the CTQ (Walker et al., 1999) or reported by the parent on the JVQ or UCLA PTSD-RI trauma screen. Domestic violence was assessed by child-report only on the CECA interview, VEX-R, and UCLA PTSD-RI. A total of 55 children (65.5%) had experienced violence.

Psychopathology

Psychopathology symptoms were assessed using child- and/or parent-report. The Children's Depression Inventory-second edition (CDI-2) was used to assess symptoms of depression (Kovacs, 2011). The CDI-2 is a recently revised version of the CDI, a widely used self-report measure of depressive symptoms in children and adolescents (Kovacs, 1992). The CDI has demonstrated good reliability and validity among children and adolescents (Craighead, Smucker, Craighead, & Ilardi, 1998).The CDI demonstrated excellent internal consistency in our sample (α = .89).

Anxiety symptoms were assessed with the Screen for Child Anxiety Related Emotional Disorders (SCARED), which includes five symptom domains (panic/somatic, generalized anxiety, separation anxiety, social phobia, and school phobia) (Birmaher et al., 1997). The SCARED has sound psychometric properties (Birmaher et al., 1999, 1997) and had excellent internal consistency in our sample (α = .93).

Child- and parent-report versions of the UCLA PTSD-RI were used to assess PTSD symptoms according to DSM-IV criteria (Steinberg et al., 2004). The UCLA PTSD-RI has sound psychometric properties (Steinberg et al., 2013), and had excellent internal consistency in our sample (α = 0.90). Items were summed to generate a total symptom severity score, and the higher of the child- or parent-reported score was used.

Child-report on the Youth Self Report (YSR) and parent-report on the Child Behavior Checklist (CBCL) were used to assess externalizing symptoms (Achenbach, 1991). The higher raw score between the YSR and CBCL was used from the attention problems, rule breaking behavior, and aggressive behavior subscales. The YSR and CBCL are among the most widely used measures of youth emotional and behavioral problems.

We used each of these indicators to define a transdiagnostic psychopathology factor, or p-factor. To do so, we estimated p-factor scores as previously described in this sample (Weissman, Bitran, et al., 2019) using a standard bi-factor model (Caspi et al., 2014; Lahey et al., 2017) including multiple forms of internalizing and externalizing psychopathology. Following prior studies, we tested two models: correlated-factors and bi-factor models (Caspi et al., 2014; Lahey et al., 2017). In the correlated factors model, internalizing and externalizing latent constructs are defined from manifest indicators. In the bi-factor model, the latent p-factor is regressed onto the manifest indicators followed by internalizing and externalizing latent constructs regressed onto the residualized variance in these indicators (Supplemental Figure 1). These models were tested using confirmatory factor analyses in MPlus 8.1 with the robust maximum likelihood estimator, which is robust to non-normality of observed indicators as in the present study. Model fit was assessed using the Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC) and the Sample Adjusted BIC. Both models had similar model fit with indicators loading significantly (all p's<.001) with slightly better fit for the bi-factor model (Supplemental Table 1). In the present analyses, we used the bi-factor model because we were interested in creating an indicator of transdiagnostic psychopathology, and it is the most commonly used general factor model of psychopathology (Caspi et al., 2014; Lahey et al., 2017). Our goal here was not to estimate the factor structure of psychopathology in our sample, but to reduce the multiple indicators of internalizing and externalizing problems into a single score reflecting the level of transdiagnostic psychopathology for each participant. We extracted the factor scores for the p-factor latent construct for subsequent analyses.

Associative Learning Tasks

Participants completed four tasks designed to assess associative learning across visual, auditory, contextual, and temporal domains. Each task involved an encoding phase and tests for associative and item memory, completed approximately 20 minutes after encoding. Different stimuli were used for each task. We created two versions of each task based on different stimuli pairings and sequences of stimuli. The task versions and order of presentation of the four tasks were counterbalanced across violence-exposed and control participants. With the exception of the task assessing memory for temporal sequences, all tasks shared a common structure and assessed associative learning of stimulus pairings. We first describe this general structure, and then describe the specific stimuli used in each task in more detail.

During encoding, participants were presented with pairs of stimuli across 6 blocks. Each trial presented a stimulus pair for 2000ms. Each pair was presented 6 times during the encoding period, once per block. Pairs were presented randomly within each block. The inter-trial interval was 2000ms, and participants were given a 10s break between each block. Each pair included a face with a neutral, happy, or angry expression as one of the stimuli. Following encoding, participants engaged in an unrelated task (e.g., completing survey measures) for at least 20 minutes prior to the memory test.

The memory test occurred in two blocks, one assessing memory of pairs and the other assessing memory of items that comprised the pairs. During the pair memory test, participants were presented with correct and incorrect pairings of items and indicated whether they had seen the pair during encoding. All stimuli were familiar (i.e., had previously been presented as part of a pair during encoding). To respond correctly, participants therefore needed to remember the specific pairings between stimuli observed during encoding and could not simply rely on familiarity of items. During the item memory test, participants were presented with single items

that had previously been part of a pair during encoding and novel items and were asked if they had seen the items during encoding. For both tests, participants had unlimited time to respond, the inter-trial interval was 1000ms, and participants were given a 10s break after 20 or 30 trials depending on the task. Pairs and items were presented randomly within each block. Participants completed the item memory test before the pair memory test, and emotion category was presented evenly across pair and item trials.

Performance on the pair and item memory tests were assessed using discrimination sensitivity (*d′*), which was calculated using the following formula (Stanislaw & Todorov, 1999):

$$d' = \Phi^{-1}(hits) - \Phi^{-1}(false\ alarms)$$

We first calculated the hit rate (responding "seen before" on correct trials / total number of correct trials) and the false alarm rate (1 – responding "not seen before" on incorrect trials / total number of incorrect trials). If either the hit rate or false alarm rate was 0 or 1, we converted the score to half of the distance between the next best or worse possible score (e.g., a hit rate or false alarm rate of 30/30 were converted to 29.5/30 and a hit rate or false alarm rate of 0/30 were converted to 0.5/30). Scores were then standardized using an inverse phi function, which converts accuracy scores to the portion of the normal distribution that lies to the left of the z-score. We calculated *d′* by subtracting the standardized false alarm rate from the standardized hit rate. A higher *d′* score indicates a greater ability to distinguish signal (correct trials) from noise (incorrect trials), or a greater distance between the mean of the signal distribution and the mean of the noise distribution in standard deviation units (Stanislaw & Todorov, 1999). For pair memory performance on each task, we calculated *d′* overall (regardless of emotion) and

separately for trials involving faces with different emotional expressions (neutral, happy, and angry).

Visual PAL task. The visual PAL task assessed the ability to pair two visual cues (Figure 1). A similar task has been shown to recruit the hippocampus in children and adolescents (Lambert et al., 2019; Rosen et al., 2018). Participants encoded 30 pairs made up of 30 faces (10 unique people, with neutral, happy, and angry expressions) (Tottenham et al., 2009) and 30 objects that reflected a variety of activities (e.g., book, bike, soccer ball). Participants were instructed that the emotional expression on the face reflected how the person felt about the activity (i.e., a neutral face meant the person did not like or dislike the activity, a happy face meant the person liked the activity, and an angry face meant the person did not like the activity) and to remember the pairings. Participants were asked to indicate whether a dot that appeared on a white background for 500ms between the pair presentation and the ITI was on the right or left side of the screen. The dot appeared on 18 trials randomly interspersed throughout the encoding phase. This behavioral response was recorded to ensure participants were paying attention during the encoding phase.

The memory test included 90 pair trials (30 correct and 60 incorrect pairings) and 90 item trials (60 familiar and 30 novel items).

Audio-visual PAL task. The audio-visual PAL task assessed the ability to integrate auditory and visual information. Similar audio-visual integration tasks engage the hippocampus (Gonzalo, Shallice, & Dolan, 2000) and are sensitive to hippocampal damage (Vargha-Khadem et al., 1997) in adults. Participants encoded 30 pairs comprised of faces and voices (30 unique people and voices, 10 neutral, 10 happy, and 10 angry). Faces were drawn from the standardized Interdisciplinary Affective Science Laboratory (IASLab) Face Set (www.affective-science.org).

Each recording was of a person saying the phrase: “What are you doing right now?” in either a neutral, happy, or angry tone. We used Audacity, an audio editor software, for volume normalization. Face-voice pairings were matched on sex and emotion. Participants were asked to remember each person’s voice. On 18 trials participants were asked whether the person they just saw was male or female after the pair presentation as an attention check.

The memory test included 60 pair trials (30 correct and 30 incorrect face-voice pairings) and 90 item trials (60 familiar and 30 novel items).

Context encoding task. The context encoding task assessed the ability to bind background contexts with foreground cues, has previously been used with children and adolescents, and recruits the hippocampus (Lambert, Sheridan, et al., 2017) (Figure 2). During encoding, participants completed a delayed match-to-sample working memory task. Each trial involved a context-face pairing (2000ms), delay (2000ms), and probe (2000ms). Pairs were neutral, happy, and angry faces embedded in realistic background scenes. Participants were instructed to focus on the faces and were not instructed to focus on the scenes. Probes were faces without scenes. Participants were asked if the probe matched the face they saw before the delay. There were 45 correct trials and 45 incorrect trials. This design ensured that context was not the focus of the task and remained in the background. A total of 15 pairs were used (5 unique people, each feeling neutral, happy, and angry) drawn from a standardized stimulus set (Tottenham et al., 2009) and 15 contexts (5 outdoor street scenes, 5 outdoor nature scenes, and 5 indoor scenes).

The memory test included 60 pair trials (15 correct and 45 incorrect face-context pairings) and 60 item trials (30 familiar and 30 novel items).

Temporal encoding task. The temporal encoding task measured the ability to remember the order of events. We created a developmentally appropriate version of a task that evaluated

the ability to retrieve the sequence of events using a naturalistic paradigm and recruited the hippocampus in adults (Lehn et al., 2009). During encoding, participants watched the first 40min of a movie and were instructed to remember as much of the movie as possible, without specifying which aspect of the movie they would be asked to recall later. Participants watched the 1975 version of Escape to Witch Mountain (Hough, 1975), which is publicly available and appropriate for all ages. We selected this movie because it involved real people and events that unfold over days and was unlikely to have been seen by participants in our study.

The memory test was structured similarly as the other three tasks, with several differences. The sequence test occurred over 30 trials. Each trial presented four images, one in each of the four quadrants of the computer screen. Participants indicated the order in which they saw the images during the movie. All images had previously been seen during encoding. Images in each quadrant were distinct, and there was no logical ordering among them. During the item memory test, participants were presented with still images alone and were asked if they saw the image during the movie. There were 30 trials (15 familiar and 15 novel scenes). Images were screen shots taken at approximately 15s intervals throughout the movie. The novel images were taken from the last portion of the movie that the participants did not watch and were inspected to make sure that they appeared distinct (e.g., different location, time of day, characters, objects) from the familiar images.

Accuracy was calculated by dividing the correct responses by the total number of responses, with a higher number indicating higher accuracy. For the temporal sequence memory test, participants could receive a maximum score of 4 on each trial—1 point for each image in the correct position in the sequence. We could not calculate *d*’ for the temporal sequence test as there were no incorrect trials to use to calculate a false alarm rate.

Inhibitory Control

The arrows task, a subtest of the Developmental Neuropsychological Assessment II, measures inhibition of an automatic response (Brooks, Sherman, & Strauss, 2009). Participants viewed several rows of black and white arrows pointing either up or down. In the baseline trial, participants were asked to say the direction that each arrow was pointing. In the inhibition trial, participants were asked to say the opposite direction that each arrow was pointing. The time taken to complete the baseline trial was subtracted from the time required to complete the inhibition trial. Larger differences indicated worse inhibitory control.

Structural MRI Acquisition and Processing

Scanning was performed on a 3T Phillips Achieva scanner at the University of Washington Integrated Brain Imaging Center using a 32-channel head coil. T1-weighted MPRAGE volumes were acquired (repetition time = 2530 ms, TE=3.5ms, flip angle=7°, FOV=256×256, 176 slices, in-plane voxel size=1mm^3). Measures of left and right hippocampal volume and total intracranial volume were obtained using automatic segmentation in FreeSurfer 5.3. T1 images and subcortical segmentation were visually inspected. No manual edits were performed on subcortical segmentations.

Statistical Analysis

All participants performed above chance on the attention checks; thus, nobody was excluded due to low memory performance. One participant was excluded from the visual PAL item memory test due to experimenter error during task administration. Two participants did not complete the temporal encoding task. One participant was excluded from analyses involving hippocampal volume due to artifacts from excessive head movement, and nine participants did not complete the MRI scan. One participant did not complete psychopathology measures.

We first examined whether the association of violence exposure with pair memory varied as a function of the type of information being learned and whether that information was threatening or not. Specifically, we used a repeated-measures analysis of variance (ANOVA) with task type (visual, audio-visual, and context) and facial emotion (neutral, happy, and angry) as within-subjects factors and violence exposure as a between-subjects factor. Because the association did not vary based on task or emotion, we ran all further analyses on an aggregate of pair memory. We computed this aggregate by averaging *d*' overall across the three tasks for each participant. We computed an item memory aggregate in the same way.

We examined whether pair memory differed between participants with and without violence exposure using a univariate ANOVA. To examine whether the association of violence exposure with pair memory varied by age and sex, we examined whether violence and these factors interacted in predicting pair memory. We estimated simple slopes using the interActive data visualization tool to probe the interactions (McCabe, Kim, & King, 2018). Finally, we examined whether the association of violence exposure with performance differed based on whether participants were asked to remember pairings of items or individual items using a repeated measures ANOVA with test type (pair, item) as a within-subjects factor and violence exposure as a between-subjects factor. Associations of violence exposure with performance on the temporal encoding task were examined using the same analytic approach. Finally, we conducted a sensitivity analysis to evaluate whether associations of violence exposure with memory performance persisted after adjustment for inhibitory control ability on the arrows task.

We next used the same analytic approach to examine whether hippocampal volume differed based on violence exposure and whether this association varied by age and sex. We also used linear regression to examine whether hippocampal volume was associated with memory

performance. We examined whether the association of violence exposure with memory performance was mediated by variation in hippocampal volume. To do so, we used a standard test of statistical mediation to test significance of indirect effects using a bootstrapping approach that provides confidence intervals for the indirect effects (Hayes, 2018).

To evaluate associations of memory with transdiagnostic psychopathology, we used linear regression to examine whether associative and item memory performance from the pair and temporal tasks were associated with p-factor scores. We also tested whether memory performance mediated the association of violence exposure with p-factor scores.

Age and sex were included as covariates in all analyses. Total intra-cranial volume was added as an additional covariate in analyses involving hippocampal volume.

Results

Task Performance

We first describe memory performance in the entire sample. Pair and item memory, aggregated across the three tasks, were both above chance (i.e., mean d' scores were positive): pair memory (M = 1.20, SD = 0.70); item memory (M = 2.07, SD = 0.71). Pair memory improved with age, $\beta = 0.45$, $p < .001$, but did not differ between male and female participants, $p = .25$. Performance differed as expected for pair and item memory, $F(1, 81) = 391.50$, $p < .001$, with better memory for items than pairs.

For the temporal encoding task, sequence memory (M = 0.78, SD = 0.10) and item memory (M = 0.94, SD = 0.05) were above chance (i.e., mean accuracy was above .50). Sequence memory was not associated with age, $p = .17$, or sex, $p = .98$. Performance differed between the two memory tests, $F(1, 79) = 220.33$, $p < .001$, with higher scores for item than sequence memory.

Violence Exposure and Memory Performance

The association of violence exposure with pair memory did not differ based on task (i.e., visual, auditory, or contextual), $p = .07$, or facial emotion type (i.e., angry, happy, or neutral), $p = .51$. To reduce multiple comparisons, we ran all further analyses on the pair and item memory aggregates. However, for completeness, we show pair memory performance by violence exposure for each task separately and for each emotion condition within each task in the supplement (Supplemental Table 2; Supplemental Figure 2).

Violence exposure was associated with lower pair memory, $F(1,80) = 12.57$, $p = <.001$ (see Table 2). The association of violence exposure with pair memory varied marginally as a function of age, $F(1,78) = 3.64$, $p = .060$, but not by sex, $p = .44$. We next estimated the simple slopes of the association between age and pair memory for participants with and without violence exposure (Figure 3). For children and adolescents without violence exposure, pair memory increased significantly with age, b = 0.12, 95% CI = [0.08, 0.17], but no age-related improvement in pair memory occurred in children exposed to violence, b = 0.04, 95% CI = [-0.06, 0.15].

Participants exposed to violence also had lower accuracy for remembering sequences of events than participants without violence exposure, $F(1,78) = 7.86$, $p = .006$. This association did not vary based on age, $p = .91$, or sex, $p = .16$.

The association of violence exposure with memory performance did not differ based on whether participants were asked to remember associations of items or individual items on the tasks involving pairs of stimuli, $p = .96$, or temporal sequences, $p = .14$. In fact, violence exposure was also associated with lower memory of items making up the pairs, $F(1,80) = 11.85$, $p <.001$, and individual scenes from the temporal task, $F(1,78) = 5.24$, $p = .025$.

Sensitivity analysis. In the next analysis, we evaluated associations of violence exposure with memory performance while controlling for inhibitory control ability on the arrows task. The results were unchanged. Specifically, there were no interactions between violence and task type, $p = .21$, or emotion type, $p = .47$, on pair memory performance. Thus, all further results involve the pair and item memory aggregates. Children exposed to violence performed worse on the pair memory tasks than children without violence exposure, $F(1,70) = 10.03$, $p = .002$. This association varied with age, $F(1,68) = 5.12$, $p = .027$, but not by sex, $p = .81$. Children exposed to violence also exhibited worse sequence memory than children without violence exposure, $F(1,68) = 6.64$, $p = .012$. This association did not vary based on age or sex, *p's* = .10-79.

Finally, there was no violence x test type (associative, item) interaction on performance on the pair or temporal tasks, *p's* = .20-.97. Indeed, violence exposure was also associated with lower item memory on the pair tasks, $F(1,70) = 10.81$, $p = .002$, and on the temporal task, $F(1,68) = 4.40$, $p = .040$.

Violence Exposure, Hippocampal Volume, and Memory Performance

Violence exposure was associated with bilateral hippocampal volume, $F(1,69) = 11.25$, $p = .001$ (Figure 4A). Participants exposed to violence had smaller hippocampal volume (M = 8310.91, SD = 721.93) than those without exposure to violence (M = 9041.81, SD = 762.54). The association of violence exposure with hippocampal volume did not vary by age, $p = .49$, or sex, $p = .27$.

Smaller hippocampal volume predicted worse pair memory, $\beta = 0.30$, $p = .007$ (Figure 4B), but not sequence memory, $p = .46$, or item memory on the pair or temporal tasks, *p's* = .07- .57. Smaller hippocampal volume mediated the association of violence exposure with pair memory (indirect effect = -.12, 95% CI: -0.3293 to -0.0145) (Figure 4C).

Violence Exposure, Memory Performance, and Psychopathology

Violence exposure was associated with higher p-factor scores, $F(1, 79) = 18.59$, $p \leq .001$. See Supplemental Table 3 for associations of pair memory with each individual psychopathology indicator that was used to define the p-factor. Lower pair memory was associated with higher p-factor scores, $\beta = -0.35$, $p = .004$ (Figure 5), as was lower sequence memory, $\beta = -0.33$, $p = .002$. Lower memory of items that made up the pairs was also associated with higher p-factor, $\beta = -0.29$, $p = .018$, but memory of individual scenes on the temporal task was not, $p = .06$.

The association of violence exposure with p-factor was not mediated by pair memory (95% CI: -0.0067 to 0.3188) but was mediated by sequence memory (indirect effect = .13, 95% CI: 0.0184 to 0.3249). Memory of items that made up the pairs did not mediate the association of violence exposure with p-factor (95% CI: -0.0403 to 0.2668).

Discussion

Exposure to childhood trauma is associated with elevated risk for psychopathology that spans multiple internalizing and externalizing problems (Caspi et al., 2014; Keyes et al., 2012; Schaefer et al., 2018). Here we investigated the hypothesis that poor associative learning may be a functional consequence of the well-established reductions in hippocampal volume among children exposed to trauma that may, in turn, be associated with greater transdiagnostic psychopathology. Consistent with this hypothesis, children exposed to violence performed worse on associative memory tasks compared to children without violence exposure, and smaller hippocampal volume explained this association. Difficulties were broad, occurring for multiple types of information and regardless of whether a threat cue was present or not. We also observed age-related variation, such that children exposed to violence did not exhibit associative memory improvements with increasing age displayed by children without violence exposure. Importantly,

childhood violence exposure was associated with associative memory difficulties independent of other cognitive abilities that contribute broadly to task performance, suggesting that these effects are specific to functions subserved by the hippocampus. Finally, worse associative memory was associated with greater transdiagnostic risk for psychopathology and mediated the association of childhood trauma with the general psychopathology factor. Hippocampus-dependent associative learning difficulties may therefore be one transdiagnostic mechanism linking trauma exposure in childhood to psychopathology.

Animal research documents the toxic and lasting effects of early-life stress on hippocampal neurons (Brunson et al., 2005; Eiland et al., 2012; Ivy et al., 2010; Rice, Sandman, Lenjavi, & Baram, 2008), and childhood trauma exposure is associated with smaller hippocampal volume in humans (Hanson et al., 2015; Lambert, Sheridan, et al., 2017; McLaughlin et al., 2019; Teicher et al., 2012). Difficulties with associative learning could be one functional consequence of altered hippocampal development following childhood violence exposure. Three findings from our study support this hypothesis. First, children exposed to violence exhibited worse associative memory than children who had never experienced violence. Childhood violence exposure was also associated with smaller hippocampal volume, which in turn was associated with worse associative memory and mediated the link between violence exposure and associative memory. This finding builds upon a prior study showing that children exposed to violence had smaller hippocampal volume, which was in turn associated with worse memory of faces paired with contexts (Lambert, Sheridan, et al., 2017).

Second, associative memory difficulties occurred in multiple information processing domains, including visual, audio-visual, contextual, and for temporal sequences of events. Impairments in associative learning of face-object and face-context pairs have been observed in

children exposed to violence in prior research (Lambert et al., 2019; Lambert, Sheridan, et al., 2017). We extend this work by documenting impairments in associative learning for audio-visual and temporal information as well. This domain-general pattern suggests that some type of disruption is occurring in the integration of different types of information in the hippocampus. If the impairment reflected problems in processing specific types of sensory information in sensory cortex, then associative learning difficulties may have been more pronounced on tasks involving certain types of sensory information (e.g., auditory versus visual). Similarly, if the impairment reflected problems in forming specific item representations in medial temporal lobe cortical areas, then associative learning difficulties may have been more pronounced on tasks involving certain item types (e.g., contexts versus objects).

Third, associative learning difficulties were not conditional on the presence of threat. The degree of associative memory impairment in children exposed to violence did not vary depending on whether the pairs involved neutral, happy, or angry faces. Our findings are consistent with prior work showing that children exposed to violence exhibit difficulties with associative learning regardless of the presence or absence of threat. In one study, children exposed to violence exhibited less hippocampal activation during encoding of face-object pairs compared to children who never experienced violence, and less hippocampal activation was associated with worse associative memory (Lambert et al., 2019). Importantly, these results did not vary depending on whether the face was angry, happy, or neutral (Lambert et al., 2019). In another study, violence exposure was associated with worse memory for pairs of neutral shapes in young children (Rosen, Meltzoff, Sheridan, & McLaughlin, 2019). Thus, it is unlikely that attentional narrowing on threat cues is the only explanation for associative memory difficulties. Instead, associative memory difficulties following childhood violence exposure may reflect

impairment in basic learning functions of the hippocampus that manifests regardless of the emotional nature of the situation.

Given that we focused on violence exposure occurring during a developmental window when episodic memory ability improves dramatically (DeMaster & Ghetti, 2013; Ghetti et al., 2010; Ofen et al., 2012; Rosen et al., 2018; Selmeczy et al., 2018), we examined whether the association of violence exposure with associative memory changed with age in our sample. Children exposed to violence did not exhibit age-related improvements in pair memory performance characterized by children not exposed to violence. This pattern may emerge if violence exposure influences hippocampal development and the hippocampus increasingly contributes to improvements in associative learning with age. Indeed, some studies show developmental variation in the contribution of the hippocampus or of different hippocampal sub-regions to episodic memory (DeMaster & Ghetti, 2013; DeMaster, Pathman, & Ghetti, 2013; Selmeczy et al., 2018). This pattern may also reflect the impact of childhood violence exposure on the development of fronto-parietal cortical regions that are involved in top-down executive control (Curtis & D'Esposito, 2003; Feredoes, Heinen, Weiskopf, Ruff, & Driver, 2011; Nelson et al., 2000; Peverill, McLaughlin, Finn, & Sheridan, 2016; Sakai, Rowe, & Passingham, 2002; Todd & Marois, 2004) and exhibit age-related differences in their contribution to episodic memory (DeMaster & Ghetti, 2013; DeMaster et al., 2013; Ofen et al., 2012; Selmeczy et al., 2018). Indeed, in prior work, we show that violence-exposed children exhibit difficulties remembering face-object pairs that become more pronounced with age, along with atypical recruitment of fronto-parietal regions during encoding that also became more noticeable with age (Lambert et al., 2019).

We have argued that childhood trauma may have specific impacts on hippocampus-based functions like associative learning given that the hippocampus is particularly sensitive to stress early in life. To test this, we controlled for inhibitory control, a type of executive function that contributes broadly to cognitive performance but is subserved by fronto-parietal cortical regions and not the hippocampus (Munakata et al., 2011; Zhang et al., 2017). Even after adding this control, the associations of childhood violence exposure with associative memory difficulties remained statistically significant, which would suggest that the pathway linking childhood violence to associative learning is specific and not merely a symptom of a broader impairment in cognitive function.

We next hypothesized that these associative learning difficulties would explain the increased general risk for psychopathology following childhood violence exposure. Childhood violence exposure was associated with higher p-factor scores, replicating prior work (Caspi et al., 2014; Schaefer et al., 2018). Worse memory of pairs and sequences was associated with higher p-factor scores, and worse memory of sequences mediated the association of childhood violence exposure with higher p-factor scores. We therefore show that associative learning impairments may contribute non-specifically to a wide range of psychopathology symptoms, which broadens existing work documenting associative learning deficits in people with PTSD (Lambert & McLaughlin, 2019). While we have speculated that problems with associative learning could cause an inability to draw on specific memories in appraising different situations thus leading to inappropriate or ineffective responses characteristic of multiple disorders (Kashdan & Rottenberg, 2010), further research is needed to test the pathways through which poor associative memory could influence different forms of psychopathology. Identifying transdiagnostic mechanisms underlying psychopathology could help develop novel treatments

that could address a wide range of mental health problems in children exposed to trauma. For example, a psychotherapy approach that helps patients develop or recall specific memories of relevant past experiences could target the mechanisms identified here and potentially have utility for reducing symptoms from a wide range of disorders. For those with PTSD and anxiety, recalling where the trauma took place, who or what was present during the trauma, or the sequence of events during the trauma—already a component of exposure-based treatments (Foa, Chrestman, & Gilboa-Schechtman, 2008; Foa, Hembree, & Rothbaum, 2007)—could help reduce generalization of fear. In depression, recalling past experiences where effective problem-solving strategies were used or positive interpersonal interactions occurred could be a helpful reference for problem-solving in current stressful situations and improve negative mood. For people with externalizing problems, reflecting on different situations where the same behavior was appropriate and inappropriate could support the flexible regulation of behavior based on context.

Finally, we wanted to understand whether memory impairment was specific to learning associations between items or extended to remembering the individual items that made up the associations. We found that childhood violence exposure was also associated with worse memory of the items that made up the pairings or sequences. However, hippocampal volume was not associated with item memory. Item memory difficulties may be a consequence of the effects of traumatic stress in childhood on surrounding cortical areas of the medial temporal lobe (e.g., perirhinal and parahippocampal cortices) that are responsible for forming representations of individual items prior to their integration in the hippocampus (Davachi, 2006; Eichenbaum et al., 2007). Consistent with this possibility, exposure to violence in childhood is associated with reduced thickness of the parahippocampal gyrus (Busso et al., 2017; Gold et al., 2016). Impaired

item encoding could contribute to difficulties with associative learning in children exposed to violence, as item encoding is a clear pre-requisite for associative learning to occur. Future research is needed to determine how difficulties in these specific types of learning relate to one another. Finally, worse item memory was associated with higher p-factor scores but did not mediate the association of childhood violence exposure with higher p-factor scores. Further research is needed to examine the mechanisms linking item memory to higher p-factor scores.

Several limitations are important to address. First, we were unable to determine conclusively that the observed associations were truly the result of violence exposure rather than other broader environmental factors. In our sample, all of the subjects living in poverty or experiencing other co-occurring forms of deprivation (e.g., neglect) were also exposed to violence, making these exposures entirely collinear and impossible to disentangle. Importantly, childhood adversity involving deprivation (e.g., poverty, neglect, institutionalization) often co-occurs with childhood trauma (Finkelhor, Ormrod, & Turner, 2007; McLaughlin et al., 2012) and is reliably associated with cognitive deficits across multiple domains (Farah et al., 2006; Noble, McCandliss, & Farah, 2007; Noble, Norman, & Farah, 2005). An inability to adjust for co-occurring experiences of deprivation makes it difficult to parse apart unique consequences of childhood trauma from those of deprivation. However, existing research suggests a specific link between childhood trauma exposure and impairments in hippocampus-dependent associative learning. A recent systematic review showed that reduced hippocampal volume was more consistently associated with childhood trauma than with deprivation (McLaughlin et al., 2019). Additionally, a prior study that controlled for levels of cognitive stimulation and the quality of the physical environment in the home showed that young children exposed to violence exhibited difficulties remembering pairs of cues (Rosen et al., 2019). Future research in samples with less

overlap of different types of adverse exposures is needed to further disentangle the unique forms of adversity that contribute to associative learning difficulties.

Second, faces are emotionally salient regardless of emotional expression (Thomas et al., 2001). Future research should clarify whether associative learning difficulties following childhood violence exposure are specific to emotionally salient cues (e.g., faces) or extend to stimuli lacking in emotional content (e.g., objects or shapes). There is some reason to believe that difficulties with higher order cognition and memory following childhood violence exposure are specific to emotionally salient cues. In previous work we have shown that childhood violence exposure was associated with worse inhibition of a dominant response in a task using emotional faces, but not in an identical task using neutral arrows as stimuli (Lambert, King, Monahan, & McLaughlin, 2017). However, in another study, we found that children exposed to violence had worse memory of pairs of shapes (Rosen et al., 2019), suggesting broad impairments that extend to stimuli lacking in emotional content.

Third, the associative learning tasks were conducted behaviorally and not in the scanner, meaning we cannot assume behavioral differences were driven by differences in hippocampal recruitment during associative learning. However, the tasks were simple associative learning tasks (e.g., learning stimulus-stimulus pairings), and prior work consistently shows involvement of the hippocampus during encoding and retrieval in these types of tasks involving visual, audio-visual, contextual, and temporal information (Gonzalo et al., 2000; Lambert et al., 2019; Lambert, Sheridan, et al., 2017; Lehn et al., 2009; Rosen et al., 2018). Greater research is clearly needed on how hippocampal function during associative learning and retrieval relates to associative memory performance in children exposed to violence.

Fourth, the idea that there are transdiagnostic mechanisms underlying all disorders is controversial. Critics have argued that the positive correlations among disorders may occur due to a tendency to report on oneself in a negative way, life impairment that results from many disorders, or networks of symptoms causing other symptoms (Caspi & Moffitt, 2018). However, certain risk factors that are associated with many forms of psychopathology—including childhood maltreatment, neuroticism, poor impulse control over emotions, low intellectual functioning, and disordered thought—are also associated with p-factor, highlighting its utility (Caspi & Moffitt, 2018; Snyder & Hankin, 2017). Identification of the mechanisms that underlie this general vulnerability to psychopathology may advance our understanding of psychopathology and inform the development of transdiagnostic treatment.

Finally, in the absence of longitudinal data, it is impossible to establish whether childhood violence exposure preceded changes in hippocampal volume and associative memory, and whether changes in hippocampal volume and associative memory preceded the onset of psychopathology. Prospective research on children at high risk for trauma exposure could more clearly establish the causal links between these variables.

Conclusion

Difficulties with hippocampus-dependent associative learning may be a transdiagnostic mechanism linking childhood trauma exposure with psychopathology. Future research is needed to examine whether alterations in hippocampal function contribute to associative learning problems in children exposed to violence and the pathways through which associative learning problems contribute to multiple forms of psychopathology. Identifying mechanisms that explain this transdiagnostic risk is necessary for developing interventions that address a wide range of mental health problems in children exposed to trauma.

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Table 1
Distribution of Demographics by Violence (N = 84)

	Violence-Exposed (n = 55)		Controls (n = 29)			
	%	(n)	%	(n)	χ^2	p-value
Female	41.8	23	41.4	12	0.00	.969
Racial/ethnic minority	83.6	46	31.0	9	23.24**	<.001
	M	(SD)	M	(SD)	t-value	p-value
Age	14.17	2.80	13.98	2.82	-0.29	.771
Internalizing symptoms[a]	61.98	10.97	52.93	7.49	-4.43**	<.001
Externalizing symptoms[a]	57.46	11.51	46.28	6.82	-5.55**	<.001

Notes. [a]T-scores based on highest of child-report on the YSR or parent-report on the CBCL internalizing or externalizing problems subscales; *$p < .05$, **$p < .01$.

Table 2
Associative and Item Memory Performance by Violence (N = 84)

	Violence-Exposed (n = 55)		Controls (n = 29)	
	M	(SD)	M	(SD)
Pair Tasks[a]				
Pair Aggregate	1.05	0.60	1.50	0.80
Visual	1.33	0.97	2.01	1.18
Audio-Visual	0.86	0.50	1.08	0.67
Context	0.95	0.80	1.40	0.93
Angry	1.13	0.71	1.54	0.67
Happy	1.14	0.63	1.47	0.76
Neutral	0.87	0.55	1.33	0.71
Item Aggregate	1.91	0.70	2.36	0.65
Temporal Task[b]				
Sequence	0.76	0.09	0.82	0.09
Item	0.93	0.06	0.96	0.04

Notes. Memory measured in terms of [a]*d'* and [b]accuracy.

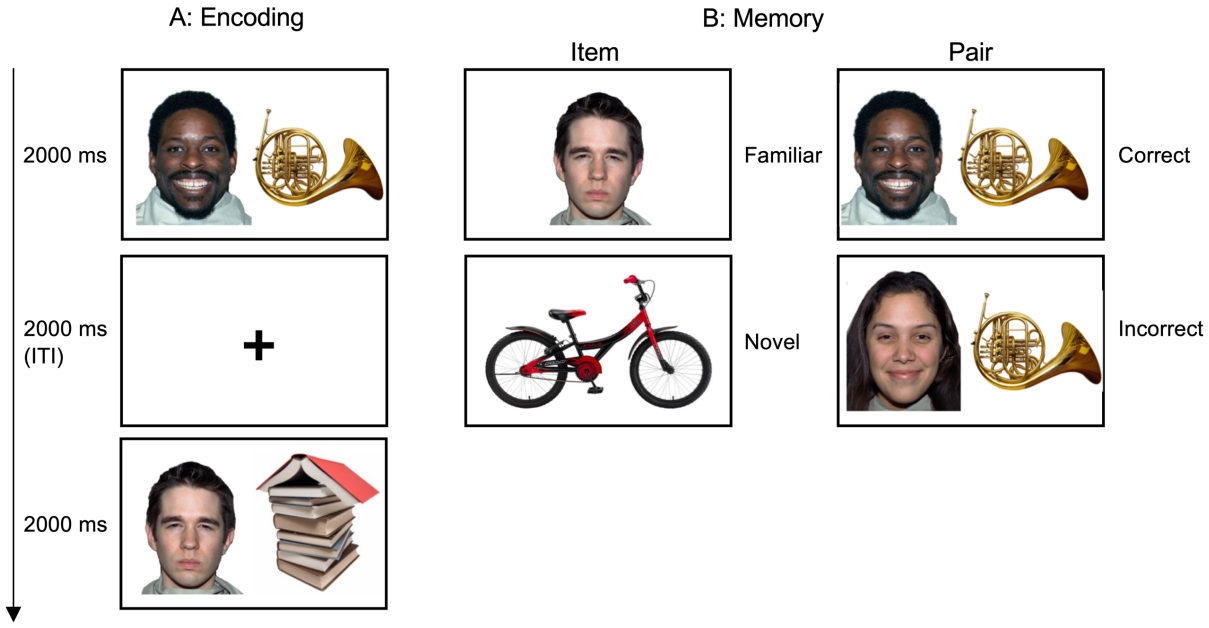

Figure 1. Visual Paired Associate Learning Task. (A) Encoding. Participants were presented with pairs of stimuli (face and object). (B) Memory. Participants saw single items (face or object alone) or pairings of faces and objects. Single items were either familiar or novel. Face-object pairings involved only familiar stimuli but were correct or incorrect. Participants indicated whether the person was feeling the same way about the activity that they saw before.

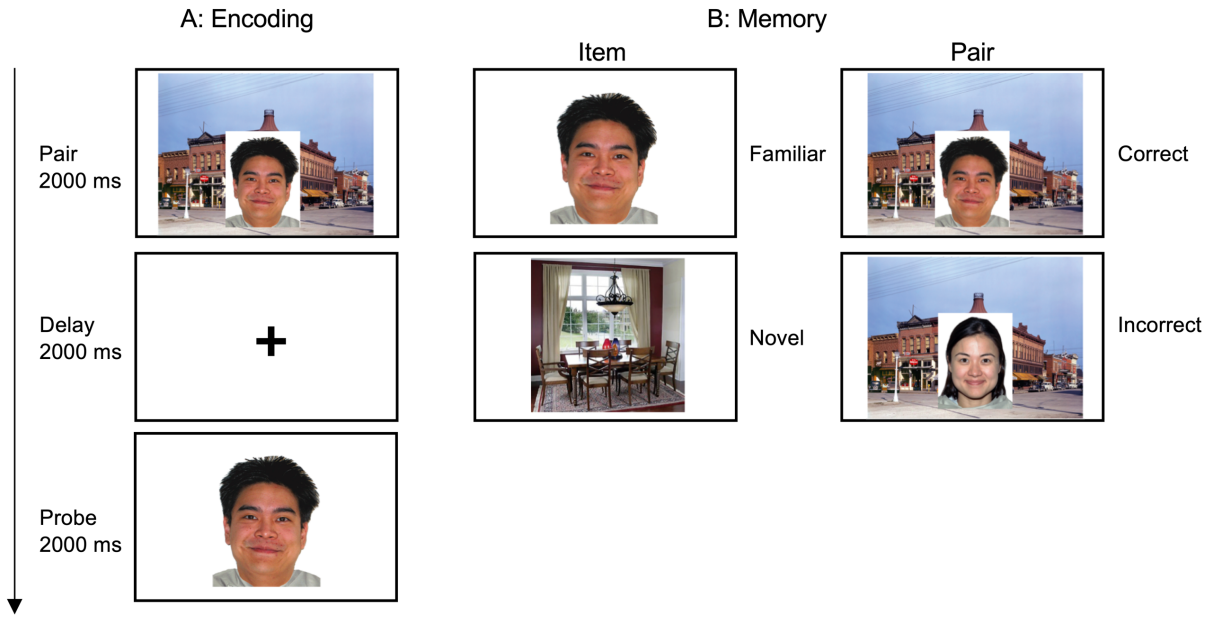

Figure 2. Context Encoding Task. (A) Encoding. Participants were presented with a pair (face in context), delay, and probe (face alone). (B) Memory. Participants saw single items (face or context alone) or pairings of faces and contexts. Single items were either familiar or novel. Face-context pairings involved only familiar stimuli but were correct or incorrect pairings. Participants indicated whether the person was feeling the same way in the same place that they saw them before.

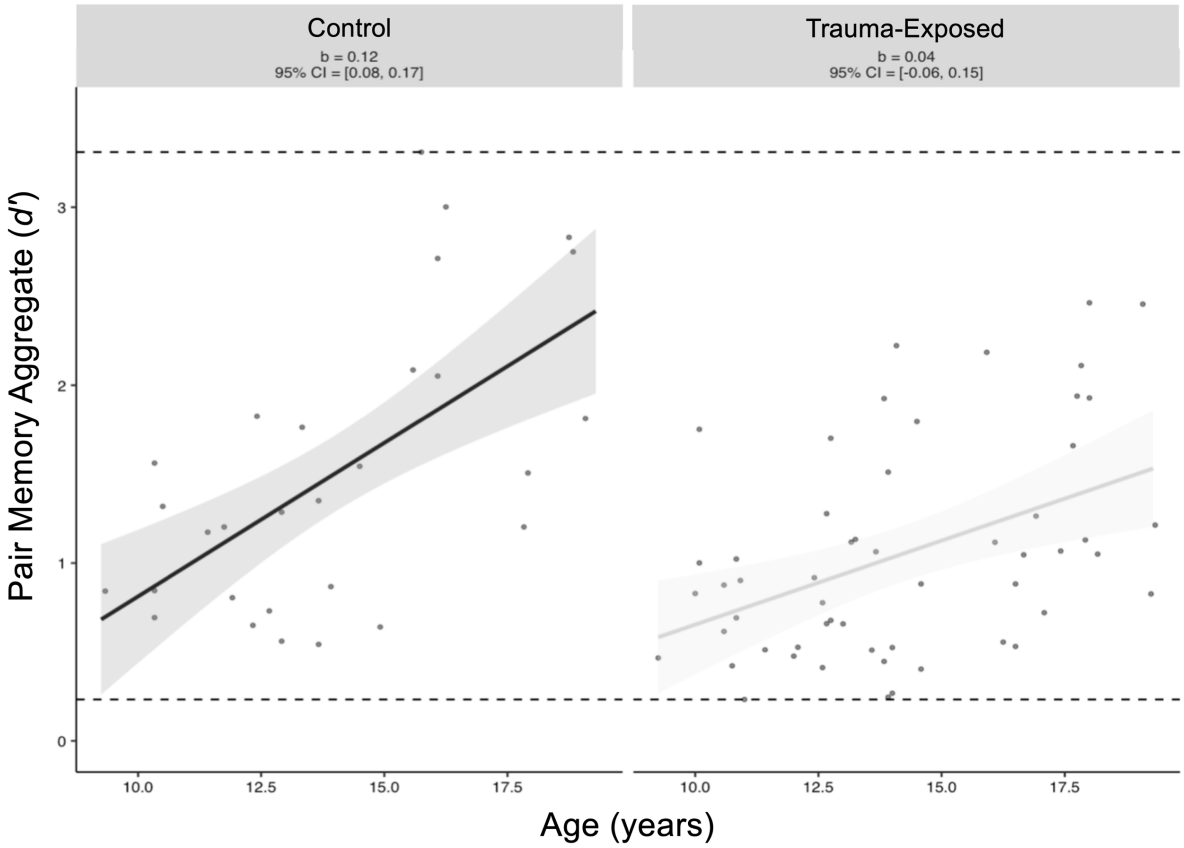

Figure 3. Simple slopes of the association between age and associative memory (i.e., pair memory aggregate) for participants with and without trauma exposure.

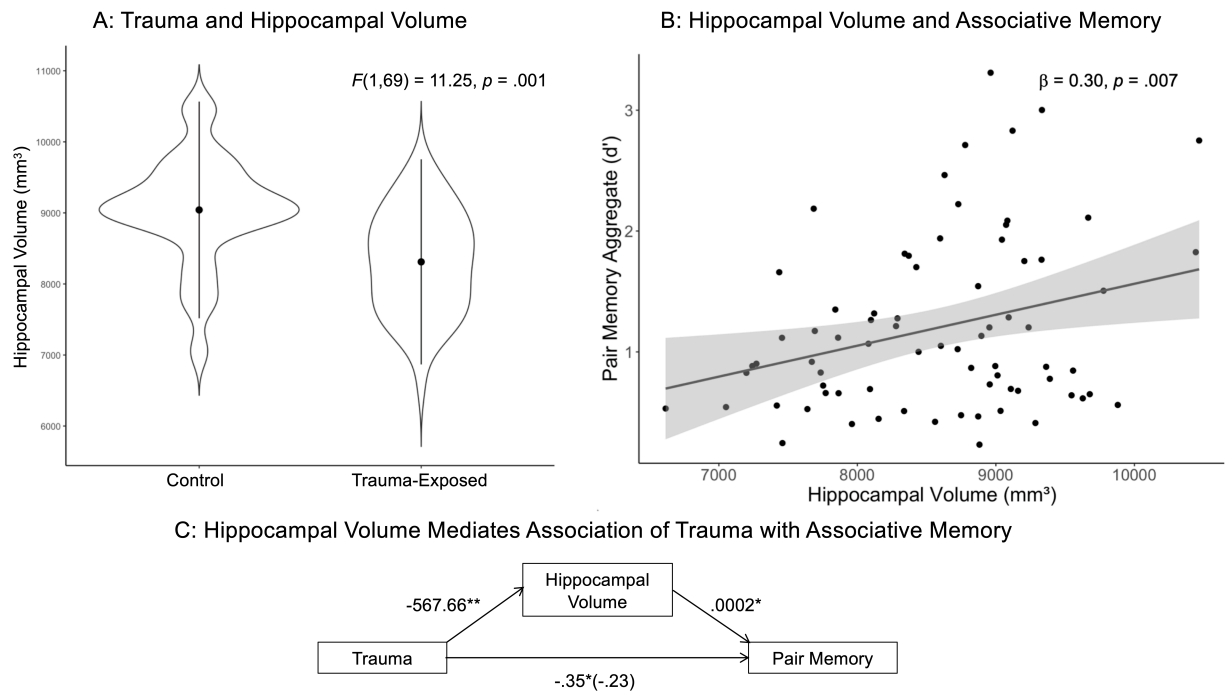

Figure 4. (A) Childhood trauma and bilateral hippocampal volume. (B) Bilateral hippocampal volume and associative memory (i.e., pair memory aggregate). (C) Unstandardized regression coefficients for the association of childhood trauma with pair memory as mediated by hippocampal volume. The unstandardized regression coefficient is in parentheses for the association of childhood trauma with pair memory, controlling for hippocampal volume; $*p \leq 0.05$, $**p \leq 0.01$.

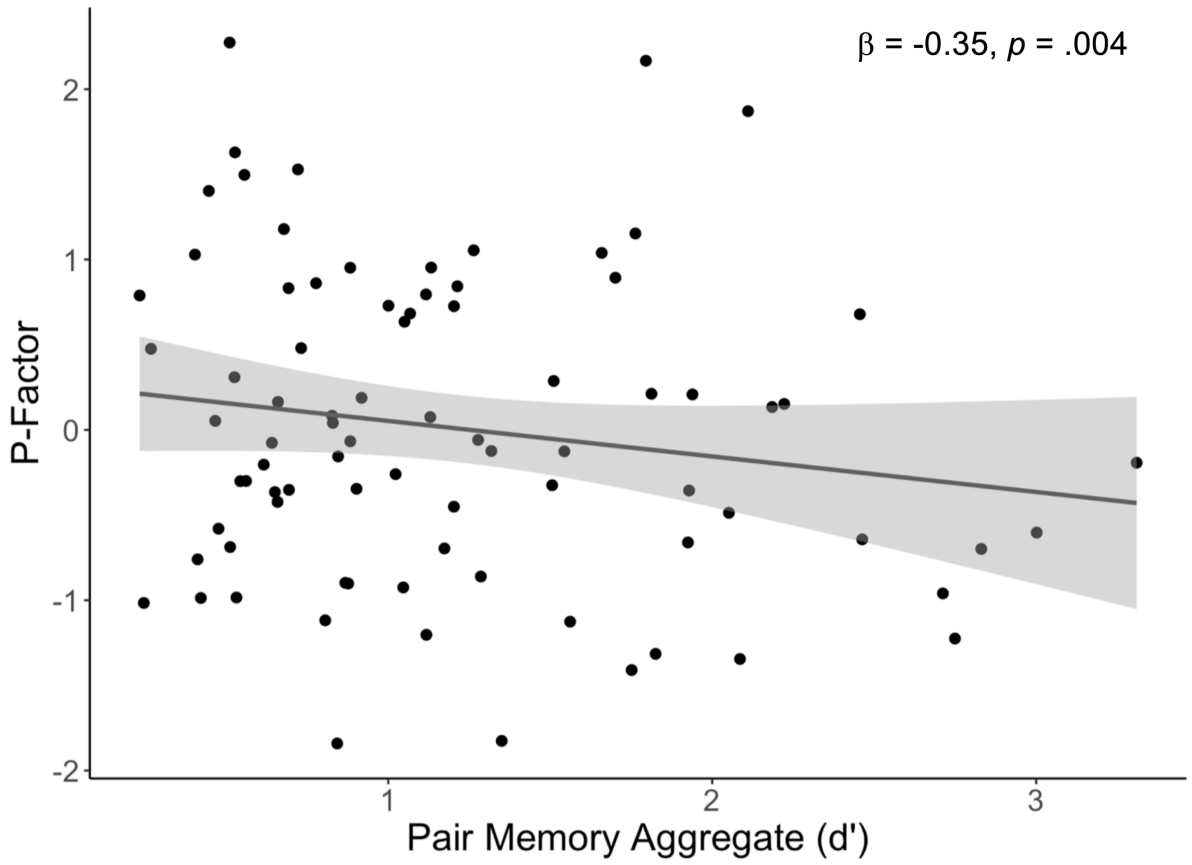

Figure 5. Associative memory (i.e., pair memory aggregate) and transdiagnostic psychopathology (i.e., p-factor).