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Original Article

Trauma moderates the development of the oscillatory dynamics serving working memory in a sex-specific manner

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Abstract

Working memory, the ability to hold items in memory stores for further manipulation, is a higher order cognitive process that supports many aspects of daily life. Childhood trauma has been associated with altered cognitive development including particular deficits in verbal working memory (VWM), but the neural underpinnings remain poorly understood. Magnetoencephalography (MEG) studies of VWM have reliably shown decreased alpha activity in left-lateralized language regions during encoding, and increased alpha activity in parieto-occipital cortices during the maintenance phase. In this study, we examined whether childhood trauma affects behavioral performance and the oscillatory dynamics serving VWM using MEG in a cohort of 9- to 15-year-old youth. All participants completed a modified version of the UCLA Trauma History Profile and then performed a VWM task during MEG. Our findings indicated a sex-by-age-by-trauma three-way interaction, whereby younger females experiencing higher levels of trauma had the lowest *d'* accuracy scores and the strongest positive correlations with age (i.e. older performed better). Likewise, females with higher levels of childhood trauma exhibited altered age-related alpha changes during the maintenance phase within the right temporal and parietal cortices. These findings suggest that trauma exposure may alter the developmental trajectory of neural oscillations serving VWM processing in a sex-specific way.

Key words: MEG; magnetocencephalography; executive function; memory; alpha.

Introduction

According to the developmental traumatology framework (De Bellis 2001), childhood trauma activates the body's biological stress response systems which, if prolonged, can cause a shift from brain development and growth to neural preservation and survival, with abnormal pruning and inhibition of neurogenesis (Kavanaugh et al. 2017). Clinical data suggest that childhood trauma is associated with executive dysfunction during adolescence (Nikulina and Widom 2013; Mothes et al. 2015) leading to poorer memory function, lower academic performance, and lower IQ (Kendall-Tackett and Eckenrode 1996; Deprince et al. 2009; Majer et al. 2010). Studies consistently report deficits on behavioral measures of verbal working memory (VWM) in trauma-exposed children (Deprince et al. 2009; Perfect et al. 2016; Hawkins et al. 2021), suggesting that trauma exposure during childhood may be a significant precursor to VWM dysfunction (Majer et al. 2010).

Working memory is the capacity to maintain and manipulate information for the purpose of goal-directed behavior (Finn et al. 2010; Sander et al. 2012) and is integral in facilitating language comprehension, learning, and reasoning (Baddeley 1992; Satterthwaite et al. 2013). Investigations of healthy cognitive development have found that the ability to maintain and manipulate stimuli in working memory improves across childhood and adolescence, well into young adulthood (Crone et al. 2006). Studies show that these age-related increases in accuracy are concomitant with increased connectivity and task-related recruitment of parietal and prefrontal cortical regions (Crone et al. 2006; Ullman et al. 2014). Studies of VWM-related neural oscillations show that healthy adults and children exhibit widespread alpha desynchronizations during encoding and early maintenance in left hemispheric language regions, and large increases in parieto-occipital alpha during later maintenance (Embury et al. 2019; Heinrichs-Graham and

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Wilson 2015; Koshy et al. 2020; Proskovec et al. 2016; Proskovec, Heinrichs-Graham, et al. 2019; Tuladhar et al. 2007). A recent developmental study showed an increase in many of these responses with age, as well as distinct sex differences (Embury et al. 2019).

Neuroimaging studies have begun to probe the development of VWM processing in people with subclinical and clinical levels of trauma symptomology. A magnetoencephalography (MEG) study in adult patients with PTSD found significant inverse correlations between alpha activity in the right supramarginal gyrus and the parieto-occipital cortices with PTSD severity. The alpha activity seen in the right supramarginal and inferior frontal gyri of PTSD patients resembled alpha activity in the left hemispheric homologue areas of both controls and adults with PTSD, suggesting that right hemispheric cortices were being strongly recruited to aid in task performance (Mcdermott, Badura-Brack, Becker, Ryan, Bar-Haim, et al. 2016a). Moreover, functional magnetic resonance imaging (fMRI) studies have shown decreased accuracy on VWM tasks in adults with PTSD and those exposed to childhood trauma without PTSD compared with nontrauma-exposed controls (Philip et al. 2013, 2016). These studies showed that healthy adults exposed to childhood trauma exhibited recruitment of additional cognitive resources during VWM processing, with significantly greater activation in the right superior and middle temporal gyri, left and right inferior parietal lobules, and left posterior cingulate cortex, when compared with non-trauma-exposed controls, which may suggest compensatory processes following trauma (Philip et al. 2016).

While adult neuroimaging studies provide evidence for neural compensation during VWM processing among people exposed to childhood trauma, to our knowledge, no neuroimaging studies to date have examined the impact of trauma exposure on this cognitive process in a developmental sample. Embury et al. (2019) demonstrated that the developmental process of VWM maturation is highly dynamic and protracted, with sex-specific trajectories in neural recruitment across phases of VWM. Thus, trauma is likely to impact this developmental trajectory, and sex differences may play a role in the overall trajectory. In the present study, we utilized a subset of the sample used in Embury et al. (2019) to examine the role that trauma exposure plays in the development of VWM-related oscillatory dynamics. We hypothesized that trauma would differentially moderate sex-by-age neural recruitment during the encoding and maintenance phases of working memory. Specifically, we expected that increased trauma would lead to greater overall neural recruitment with age, differentially by sex, particularly in the posterior cortices, thus altering the trajectory of development in these neural processes. Importantly, the sample of youth in this study is healthy and typically developing, which has the benefit of being less affected by potentially confounding disease processes related to PTSD and similar conditions, while having the limitation that the degree of trauma is typically less severe. Such an approach is not uncommon in the literature and many studies cited above focused on healthy children and/or adults. Relatedly, we use the terms high and low trauma to describe our continuous measure of trauma exposure in an effort to simplify the interpretation of the relationships we find between development, sex, and trauma exposure, but it is critical to recognize that our use of these terms (i.e. high/low trauma) is restricted to the context of typical development.

Materials and Methods

Participants

We examined data from a subset of the sample used by Embury et al. (2019), which was based on the Developmental Chronnecto-Genomics (Dev-CoG) project (Stephen et al. 2021). All participants from Embury et al. who completed the Trauma History Profile (THP), the MEG working memory task, and had accepted structural MRI data (i.e. limited motion) were included in the current study. Of the 49 participants (24 males, 9–15 years of age) who met these criteria, all identified English as their primary language, were typically developing, and did not have diagnosed psychiatric conditions, previous head trauma, or other disorders affecting brain function. Other exclusionary criteria, determined by parent report, included the presence of metal implants, dental braces or permanent retainers, or other metallic or otherwise magnetic nonremovable devices as well as major medical conditions such as cancer, history or diagnosis of alcohol or substance use disorder, or pregnancy. All procedures were completed at the University of Nebraska Medical Center (UNMC) and approved by the UNMC Institutional Review Board. Informed assent/consent was obtained from the child/child's parent or legal guardian, respectively, before proceeding with the study.

Trauma History Profile

Participants completed the THP, a modified self-report version of the UCLA-THP—a psychometrically sound and frequently used measure of childhood trauma (Steinberg et al. 2013; Badura-Brack et al. 2020). The THP includes items encompassing a broad range of potentially traumatic events that meet Criterion A1 for a DSM-V diagnosis of PTSD. Participants answered yes or no to whether they had experienced each of 12 potentially traumatic events, which was shortened from the original 15 event measure to exclude items about sexual or physical abuse occurring specifically in the home. Personal experiences of violence and witnessing violence to family members were still assessed, including items such as having someone close to them die, being hit, punched or kicked very hard, and seeing or hearing about violence to a loved one. The number of traumatic events endorsed was summed into each participant's total THP score, which was used as a quantitative measure of lifetime trauma.

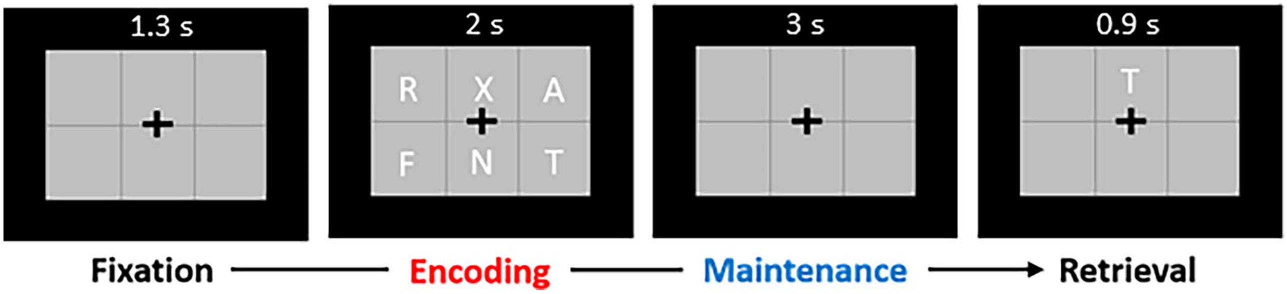

Figure 1. Participants completed a modified Sternberg working memory task. The fixation period (baseline) lasted for 1.3 s, followed by an encoding period (2 s) where six letters were presented to the participant. Afterward, the letters disappear from the screen for a maintenance period of 3 s. In the retrieval phase (0.9 s), participants were presented with a letter that was either present during the encoding phase (in set) or a letter that was not present during the encoding phase (out of set). Participants responded with their index finger if the presented letter was in set, or with their middle finger if the presented letter was out of set.

Task Paradigm

Participants completed a modified Sternberg working memory task (Figure 1) utilized in several previous studies of adults (Embury et al. 2018; Heinrichs-Graham and Wilson 2015; Mcdermott, Badura-Brack, Becker, Ryan, Bar-Haim, et al. 2016a; McDermott, Badura-Brack, Becker, Ryan, Khanna, et al. 2016b; Proskovec et al. 2018; Proskovec, Wiesman, et al. 2019; Wilson et al. 2017) and youths (Embury et al. 2019), in which they were shown a centrally presented fixation cross embedded in a 3×2 grid for 1.3 s (i.e. baseline period). An array of six consonants then appeared at fixed locations within the grid for 2 s (i.e. encoding period). The letters disappeared from the array leaving an empty grid shown for 3 s (i.e. maintenance period), during which participants had to maintain to the encoded letters from the previous phase. A probe letter then appeared on the grid for 9 s (i.e. retrieval period), and participants were instructed to respond by button press with their right index if the probe letter was found in the previous array of letters or with their middle finger if it was not. Participants completed 128 trials, split equally and pseudorandomized between in- and out-of-set trials, for a total run time of about 15 min. Responses were recorded concurrently with the MEG data to allow for accuracy, d' [i.e. z(Hits) − z(False Alarms)], and reaction time to be calculated offline. Briefly, following signal detection theory (Huang-Pollock et al. 2002), responses on the VWM task were first categorized into hits, misses, false alarms, and correct rejections. "Hits" were responses where participants responded in-set to a probe stimulus that was in fact in-set, whereas "misses" were responses where participants responded out-of-set to a probe stimulus that was actually in-set. "Correct rejections" were responses where participants correctly responded out-of-set to an out-of-set stimulus, while "false alarms" were responses where participants incorrectly responded in-set to an out-of-set probe stimulus. Thus, each participants' d' value was calculated as the normalized hit rate minus the normalized false alarm rate to produce the likelihood of correctly identifying in-set probe stimuli.

MEG Acquisition

We closely followed the pipeline used by Embury et al. (Embury et al. 2019) for MEG/MRI data acquisition and statistical analysis. Briefly, participants underwent MEG during their first visit, in a one-layer magnetically shielded room with active shielding engaged. Neuromagnetic responses were collected using an MEG system with 306 magnetic sensors (204 planar gradiometers and 102 magnetometers; MEGIN, Helsinki, Finland) using a bandwidth of 0.1–330 Hz, sampled continuously at 1 kHz. Each participant's data were individually corrected for head motion, and noise reduction was applied using the signal space separation method with a temporal extension (Taulu and Simola 2006).

Structural MRI Data Acquisition

Structural T_1-weighted MRI images were acquired using a Siemens Skyra 3-Tesla MRI scanner with a 32-channel head coil and an MPRAGE sequence with the following parameters: TR = 2400 ms; TE = 1.94 ms; flip angle = 8°; FOV = 256 mm; slice thickness = 1 mm (no gap); voxel size = $1 \times 1 \times 1$ mm. Following source analysis (i.e. beamforming), participants' $4.0 \times 4.0 \times 4.0$ mm functional images were also transformed into standardized space using the transform that was previously applied to the structural MRI volume and spatially resampled. Before MEG recording, four coils were attached to participants' heads and localized, together with the three fiducial points and scalp surface, using a 3D digitizer (Fastrak 3SF0002, Polhemus Navigator Sciences, Colchester, VT, USA). Once participants were positioned for MEG recording, an electrical current with a unique frequency label (e.g. 322 Hz) was fed to each of the coils. This induced a measurable magnetic field and allowed each coil to be localized with reference to the sensors throughout the recording session. Since coil locations were also known in head coordinates, all MEG measurements could be transformed into a common coordinate system. Using this coordinate system, participants' MEG data were coregistered with their individual structural T_1-weighted MRI images prior to source space analysis using BESA MRI (Version 2.0).

Time–Frequency Decomposition and Statistical Analysis

Cardiac artifacts were removed from the data using signal-space projection, which was accounted for during source reconstruction (Uusitalo and Ilmoniemi 1997). The continuous magnetic time series was segmented into 7.2-s duration epochs (−1.3 to 5.9 s, 0.0 s = encoding onset), with the −0.4 to 0.0 s before the onset of the encoding grid defined as baseline. Epochs containing artifacts (e.g. eye blinks, muscle artifacts, and coughing) were rejected based on a fixed threshold method, supplemented with visual inspection. The distribution of amplitude and gradient values per participant was computed using all trials, and the highest amplitude/gradient trials relative to the total distribution were excluded by selecting a threshold that rejected extreme values. Thresholds were participant-specific due to differences among individuals in head size and sensor proximity, which strongly affect MEG signal amplitude. Trials were also randomly excluded from participants with the highest total trial counts so that the total number of accepted trials used in the final analyses did not differ by age. An average of 118 ($SD = 10.54$) trials per participant were included in the final analysis. Artifact-free epochs were transformed into the time–frequency domain using complex demodulation (resolution: 1.0 Hz, 50 ms; Kovach and Gander 2016), and the resulting spectral power estimations per sensor were averaged over trials to generate time–frequency plots of mean spectral density. These sensor-level data were normalized using the respective bin's baseline power, which was calculated as the mean power during the −0.4- to 0.0-s time-period. The specific time–frequency windows used for imaging were determined by statistical analysis of the sensor-level spectrograms across the entire array of gradiometers during the 5.0-s encoding and maintenance time windows. To reduce the risk of false-positive results while maintaining reasonable sensitivity, a two-stage procedure was followed to control for Type 1 error (Ernst 2004; Maris and Oostenveld 2007). Stage one included paired-sample t-tests against baseline across participants and sensors using an uncorrected threshold of $P < 0.05$. To control for multiple comparisons, significant clusters were then subjected to nonparametric permutation testing using 10,000 permutations and a threshold of $P < 0.001$. Based on these analyses, the time–frequency windows that contained significant oscillatory events across all participants were divided into equal duration time windows of 400 ms and subjected to a beamforming analysis.

MEG Source Imaging and Statistics

Cortical activity was imaged through an extension of the linearly constrained minimum variance vector beamformer (Van Veen et al. 1997; Gross et al. 2001; Hillebrand et al. 2005), which employs spatial filters in the frequency domain to calculate source power for the entire brain volume. The single images were derived from the cross-spectral densities of all combinations of MEG gradiometers averaged over the time–frequency range of interest, and the solution of the forward problem for each location on a grid specified by input voxel space. This use of the cross-spectral densities is often referred to the dynamic imaging of coherent sources beamformer (Gross et al. 2001). Following convention, we computed noise-normalized, source power per voxel in each participant using active (i.e. task) and passive (i.e. baseline) periods of equal duration and bandwidth (Hillebrand et al. 2005). Such images are typically referred to as pseudo-*t* maps, with units (i.e. pseudo-*t*) that reflect noise-normalized power differences (i.e. active vs. passive) per voxel. MEG preprocessing and imaging used the BESA (Version 6.1) software. For more information on the analysis pipeline, see (Arif et al. 2020; Heinrichs-Graham and Wilson 2015; Koshy et al. 2020; Meehan et al. 2021; Proskovec, Heinrichs-Graham, et al. 2019). Normalized differential source power was computed for the statistically selected time–frequency bands, using a common baseline, over the entire brain volume per participant at 4.0 × 4.0 × 4.0 mm resolution, resulting in 3D maps of brain activity. These maps were averaged, per participant, across the time windows used to image neural activity during encoding and maintenance periods, separately, which were of equal duration and identified via sensor-level analysis. These averaged maps per participant were subjected to full factorial analysis using SPM12 (Kiebel et al. 2007). Participant sex, age, and trauma scores, as well as all second- and third-order interactions, were used as predictors in the model, to compute voxel wise maps reflecting the main and interactive effects of age, sex, and trauma. Of note, prior to analyses, THP scores were linearly transformed to accurately capture interactions with trauma scores of zero.

Statistical Analysis of Behavior

To examine the relationship between behavioral measures and participant sex, age, and trauma, we conducted a multiple analysis of covariance (MANCOVA) using SPSS (Version 25.0). The independent variables used in the MANCOVA model included sex as a factor, age and trauma scores as co-factors, and all second- and third-order interactions between sex, age, and trauma (i.e. sex-by-age, sex-by-trauma, trauma-by-age, and sex-by-age-by-trauma). Behavioral measures used as dependent variables were *d*' scores and overall reaction time. Follow-up univariate analysis was conducted to examine each predictor's relationship with each dependent variable, controlling for the other independent variables in the model.

Results

Trauma, Sex, and Age Effects on Task Performance

Participants had an accuracy of 70.79% ± 10.94%, *d*' scores of 1.51 ± 0.56, and reaction times of 1084.19 ± 243.21 ms. Consistent with Embury et al. (2019), we

Table 1. Demographic data for the sample included in the models, separately for each sex.

	Full sample		Males		Females			
Variable	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	t	P
Age	11.97	1.65	12.28	1.74	11.67	1.53	1.29	.204
Accuracy	70.79	10.94	70.70	10.70	70.88	11.39	-0.054	.957
d' score	1.51	0.56	1.50	0.60	1.53	0.53	-0.162	.872
Overall reaction time	1084.19	243.20	1051.42	254.30	1115.66	232.84	-0.923	.361
THP Score	2.06	1.94	2.33	2.16	1.80	1.71	0.961	.342

reproduced the significant correlation between age and d' scores, $r = 0.31$, $P < 0.05$, and the significant correlation between age and reaction time, $r = -0.39$, $P < 0.01$. There were no significant sex-by-age interactive effects on d' scores, $F = 0$, $P = 0.99$, or reaction time, $F = 0.97$, $P = 0.33$. Participants reported experiencing 2.06 (±1.94) different types of traumatic events, on average. Additionally, all variables of interest did not significantly differ between sexes (Table 1).

MANCOVA analysis testing the effect of sex, age, trauma, and all interaction effects on both d' score and reaction time showed significant sex-by-age-by-trauma ($P = 0.026$; Fig. 2) and sex-by-trauma ($P = 0.024$) effects at the multivariate level (Table 2). At the univariate level, there was a significant interaction effect of sex-by-age-by-trauma score on d' scores. To determine the origin of these effects, we split the model by sex and found a significant univariate positive relationship between trauma and age in females on d' scores, $\beta = 2.806$, $t(24) = 2.150$, $P = 0.043$, whereas this relationship was nonsignificant and negative, but not significant in males, while controlling for all other predictors in the model (Table 3). Univariate analysis also showed a significant interaction between sex and trauma on d' scores, $\beta = 3.693$, $t(48) = 2.188$, $P = 0.034$, such that increasing trauma significantly predicted lower d' scores in females, collapsing across age, $\beta = -2.683$, $t(24) = -2.105$, $P = 0.047$, with a nonsignificant positive relationship with d' scores in males (Table 3). There were no significant main effects of sex, age, or trauma on d' scores, and no significant univariate effects were found on overall reaction time.

Table 2. Multivariate results predicting behavioral outcomes.

	Pillai's Trace	df	F	η^2_p
Main effect				
Sex	0.077	1	1.66	0.077
Covariates				
Age	0.114	1	2.567	0.114
Trauma	0.005	1	0.107	0.005
Interaction effects				
Age × Sex	0.073	1	1.563	0.073
Age × Trauma	0.005	1	0.105	0.005
Sex × Trauma	0.169	1	4.080*	0.169
Age, Sex, and Trauma	0.167	1	4.012*	0.167

*$P < 0.05$ (two-tailed), Trauma = THP score.

Table 3. Univariate results of the behavioral measures, controlling for covariates.

	d' Score		Reaction Time	
	F	η^2_p	F	η^2_p
Main effect				
Sex	2.90	0.066	0.617	0.015
Covariates				
Age	1.815	0.042	3.646	0.082
Trauma	0.217	0.005	0.001	0.000
Interaction effects				
Age × Sex	2.812	0.064	0.481	0.012
Age × Trauma	0.212	0.005	0.005	0.000
Sex × Trauma	4.787*	0.105	3.913	0.087
Age, Sex, and Trauma	5.107*	0.111	3.441	0.077

*$P < 0.05$ (two-tailed).

Sensor Level Results

To identify the time–frequency windows containing significant oscillatory responses across all participants, the sensor-level spectrograms were examined using paired-samples t-tests against baseline followed up with permutation testing to control for Type 1 error. This analysis showed a significant decrease in 9–16 Hz alpha activity throughout the encoding period, from 200 to 2200 ms, in posterior and left hemisphere sensors ($P < 0.001$, corrected; Fig. 3). There was also a significant increase in 8–11 Hz alpha activity during the maintenance period, from 3400 to 5000 ms, in posterior sensors ($P < 0.001$, corrected; Fig. 3).

Source Level Results

We computed a full factorial model using the whole brain alpha averages for each participant's encoding and maintenance periods. These whole-brain averages were computed per participant by averaging over the respective time windows identified through the sensor-level analysis (See Sensor level results) and were of equal duration (i.e. five 400-ms windows for encoding and four 400-ms windows for maintenance). Predictors included participant sex, age, THP score, and each higher order interaction. There were no significant findings for the encoding phase (all Ps > 0.005, corrected). However,

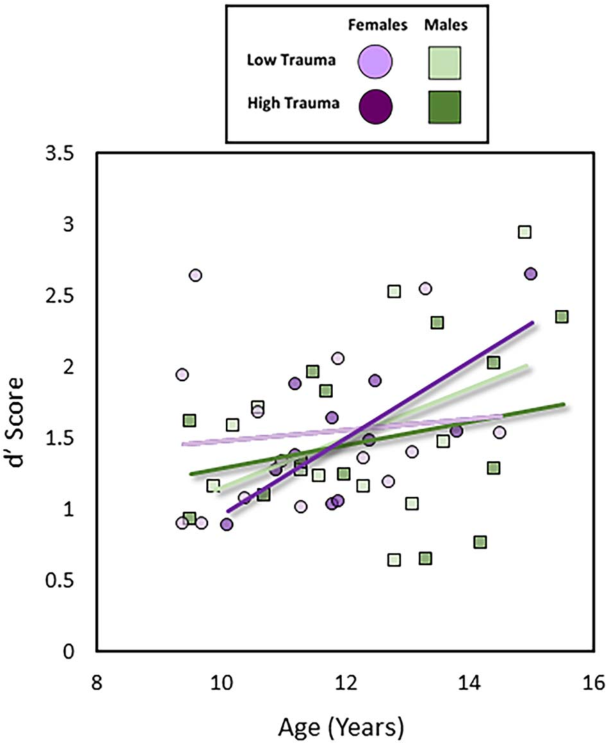

Figure 2. MANCOVA analysis shows a sex-by-age-by-trauma interaction effect on *d*' scores of accuracy. In females who endorsed high trauma exposure, age was tightly correlated with *d*', with younger females with high trauma having significantly lower *d*' scores and older females with high trauma having significantly greater *d*' scores. Note that for display purposes, we have separated the sample into low and high trauma groups using a THP score of 2.06. However, all statistical analyses treated THP scores as a continuous measure.

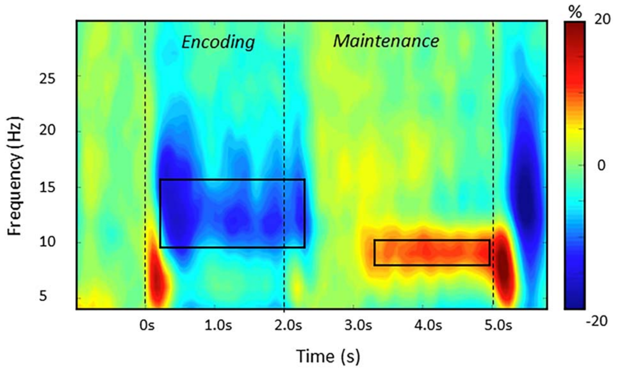

Figure 3. Grand-averaged spectrogram from a representative sensor near the parieto-occipital cortices. The same sensor was chosen in each participant. Time is denoted on the *x*-axis in seconds and frequency is shown on the *y*-axis in Hz. Time–frequency windows for source imaging were derived from statistical analysis of the sensor-level spectrograms, and the significant windows are outlined by black boxes. Increases in power are denoted in red and decreases in power are denoted in blue, all relative to baseline, with a scale bar on the far right. A significant decrease in alpha from 9–16 Hz was observed throughout the encoding period into early maintenance, followed by a significant alpha increase from 8 to 11 Hz lasting from mid to late maintenance.

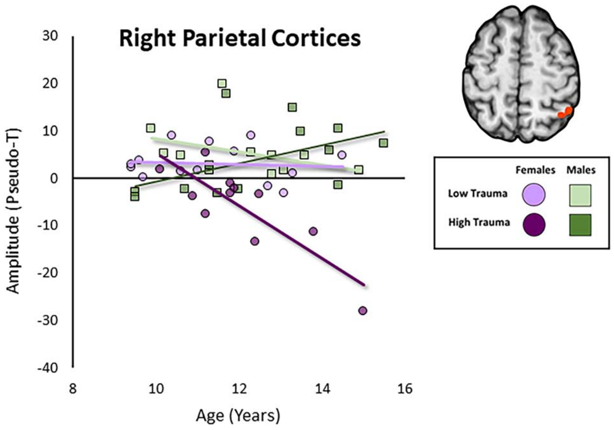

Figure 4. Sex-by-age-by-trauma interaction effect in the right parietal cortex. Females who endorsed high trauma exposure had a significantly stronger decrease in alpha oscillatory power with increasing age during the maintenance phase of the VWM task. Note that for display purposes, we have separated the sample into low and high trauma groups using a THP score of 2.06. However, all statistical analyses treated THP scores as a continuous measure.

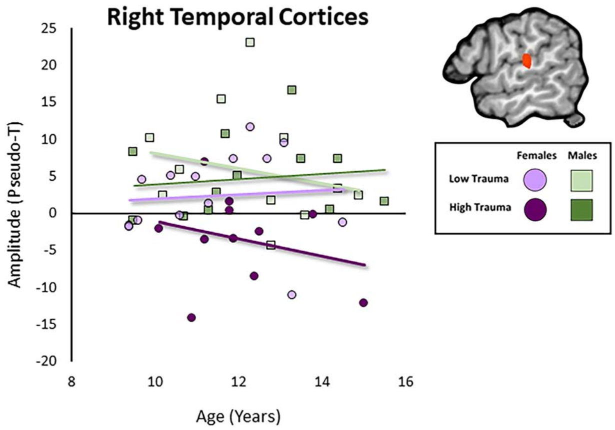

Figure 5. Sex-by-age-by-trauma interaction effect in the right temporal cortex. Females who endorsed high trauma exposure had a significantly stronger decrease in alpha oscillatory power with increasing age during the maintenance phase of the VWM task. Note that for display purposes, we have separated the sample into low and high trauma groups using a THP score of 2.06. However, all statistical analyses treated THP scores as a continuous measure.

analysis of the maintenance phase showed significant sex-by-age-by trauma interaction effects in the right inferior parietal cortices ($P < 0.005$; Fig. 4), right temporal cortices ($P < 0.005$; Fig. 5), and the medial parietal cortices ($P < 0.005$; Fig. 6). In these three regions, the directionality indicated that females endorsing high trauma had reduced alpha oscillatory amplitude compared with baseline, while other participants maintained positive oscillatory amplitude. This desynchronization among females who experienced high trauma became significantly greater with increasing age, whereas this relationship was not present in all other participants.

To identify the relationship between these findings and behavior, we conducted Pearson correlation analyses and found that *d*' is related to alpha neural response strength in the voxel showing the strongest three-way interaction (sex-by-age-by-trauma) effect in the high-trauma females. Specifically, our results show robust correlations with stronger alpha oscillatory responses (i.e. more negative) in the right parietal cortices, $r = -0.642$, $P < 0.05$, and the medial inferior

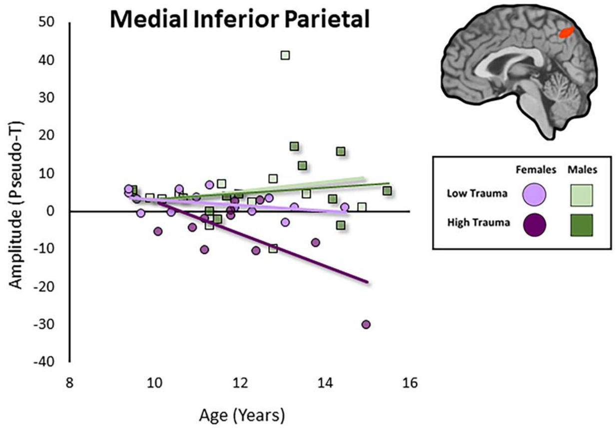

Figure 6. Sex-by-age-by-trauma interaction effect in the medial parietal cortex. Females who endorsed high trauma exposure had a significantly stronger decrease in alpha oscillatory response with increasing age during the maintenance phase of the VWM task. Note that for display purposes, we have separated the sample into low and high trauma groups using a THP score of 2.06. However, all statistical analyses treated THP scores as a continuous measure.

parietal cortex, $r = -0.624$, $P < 0.05$, correlating with increased *d*' scores.

Discussion

In this study, we used high-density MEG to assess the relationship between trauma exposure and the neural dynamics of the encoding and maintenance phases of working memory. Regarding task performance, a complex relationship between sex and trauma across adolescence emerged; as age increased, higher levels of prior trauma significantly predicted higher *d*' accuracy scores, specifically in females. Conversely, in males, with increasing age, there was no relationship between trauma and *d*' scores. With respect to the neural oscillatory dynamics underlying VWM, our data show that trauma significantly moderates the effects of sex and development on maintenance in the right inferior parietal, medial parietal, and the right superior temporal gyrus. Taken together, these findings demonstrate strong sex-specific effects of trauma on VWM processing during the transition from childhood to adolescence. Below, we examine possible mechanisms and implications of these results for understanding the effects of trauma on brain and cognitive development.

We replicated the significant positive correlation between age and *d*' scores previously reported in Embury et al. (2019) and found that in females, this relationship is stronger in those with higher trauma exposure. No such relationship was observed in males. Previous work in trauma-exposed children (Bücker et al. 2012), adolescents (Kavanaugh et al. 2015; Vasilevski and Tucker 2016; Carvalho et al. 2020), and adults (Majer et al. 2010; Philip et al. 2016) demonstrates reliable performance decrements on working memory tasks relative to nontrauma-exposed persons. While younger high-trauma females exhibited such trauma-related VWM deficits in this study, our older high-trauma females actually had numerically higher *d*' scores than all other participants. This distinctly improved performance with age has not been previously reported. One mechanism that may be driving this sex- and age-specific effect, at least in part, could be earlier cognitive maturation triggered by the effect of trauma exposure on pubertal onset. Pubertal timing, between 8 and 14 years of age in females and 9 and 15 years in males, is the most consistently examined marker of accelerated development in relation to early life trauma-exposure (Colich et al. 2020). Because trauma exposure is associated with early menarche in females (Kim and Smith 1998; Rowe 2000; Graber et al. 2006; Foster et al. 2008), future studies should examine the relationship between trauma exposure and the development of higher order cognitive processes, specifically within the context of pubertal status, accounting for the sex-specificity of these effects.

Our neural results showed that trauma modulated VWM processing among high-trauma females during the maintenance period, with these participants exhibiting weaker alpha activity in temporal and parietal regions than other participants in our sample. Furthermore, our correlational analyses showing that *d*' was related to alpha neural response strength in the voxel showing the strongest sex-by-age-by-trauma effect suggests that high-trauma females' improved accuracy with age seems to be supported by stronger alpha activity in the right superior and medial parietal cortices. This could reflect a compensatory process by which females utilize more neural resources to maintain the representations of the encoded stimuli for accurate retrieval, which would be consistent with the fMRI literature (Philip et al. 2013; Philip et al. 2016). Studies show that the neuronal oscillations that support sex-specific VWM strategies emerge early in adolescence and strengthen with age (Embury et al. 2019). Embury et al. (2019) showed that, much like adult males (Hill et al. 2014), adolescent males exhibit greater posterior activity, including occipital, parietal, and cerebellar cortices, during maintenance with stronger alpha oscillations than females. Embury et al. (2019) also showed that adolescent females exhibit stronger recruitment of right inferior prefrontal regions during encoding than males. Thus, we propose that trauma exposure magnifies sex-specific developmental trajectories of the neural activity serving VWM processing, such that males and females employ different cognitive strategies to yield sexually dimorphic neuronal oscillatory patterns following trauma exposure, although this precise pattern is speculative and future work will need to further disentangle trauma-specific sex effects on cognitive strategies used during VWM, particularly among those who have experienced more severe instances of trauma.

Interestingly, the patterns of neural activity identified in the right parietal and superior temporal cortices among high-trauma females align with those found in previous studies of adults with trauma exposure, although the effect was modulated by age in our developmental sample. A study utilizing visual and VWM tasks found that when compared with nontrauma-exposed men, adult male victims of severe physical abuse during childhood had reduced activity in the right lateral and superior temporal cortices, occipital cortex, and prefrontal cortices (Raine et al. 2001). Another study examined the oscillatory dynamics associated with WM processing in a group of male PTSD patients and showed reduced alpha synchronous activity in the right superior and middle temporal gyri, as well as parieto-occipital alpha activity that was inversely correlated with PTSD severity (McDermott, Badura-Brack, Becker, Ryan, Khanna, et al. 2016b). While resting-state EEG studies in children report mixed findings in the association between early childhood adversity and resting state alpha oscillatory power (Stamoulis et al. 2015, 2017; Bick et al. 2019), to our knowledge, there are no studies examining the neural oscillatory dynamics of VWM processing in a sample of trauma-exposed youth without psychiatric illness or medication use.

Neural activity in left temporal cortices during encoding has been shown to support subvocal rehearsal (Baddeley 1992); thus, recruitment of its right hemispheric homologue during maintenance might be one strategy employed by high-trauma females to actively maintain stimuli in memory stores. It may also be the case that females exposed to high trauma levels are relying on networks typically recruited for spatial working memory during VWM processing. Proskovec, Wiesman, et al. (2019) reported that alpha and beta desynchronizations are strongly correlated with increased performance on spatial working memory tasks in regions similar to those reported here (i.e. in temporal and parietal cortices). As parietal regions have been associated with top-down control of spatial attention and retention of spatial features (Rottschy et al. 2012; Sato et al. 2018), it is possible that for females, the experience of trauma might prompt a greater reliance on spatial cues to keep stimuli within working memory stores. In fact, one EEG study (Myers et al. 2014) showed that greater right-lateralized posterior alpha desynchronization during VWM was associated with higher accuracy on a trial-by-trial basis, providing some evidence for the relationship between alpha desynchronization and working memory accuracy.

A limitation of the present analysis was that the population utilized is relatively low-risk. While participants endorsed a range of traumatic events, they endorsed about two potentially traumatic experiences on average. The effects of trauma exposure on a typically developing sample of youth may not be readily generalizable to samples with higher trauma exposure such as youth with diagnosed PTSD. Additionally, trauma was characterized in the current study by the number of events experienced excluding some types of trauma (e.g. sexual abuse), so specific effects of type, timing, and chronicity of trauma on VWM processing remain to be further elucidated. Future studies should examine the effects of trauma type, timing, and severity on the development of VWM oscillatory dynamics to understand the cascade of trauma-specific effects on neural development and functioning.

Despite these limitations, these novel findings highlight sex as a key moderator by which trauma exposure during childhood may alter the neural dynamics of working memory during the transition into adolescence. Trauma exposure negatively influences accuracy on working memory processing in a sex-and age-specific manner, and greater trauma exposure is also linked to altered alpha oscillatory power in right superior temporal and inferior parietal cortices. This study serves as a foundation for future exploration of early life trauma, providing strong suggestions for the future investigation of pubertal status in elucidating the relationship between trauma exposure, sex, age, and the neural oscillatory dynamics serving VWM processing.

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