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Examining the effects of prenatal alcohol exposure on performance of the sustained attention to response task in children with an FASD

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Abstract

Prenatal alcohol exposure (PAE), the leading known cause of childhood developmental disability, has long-lasting effects extending throughout the lifespan. It is well documented that children prenatally exposed to alcohol have difficulties inhibiting behavior and sustaining attention. Thus, the Sustained Attention to Response Task (SART), a Go/No-go paradigm, is especially well suited to assess the behavioral and neural functioning characteristics of children with PAE. In this study, we utilized neuropsychological assessment, parent/guardian questionnaires, and magnetoencephalography during SART random and fixed orders to assess characteristics of children 8–12 years old prenatally exposed to alcohol compared to typically developing children. Compared to neurotypical control children, children with a Fetal Alcohol Spectrum Disorder (FASD) diagnosis had significantly decreased performance on neuropsychological measures, had deficiencies in task-based performance, were rated as having increased Attention-Deficit/Hyperactivity Disorder (ADHD) behaviors and as having lower cognitive functioning by their caretakers, and had decreased peak amplitudes in Broadmann's Area 44 (BA44) during SART. Further, MEG peak amplitude in BA44 was found to be significantly associated with neuropsychological test results, parent/guardian questionnaires, and task-based performance such that decreased amplitude was associated with poorer performance. In exploratory analyses, we also found significant correlations between total cortical volume and MEG peak amplitude indicating that the reduced amplitude is likely related in part to reduced overall brain volume often reported in children with PAE. These findings show that children 8–12 years old with an FASD diagnosis have decreased amplitudes in BA44 during SART random order, and that these deficits are associated with multiple behavioral measures.

KEYWORDS
attention, child behavior, Fetal Alcohol Spectrum Disorder, Go/No-go, magnetoencephalography, neuropsychology, Sustained Attention to Response Task

1 | INTRODUCTION

Prenatal alcohol exposure (PAE) can result in physical, cognitive, and behavioral effects extending throughout the lifespan. Physical effects include growth restriction, characteristic facial features, and altered brain morphology/seizure activity. Cognitive effects include deficits in visuospatial functioning, verbal and nonverbal learning, attention, and executive functioning (Riley & McGee, 2005). Lastly, behavioral effects include externalizing behaviors (e.g., hyperactivity, behavioral dysregulation) and internalizing symptoms (e.g., depression, anxiety) (Hoyme et al., 2016; Kodituwakku, 2007; Riley & McGee, 2005).

Diagnoses falling under the umbrella of Fetal Alcohol Spectrum Disorder (FASD) include Fetal Alcohol Syndrome (FAS)/Partial Fetal Alcohol Syndrome (PFAS), which includes morphological anomalies coupled with functional deficits (CDC, 2002), and Alcohol-Related Neurodevelopmental Disorder (ARND), which does not result in physically discernable cranio-facial differences (Sampson et al., 1997) but is accompanied by cognitive and behavioral effects of PAE. Importantly, it is estimated that up to 90% of prenatal alcohol exposed individuals do not express the physically discernable alterations required for a FAS and PFAS diagnosis (Bertrand et al., 2005; May & Gossage, 2001). While estimates of worldwide prevalence rates vary greatly by country, with FASD diagnoses of up to 113.22 per 1000 people in South Africa (Roozen et al., 2016), current rates in the U.S.A. are generally discussed at 6–9 per 1000 for FAS and between 20 and 50 per 1000 for ARND (May et al., 2009, 2014, 2018). As compared to children with FAS/PFAS, children with ARND experience increased negative sequelae due to under- and misdiagnosis resulting in lack of access to appropriate interventions (Streissguth et al., 2004). This disparity is exacerbated in rural areas where skilled professionals and adequate behavioral health services are limited (Koren et al., 2014). Given our knowledge that children with FASD perform better academically and cognitively when early identification and interventions are applied (Kalberg & Buckley, 2007; Paley & O'Connor, 2009), understanding the neurophysiological underpinnings of these deficits is informative for development of intervention techniques.

1.1 | Task-related behavioral deficits

Numerous studies have examined the task-related behavioral characterization of FASD. Specifically, Green et al. (2009) assessed pro-saccade and anti-saccade reaction times (SRT) and accuracy in 92 healthy control (HC) children and 89 children aged 8–15 years with FAS, PFAS, or ARND. They demonstrated that children with an FASD had greater SRT and significantly more errors in both the pro- and anti-saccade tasks. Although all three FASD subgroups performed worse than the HC group in the anti-saccade task, the ARND group showed faster SRT and higher accuracy when compared to FAS and PFAS groups, indicating a potential spectrum of cognitive effects. Increased SRT may be associated with both oculomotor deficits as well as decreases in inhibition and attention. Paolozza et al. (2014) showed that compared to HC, children with an FASD diagnosis had significantly more errors on an anti-saccade task, a memory-guided saccade task, and performed worse on measures of attention and inhibition. Further, the FASD group's score on inhibition correlated with direction errors in the anti-saccade task, a relationship that was lacking in the HC group. The authors postulate common brain regions/structures are important for mechanisms regulating inhibitory control on both the NEPSY-II inhibition subscale and eye movement tasks.

1.2 | Brain morphological characterization of FASD

The most frequently reported findings in brain morphology studies in individuals with an FASD diagnosis include a decreased cranial vault and the subsequent whole brain volumetric reductions, specifically in the cerebellum, basal ganglia, caudate, hippocampus, and corpus callosum (Archibald et al., 2001; Johnson et al., 1996; Lebel et al., 2008, 2012; Li et al., 2008; Mattson et al., 1994, 1996; Willoughby et al., 2008). Volume reductions, uncorrected for brain size, in frontal, temporal, parietal, and to a lesser degree, occipital lobes have been documented in children with FASD compared to healthy children (Archibald et al., 2001; Sowell et al., 2002). When controlling for overall reductions in brain volume, the parietal lobe volume is diminished, suggesting the parietal lobes are particularly sensitive to PAE (Archibald et al., 2001; Riley & McGee, 2005; Sowell et al., 2001, 2002). In children, adolescents, and young adults prenatally exposed to alcohol, there are consistent results reporting decreases in overall brain volume however mixed results have reported both increases in regional gray matter volume and decreases in white matter volumes, most frequently in the perisylvian cortices of the temporal and parietal lobes (Archibald et al., 2001; Sowell et al., 2001). These are brain regions potentially corresponding to the language deficits seen in adolescents with FASD (Kodituwakku et al., 1995). Sowell et al. (2001) found significant reductions in brain growth in the ventral frontal lobes, predominantly in the left hemisphere, which potentially influences response inhibition, behavioral control, and executive functioning deficits seen in individuals with an FASD diagnosis (Mattson et al., 1999; Olson et al., 1998). This constrained brain growth suggests the teratogenic effects of prenatal alcohol exposure extend well beyond the prenatal period (Riley & McGee, 2005). In summary, brain structural differences in PAE reveal a common pattern of reduced total brain volume relative to controls, however, regional differences and cortical thickness and surface area results vary across studies.

1.3 | Electrophysiological techniques in FASD

Magnetoencephalography (MEG) and electroencephalography (EEG) are neurophysiological techniques that provide a direct measure of neuronal activity by capturing the resulting magnetic field or electric potential that are generated by electrical activity within neurons (Hamalainen et al., 1993). The primary advantages of these techniques over fMRI is that they provide a direct measure of neuronal activity

and high temporal resolution (on the order of milliseconds) to capture the temporal dynamics of brain activity (Hamalainen & Lundqvist, 2019; Hari & Puce, 2017). Differences in evoked responses measured with MEG/EEG are largely attributed to activation of a different number of neurons perhaps due to reduced neuronal density or desynchronization of the evoked response potentially due to altered conduction velocities, which indicate different underlying mechanisms. Despite these advantages, there remain few studies (Bolanos et al., 2017; Candelaria-Cook, Schendel, et al., 2022; Coffman et al., 2013; D'angiulli et al., 2006; Stephen et al., 2012) using these techniques to understand the effects of PAE on brain function across development with many of the early EEG studies focusing on resting brain dynamics in infants (Chernick et al., 1983; Havlicek et al., 1977; Ioffe et al., 1984). The most relevant study is a recent EEG study (Gerhold et al., 2017) using an auditory Go/No-go task in children with PAE and identified early decreases (N2/P2) and later increases (late potential) in amplitude in individuals with PAE relative to controls.

MEG has additional advantages over EEG as there is no spatial smearing when the signal of interest passes through the skull in MEG as exists in EEG (Puce & Hamalainen, 2017). Therefore, MEG facilitates accurate source localization of neural activity and provides strong potential for identifying neurophysiological changes due to PAE. In a paradigm assessing visual processing, peak latency of the M100 responses localized to the occipital cortex was delayed in adolescents with FASD compared to controls during a prosaccade task (Coffman et al., 2013). Consistent with auditory deficits in rats prenatally exposed to alcohol (Church, 1987; Church et al., 2012; Church & Gerkin, 1988) children with FASD also reveal altered auditory processing in children 3–6 years of age (Stephen et al., 2012). These findings align with multiple previous studies (Carr et al., 2010; Jirikowic et al., 2020; Margret et al., 2006; Wang et al., 2022) showing regionally distinct deficits in FASD compared to TDC in multiple sensory modalities.

1.4 | Go/No-go and associated brain structures

In addition to the studies described above, children with an FASD diagnosis demonstrate consistent attention deficits as revealed in part by the prevalence of co-morbid diagnoses of Attention-Deficit/Hyperactivity Disorder (ADHD), which is estimated to occur in 70% of children with FASD (O'Malley & Nanson, 2002). A common task to assess attention deficits in children and adults is the Go/No-go task. It is especially suitable for children based on the simple instructions, limiting the confound of performance differences due to comprehension difficulties. The Sustained Attention to Response Task (SART), a Go/No-go task in which digits 1–9 are presented either randomly or in numerical order with digit 3 denoted as the No-go stimulus, assesses inhibition and attention, both domains affected by PAE.

The literature implicates two primary regions crucial to cognitive control during Go/No-go tasks: the ventrolateral prefrontal cortex (VLPFC), including Broadmann's Area 44 (BA44), and the Anterior Cingulate Cortex (ACC). Specifically, BA44 is integral in employing top-down controlled inhibition (Aron et al., 2007; Aron & Poldrack, 2006; Rae et al., 2014). Rae et al. (2014) found bilateral activation of the lateral PFC, including BA44, associated with stopping actions during a Go/No-go task, while others found activation in these areas specifically to the detection of a meaningful and behaviorally significant target, such as the stimuli signaling Go/No-go behaviors (Cai & Leung, 2011; Hampshire et al., 2010). This indicates that BA44 is involved in both the detection of relevant stimuli and also in the action outcome associated with the stimuli. Activation of BA44 may also be related to the required button response (Koechlin & Jubault, 2006; Rizzolatti et al., 2002). Many studies assessing brain activity during a Go/No-go task have found right lateralization of BA44 activity (Aron et al., 2014; Levy & Wagner, 2011; Mazzone, 2014), while morphological studies have found right hemisphere BA44 cortical thickness predicts inhibitory performance in TDC 4–13 years of age (Curley et al., 2018). Additional differences are expected when stopping is unpredictable, as would occur when digits are presented randomly in SART_Random (SART_R), compared to designs in which stopping is predictable, such as in SART_Fix (SART_F) when digits are presented in numerical order (Jahfari et al., 2010; Verbruggen & Logan, 2009).

The Anterior Cingulate Cortex (ACC) is involved in the processes required to inhibit responses, as occurs in Go/No-go tasks (Braver et al., 2001; De Zubicaray et al., 2000; Durston et al., 2002). Additionally, the ACC is involved in outcome evaluation (i.e., error detection) and decision-making (i.e., to Go/No-go) (Botvinick, 2007). Specifically, the ACC functions in part to identify conflicts in information processing, a process known as conflict monitoring (Botvinick, 2007). ACC activity with error production is also found in EEG (Falkenstein et al., 2000), event-related fMRI (Carter et al., 1998), and in single-unit recording (Amiez et al., 2005) studies. In part, the ACC drives reactive alterations in control by evaluating action outcomes (Botvinick, 2007). Interestingly, in a study assessing the impact of the frequency of No-go stimuli on ACC functioning, one group found the ACC paramount in conflict monitoring when low-frequency (17% No-go stimuli) responses are implemented (Braver et al., 2001). This is important for our study, as the No-go stimuli occur at a frequency of 11% in the SART. The ACC, while involved in many processes, appears vital for response inhibition, action selection, attentional control, and outcome evaluations, all processes required in Go/No-go paradigms.

Numerous studies have assessed brain activity during the SART. In a SART_F ERP study in neurologically normal, right-handed volunteers ages 18–32 years, Dockree et al. (2005) found increased frontocentral activation on No-go trials, suggesting a central inhibitory mechanism intervenes to prevent the preparation and execution of a dominant motor response. Highlighting differences between fixed and random order SART, Zordan et al. (2008) looked at SART using a random order (SART_R), comparing their results to Dockree et al.'s (2005) SART_F data. They found that increased activity in right PFC was associated with fixed but not random orders. Additionally, in SART_R only, correct inhibitions increased activity in right, ventral frontal cortex and inferior parietal lobe. Interestingly, Robertson et al. (1997)

found that the decrement in performance during SART was related more to a decrease in sustained attention rather than failed inhibition. As SART_F and SART_R assess inhibition and sustained attention, these tasks are especially well suited to assess differences between FASD and HC.

Studies assessing markers of dysfunction in individuals with FASD of all age groups indicate that the functional deficits due to PAE are not confined to one specific age, behavior, brain region, or task. In this study we sought to identify the brain-function differences with high spatial/temporal resolution between children with an FASD and HC children aged 8–12 years to examine the underlying mechanisms related to attention and inhibitory control deficits experienced by children with an FASD. To assess neural activity via MEG, we administered the SART fixed and random orders. We analyzed both latency and amplitude to assess these differences. We chose to limit our MEG analysis to the regions highlighted above, BA44 and ACC, as these are both critical for inhibition, attention, action selection, error detection, and conflict monitoring. We assessed differences in behavioral performance during the SART, including reaction time, omission errors, commission errors, hit rate, and error detection. We analyzed the associations between brain function, behavioral measures of SART performance, neuropsychological functioning, and caretaker ratings of child behavior. Based on the previous work, we evaluated two primary hypotheses: (1) We hypothesized that HC children would have greater brain signal amplitude and faster latencies based on source analysis of MEG data compared to children with an FASD diagnosis on both the SART_F and SART_R. (2) We hypothesized that HC children would have decreased reaction time, fewer omission and commission errors, and increased hit rates relative to children with an FASD diagnosis on both versions of the SART. An exploratory analysis examined associations between MEG amplitudes and brain volume estimates obtained from the structural MRI.

2 | MATERIALS AND METHODS

2.1 | Participants

The study was approved by the University of New Mexico Health Sciences Center Human Research Review Committee and complies with the Declaration of Helsinki. We recruited children 8–12 years old from Albuquerque, NM and the surrounding communities. Healthy control (HC) children had IQ scores within the average or above average ranges, had no known prenatal exposure to alcohol or other substances, and did not have histories of developmental delays or neurological or psychological problems. Participants were classified as having an FASD diagnosis using the Institute of Medicine Criteria (Hoyme et al., 2005; Stratton et al., 1996) at the UNM Center for Development and Disability FASD Clinic. All diagnoses were obtained by consensus based on evaluation by a clinical psychologist, a neuropsychologist, and a pediatrician with FASD-specific training in assessment of facial dysmorphology. Maternal alcohol consumption during pregnancy for the affected child was determined through direct confirmation by maternal interview, multiple eyewitness reports of maternal drinking during pregnancy, or legal records confirming alcohol consumption during pregnancy (e.g., DWI arrest). Children with PAE met FASD criteria for confirmed alcohol exposure. For this analysis, we assessed $n = 18$ HC and $n = 25$ FASD (2 PAE, 5 ARND, 4 PFAS, and 14 FAS), group-matched on age and sex.

2.2 | Procedure

The SART is a Go/No-go paradigm, which consists of both a random and fixed version (Figure 1). Participants were presented with digits (1–9), and were instructed to press a response button for all digits except for digit 3. During the fixed condition, the digits were presented in ascending order (SART_F), and during the random condition the digits were presented in random order (SART_R). The fixed condition provides predictable stimuli and allows participants to plan for executing or withholding a motor response. The digits were presented for 150 ms with a 900–1100 ms interstimulus interval. Each digit was presented with equal probability providing a No-go frequency of 11%. We collected 945 trials (105 trials/digit–15 min/version).

Data collection occurred on two different days within 2 weeks of each other. On day one, participants' brain responses were recorded using simultaneous MEG and electroencephalography (EEG not reported here) during SART_F and SART_R tasks. Participants also completed a prosaccade task and a resting task (not discussed here). On day two, participants' brain structure was obtained during an MRI scan (see details below), while watching a self-chosen child's show without commercials. Neuropsychological measures and parent/guardian questionnaires were also completed on testing day two.

2.3 | Neuropsychological testing

All participants completed the Wechsler Abbreviated Scale of Intelligence Second Edition (WASI-II): Vocabulary and Matrix Reasoning

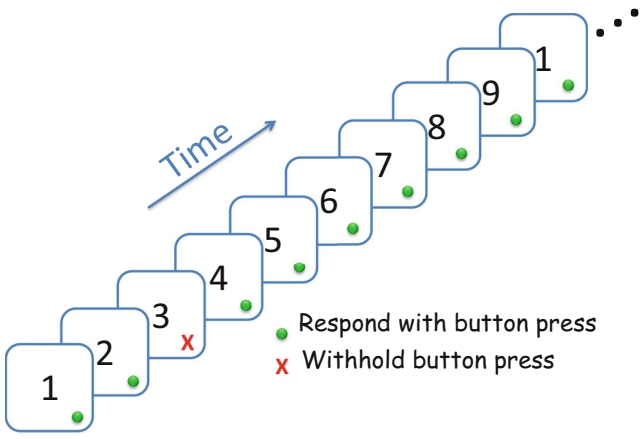

FIGURE 1 SART_F order task.

subscales (Wechsler, 2011), the Grooved Pegboard Test (GPB) (Company, 2002), and the Delis-Kaplan Trail Making Test (D-KEFS) (Delis et al., 2001).

2.4 | Parent/guardian questionnaires

Caretakers of the participants completed a set of questionnaires, including the Behavioral Rating Inventory of Executive Function (BRIEF), the Sluggish Cognitive Tempo Scale (SCTS) (Penny et al., 2009), the Conners 3-Parent Rating (C3P) scale (Conners, 1997), and the Barratt Simplified Measure of Social Status (BSSS) (Barratt, 2006) measuring socioeconomic status (SES). The BRIEF is a parent/guardian evaluation of child executive functioning. Negativity and Inconsistency subscale scores were considered when including data and no participants were excluded due to elevations on these subscales. The BRIEF consists of three composite scales: Global Executive Composite (GEC), Behavioral Regulation (BR), and Metacognition (MC), with lower *t*-scores representing better executive functioning. Sluggish Cognitive Tempo is a measurement of behaviors including inattention, underactivity, motivation, and drowsiness. The C3P is an instrument used to assess caretakers' perceptions of their child's ADHD behaviors, as well as common comorbid behaviors. Higher scores on this assessment represent poorer behavioral functioning.

2.5 | MEG data acquisition

MEG data were collected using the Elekta Neuromag 306 channel biomagnetometer (Elekta) located in a magnetically shielded room (Vacuumschmelze—Ak3B) at the Mind Research Network in Albuquerque, New Mexico. Electrocardiogram (ECG) and electrooculogram (EOG) electrodes were placed (ECG—just below the left and right clavicle, EOG—one electrode placed just above the left eye-brow and one placed lateral to the outer canthus of the right eye) to provide signals for artifact rejection of heartbeat and eye blinks/movements, respectively. MEG head position indicator (HPI) coils were placed around the head and secured with tape. HPI coil location and head shape information was obtained using the Polhemus 3-D tracking device (Polhemus). Three fiducial points (left and right preauricular and nasion) were identified to define the head-centered coordinate system in addition to points around the scalp to ease co-registration of the MEG data to the MRI structural image. The MEG data were digitized at 1000 Hz (0.01–300 Hz anti-aliasing filter). Continuous HPI recording was enabled throughout the MEG data collection.

2.6 | MRI data acquisition

All MRI images were obtained with a Siemens 3T Trio TIM scanner using the standard 32-channel phased array head coil provided with the system. Sagittal TI-weighted anatomical images were obtained with a multi-echo 3D MPRAGE sequence (TR/TE/TI = 2530/1.64, 3.5, 5.36, 7.22, 9.08/1200 ms, flip angle = 7°, field of view (FOV) = 256 × 256 mm, matrix slice thickness of 1 mm, 192 slices, GRAPA acceleration factor = 2. MRI data were processed through Freesurfer using reconall. MRI data were visually evaluated for data quality and quantitatively using the Freesurfer Euler number (Rosen et al., 2018). All MRIs obtained met quality control requirements and were included in the analysis. Total brain volume was estimated using the uncorrected Total Volume Freesurfer output variable. Left and right hemisphere frontal volumes were estimated by summing the volumes of frontal structures from the Desikan-Killiany regions of interest (superior frontal, rostral and caudal middle frontal, pars opercularis, pars triangularis and pars orbitalis, lateral and medial orbitofrontal, precentral, paracentral and frontal pole).

2.7 | MEG data analysis

The MEG data were preprocessed to eliminate artifacts. Scripted Neuromag Maxfilter (Taulu & Kajola, 2005) processing was performed on the raw data using the temporal signal space separation (TSSS) method to reduce distant and temporally correlated noise signals and to compensate for head movement during data collection (Elekta, Oy). Cardiac and eye blink artifacts were identified using the ECG and EOG channels respectively or anterior temporal MEG channels and were removed from the data using the signal space projection (SSP) method (Uusitalo & Ilmoniemi, 1997) as implemented by Neuromag Graph and Xfit software. Using a scripted shell program, MNE software (http://www.martinos.org/mne/) was employed to reject bad trials, apply the SSPs, band-pass filter the continuous data, differentiate correct from incorrect trials, and average the data by stimulus condition. Epochs were extracted relative to each condition (correct/incorrect, Go/No-go) with a 100 ms baseline and 700 ms post-stimulus interval. Data epochs with large amplitude artifacts, magnetic field at any sensor exceeding 5 pT, were rejected.

Next, we performed cortical source analysis of the MEG data using FreeSurfer, Neuromag, and MNE software packages. Using the FreeSurfer software Recon-all function, automatic cortical surface reconstruction and subcortical segmentation from MPRAGE MRIs was performed. Then, we created a bilateral hemisphere surface-based source space using eight times recursively subdivided octahedron spacing. Next, we automatically created boundary element model (BEM) meshes, using the MNE watershed algorithm (Segonne et al., 2004), which produces brain, inner skull, outer skull, and outer skin surface triangulation. Each surface triangulation was isomorphic with an icosahedron, which was recursively subdivided, yielding 5120 triangles. We utilized the inner skull surface for the single compartment forward model.

We calculated a forward solution using this subject-specific single layer BEM. The loose variable was set to 0.2, the MNE default for surface-oriented source space. For depth weighting the default coefficient of 0.8 was used. Cortical patch statistics were used to define normal orientations. Using dynamic statistical parametric mapping

(dSPM) (Dale et al., 2000), the inverse operator previously calculated was applied to the averaged evoked MEG responses for each condition. Next, a time course for each anatomical region was extracted using AAL defined regions for BA44 and ACC with the mean flip method, which averages the source estimates within each label with sign flips to reduce signal cancellation. These time courses were divided into empirically selected time windows of interest by visually inspecting all MEG time courses combined, MEG time courses by group, and then each individual subject's data to ensure observable peaks lie within the selected time windows. Visual inspection revealed three predominant peaks between times 100–200, 200–350, and 350–550 ms. Times are post stimulus onset.

Following source analysis, time courses from the regions localized to attention and inhibition networks (ACC and BA44) were processed further to extract peak amplitudes and latencies. A custom matlab script (Pervin et al., 2021; Stephen et al., 2002) evaluated the time-course for each individual region and participant relative to the user identified time intervals. The peak amplitude and latency were identified by finding the largest peak amplitude for each time course within each time window recording the amplitude and latency of this peak and written to file for statistical analysis.

2.8 | Statistical analysis

Independent samples *t*-tests were used to assess differences in age and SES. Chi-square analysis was used to ensure samples matched on sex.

We utilized ANCOVAs to evaluate the effect of SES on the group comparisons of SART task performance, neuropsychological evaluations, and parent/guardian questionnaires by including BSSS as the covariate in these models. If BSSS proved not to be a significant contributor to the model, independent samples *t*-tests were utilized to assess group differences in these measures.

To assess group differences in task-based neural activity, we performed a series of three-way ANCOVAs with BSSS as a covariate on SART_F and SART_R versions of the task. If BSSS proved not to be a significant contributor to the model, analysis was conducted again without BSSS as a covariate. We also utilized three-way ANCOVAs with trial count as a covariate on SART_F and SART_R versions of the task. We used three omnibus ANCOVAs to assess each time window separately for latency and two omnibus ANCOVAs to assess each region separately for amplitude. As the peak-latency measures are inherently different between time windows (i.e., 100–200, 200–350, 350–550 ms), we assessed group differences in latencies for group × hemisphere × ROI interactions for each time window. Group included two levels (HC, FASD), hemisphere included two levels (left hemisphere (LH), right hemisphere (RH)), and ROI consisted of two levels (ACC, BA44). We assessed group differences in amplitude for group × hemisphere × time-window interactions for each ROI (ACC, BA44) individually. In the amplitude analyses, group included two levels (HC, FASD), hemisphere included two levels (left, right), and time-window consisted of three levels (100–200, 200–350, and 350–550 ms).

Finally, we used Pearson's correlations to assess the relationships between MEG and neuropsychological evaluations, MEG and parent/guardian questionnaires, MEG and task-based performance/behavior on the SART, and MEG. Group differences in these relationships were analyzed using Fisher's *r*-to-*z* transformation. In exploratory analyses, we examined correlations between peak amplitude for variables that revealed significant group differences relative to total and frontal brain volume.

All analyses were corrected for multiple comparisons using the FDR linear step up procedure (FDR LSU) (Maxwell & Delaney, 2004). To account for differing sample sizes, we used Hedge's *g* to calculate effect sizes. All *p*-values reported are corrected using the Benjamini–Hochberg procedure (Benjamini & Hochberg, 1995).

3 | RESULTS

3.1 | Demographics

Inclusion criteria required an intelligence quotient (IQ) score of ≥70 as determined by the WASI-II two-subtest IQ score. Three of 25 FASD participants scored lower than 70. However, two of these participants scored above threshold on the WASI-II Matrix Reasoning subscale, suggesting the deficit was driven by lower than normal scores on the WASI-II Vocabulary subscale. It is well-documented that assessments of this type are culturally biased to the benefit of English-only speaking individuals (Garratt & Kelly, 2008). Given the number of dual-language household participants in our community, lower scores on the WASI-II Vocabulary were expected (Garratt & Kelly, 2008). Therefore, we only removed one FASD participant from analyses, as the IQ score was below threshold (IQ = 56) on both WASI-II subscales, resulting in FASD $N = 24$. The individuals who were retained were able to perform the SART as demonstrated by hit rates >65% in the more challenging SART_R providing support for the retention of these datasets.

Chi-square analysis revealed groups were well matched on sex: HC (female, $n = 13/18$), FASD (female, $n = 16/24$), $\chi^2(1) = 0.15$, $p = .70$. A *t*-test revealed no significant differences in age between groups: HC ($M = 9.67$, $SD = 1.37$), FASD ($M = 10.00$, $SD = 1.69$), $t(40) = 0.68$, $p = .50$. However, we found a significant group difference on the BSSS: HC ($M = 48.44$, $SD = 9.15$), FASD ($M = 29.44$, $SD = 13.37$), $t(37) = 5.09$, $p < .001$. We were unsuccessful in obtaining the BSSS measure from three FASD participants. Considering the relationship SES has with multiple measures (Perkins, 2016; Sirin, 2005), we performed analyses with and without SES as a covariate.

3.2 | Neuropsychological evaluations

We utilized ANCOVAs to control for the effect SES has on these group comparisons by including BSSS as the covariate. The BSSS failed to have a significant contribution to these models. Therefore, all

TABLE 1 Neuropsychological evaluations.

Measure	HC *M* (*SD*)	FASD *M* (*SD*)	*t*-Statistic	Adjusted *p*-value	Hedges' *g*
WASI-II: vocabulary (*t* score)	61.67 (13.69)	36.30 (13.96)	$t(39) = 5.82$	<.001	1.83
WASI-II: matrix reasoning (*t* score)	48.94 (6.50)	40.50 (8.06)	$t(38) = 3.59$	.001	1.13
WASI-II: full scale—2 (standard score)	110.72 (13.59)	79.22 (10.33)	$t(39) = 8.44$	<.001	2.66
Grooved Pegboard: Dominant Hand (*z* score)	−0.25 (0.62)	1.62 (1.90)	$t(38) = -4.00$	<.001	1.25
Grooved Pegboard: non-dominant hand (*z* score)	−0.10 (0.62)	1.51 (1.97)	$t(38) = -3.32$	.002	1.04
D-KEFS TMT 1: visual scanning (scaled score)	9.61 (3.42)	8.52 (3.93)	$t(37) = 0.91$	.37	0.29
D-KEFS TMT 2: number sequencing (scaled score)	8.33 (4.20)	6.33 (3.62)	$t(37) = 1.60$	.12	0.51
D-KEFS TMT 3: letter sequencing (scaled score)	8.83 (3.33)	6.52 (4.08)	$t(37) = 1.92$	.06	0.62
D-KEFS TMT 4: number/letters switching (scaled score)	8.5 (3.97)	3.95 (3.63)	$t(37) = 3.69$	.001	1.20
D-KEFS TMT 5: motor speed (scaled score)	8.11 (3.68)	8.47 (2.98)	$t(37) = -0.34$	.73	0.11

TABLE 2 Parent/guardian questionnaires—BRIEF and Sluggish Cognitive Tempo Scale.

Measure	HC, *M* (*SD*)	FASD, *M* (*SD*)	*t*-Statistic	Adjusted *p*-value	Hedges' *g*
BRIEF: GEC	49.78 (8.15)	66.43 (11.74)	$t(39) = -5.13$	.017	1.60
BRIEF: behavioral regulation	49.56 (9.13)	66.5 (14.99)	$t(38) = -4.20$	.03	1.32
BRIEF: metacognition	50.00 (7.89)	65.95 (11.12)	$t(38) = -5.12$	.019	1.61
Sluggish Cognitive Tempo Scale	8.56 (4.51)	16.27 (7.35)	$t(38) = -3.89$	.03	1.22

neuropsychological analyses reported are independent samples *t*-tests.

The IQ score was derived from the Vocabulary and Matrix Reasoning subscales of the WASI-II. The Vocabulary and Matrix Reasoning subscale scores and the IQ Composite scores were found to be significantly different between groups (Table 1). On the WASI-II Vocabulary and the Matrix Reasoning subscales, HC performed significantly better than FASD; therefore, HC measured significantly higher on IQ scores than the FASD group. The Grooved Pegboard Test (GPB), measuring manual dexterity and eye–motor coordination, revealed significant differences between groups. Mean scores reported were the age-normed *z*-scores. There were significant group differences between both the dominant hand and non-dominant hand, with better performance by the HC group. The D-KEFS Trail Making Test includes five conditions that assess visual scanning, motor speed, and executive functioning. Only the Number/Letter Switching condition showed significant group differences in performance with HC performing significantly better than FASD.

3.3 | Parent/guardian questionnaires

We utilized ANCOVAs to evaluate the effect SES had on group comparisons of parent/guardian questionnaires by including SES score as the covariate in these models. SES was found to have no significant contributions to the analyses except for one subscale, the Conners-3 Parent, Conduct Disorder. Therefore, all other parent/guardian questionnaire analyses reported are independent samples *t*-tests (Tables 2 and 3).

Compared to the FASD group, HCs were rated as having better overall executive functioning, behavioral regulation, and metacognition (Table 2: GEC). These findings indicate that caretakers of HC rate their children as having better/more intact executive functioning, behavioral regulation, and metacognition abilities compared to caretakers of children with FASD. Caretakers rated HC lower on Sluggish Cognitive Tempo than FASD (Table 2) with higher scores associated with more sluggish behavior in FASD (child appears apathetic, sleepy, etc.). After FDR LSU correction for multiple comparisons, all subscales revealed significant group differences between HC and FASD groups, with the FASD group rated as having more ADHD-type behaviors (Table 3). The largest differences between groups were found on the Inattention subscale, Learning Problems subscale, and the ADHD Index.

3.4 | Sustained Attention to Response Task behavioral performance

To assess behavioral performance we evaluated reaction time, hit rate, and signal detection via *d*-prime (*d*′), separately for SART_R and SART_F. Due to the predictable nature of SART_F, digit 9 signals a reset in participants giving it a different consideration than other 'go' digits. Digits 1 and 2 signal a preparatory period for the upcoming 'no-go' digit 3 (Dockree et al., 2005). To establish a comparable variable to the SART_R 'go' variable, random all go (RAG_HR), we combined digits 4–8 'go' into one variable labeled Fix_4to8_go for both reaction time correct trials (Fix_4to8_cRT) and hit rate (Fix_4to8_HR). After FDR LSU for multiple comparisons, there were no group differences in behavioral performance during the SART_F (Table 4).

TABLE 3 Parent/guardian questionnaires—Conners 3 Parent.

Measure	HC, *M* (*SD*)	FASD, *M* (*SD*)	*t*-Statistics	Adjusted *p*-value	Hedges' *g*
Inattention	52.56 (7.66)	74.23 (11.42)	$t(38) = -6.88$	.003	2.17
Hyperactivity/impulsivity	54.11 (10.28)	70.59 (16.11)	$t(38) = -3.76$	.039	1.18
Learning problems	49.44 (6.77)	75.27 (13.47)	$t(38) = -7.40$	.006	2.32
Executive function	53.44 (8.69)	71.50 (12.30)	$t(38) = -5.24$	.011	1.65
Defiance/aggression	52.28 (10.19)	69.59 (19.40)	$t(38) = -3.42$	.042	1.07
Peer relations	47.83 (7.70)	68.41 (17.34)	$t(38) = -4.66$	.022	1.46
ADHD inattentive	52.28 (7.80)	72.63 (14.27)	$t(35) = -5.34$	.014	1.70
ADHD hyperactive/impulsive	53.83 (9.52)	70.00 (16.37)	$t(35) = -3.65$	.036	1.17
Conduct disorder	51.67 (11.97)	67.68 (20.63)	$t(35) = -2.87$	.044	0.92
Defiant disorder	52.83 (10.62)	64.44 (18.96)	$t(34) = -2.29$	.050	0.73
ADHD index	18.83 (16.32)	72.00 (34.04)	$t(34) = -5.98$	.008	2.00
GI restless impulsive	53.28 (8.41)	71.84 (15.67)	$t(35) = -4.45$	.025	1.42
GI emotional lability	51.33 (9.67)	63.61 (17.34)	$t(34) = -2.83$	.047	0.84
GI total	52.61 (8.47)	72.44 (17.08)	$t(34) = -4.41$	.028	1.41

TABLE 4 SART fixed order—task-based behavioral performance.

Measure	HC, *M* (*SD*)	FASD, *M* (*SD*)	*t*-Statistic	Adjusted *p*-value	Hedges' *g*
Fix_4to8_cRT (ms)	230.37 (109.73)	256.24 (97.45)	$t(39) = -0.80$	.43	0.25
Fix_3_false_alarm_RT (ms)	252.10 (120.71)	265.99 (95.39)	$t(39) = -0.41$	.68	0.13
Fix_4to8_go_HR	0.88 (.09)	0.80 (.13)	$t(39) = 1.45$	.15	0.70
Fix_3_false_alarm_rate	0.27 (0.15)	0.33 (0.16)	$t(39) = -1.34$	.19	0.39
Fix_*d*′	1.98 (1.11)	1.37 (0.89)	$t(39) = 1.96$	.06	0.62

TABLE 5 SART random order—task-based behavioral performance.

Measure	HC, *M* (*SD*)	FASD, *M* (*SD*)	*t*-Statistic	Adjusted *p*-value	Hedges' *g*
RAG_cRT (ms)	231.00 (68.04)	261.29 (57.43)	$t(39) = -1.55$	.13	0.49
Random_3_false_alarm_RT (ms)	187.94 (75.70)	214.14 (67.71)	$t(39) = -1.17$	.25	0.37
RAG_HR	0.89 (0.08)	0.76 (0.16)	$t(39) = 3.14$	.003	0.98
Random_3_false_alarm_rate	0.72 (0.14)	0.68 (0.12)	$t(39) = 0.99$	.33	0.31
Random_*d*′	0.71 (0.72)	0.31 (0.63)	$t(39) = 1.87$	.07	0.60

The random nature of SART_R allows for a combination of variables during analysis. Due to this, we combined reaction times for all Go stimuli into one variable, Random-All-Go correct trials reaction time (RAG_cRT), and all hit rate scores into one variable, Random-All-Go hit rate (RAG_HR). After FDR LSU correction for multiple comparisons, one variable revealed significant group differences (Table 5). HC had significantly higher RAG_HR than FASD. No other variables survived FDR LSU.

3.5 | Magnetoencephalography

As expected, MEG group-averaged responses revealed similar patterns of activation across the sensor array (Figure 2). Combined with the behavioral results these signals help demonstrate the ability to collect high quality data from participants during performance of the SART. To account for differences in head position within the helmet array, statistical analyses were conducted on the MEG source analyzed data described below.

To assess group differences in task-based brain activity, we performed a series of 3-way ANCOVAs with BSSS as covariate on peak-latency from SART_F and SART_R versions of the task. FDR LSU corrections for multiple comparisons did not reveal any significant group differences in peak-latency in SART_F or SART_R versions of the task.

A three-way ANCOVA was conducted to examine the influence of three independent variables (group, hemisphere, and time window) with BSSS as covariate on the peak-amplitude from SART_F and

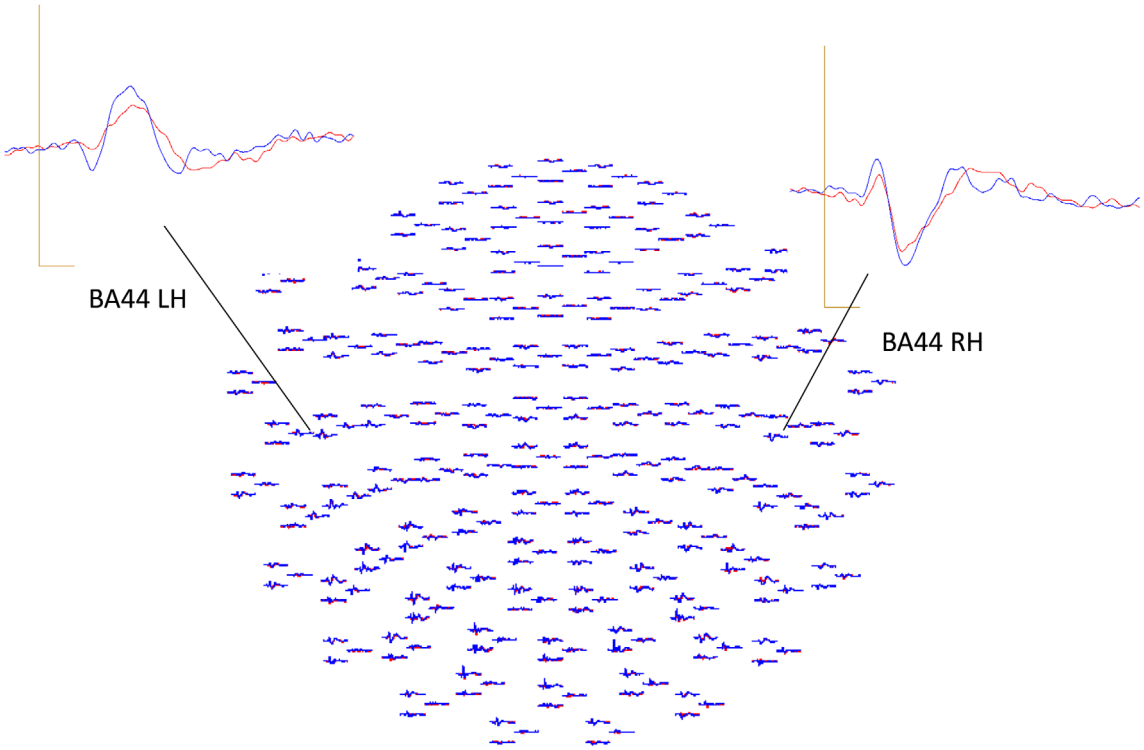

FIGURE 2 Group averaged responses (blue—HC, red—FASD) are shown in the MEG sensor data. The MEG sensor map provides a top-down view of all 306 sensors with sensors at the top/bottom denoting anterior/posterior sensor locations and left and right showing sensors over left and right hemispheres, respectively. Insets show a larger view of two sensors located over left and right BA44 regions, respectively.

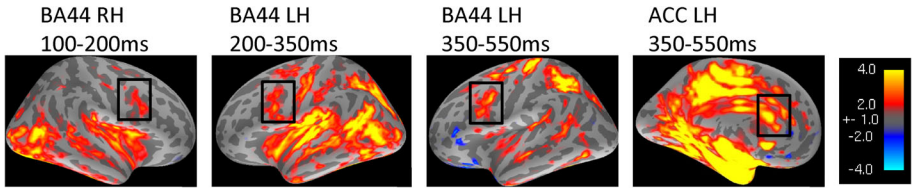

FIGURE 3 Group differences in source analyzed response in three time windows for BA44 and the third time interval for ACC are shown. Hot colors refer to responses where HC > FASD, cool colors refer to responses where HC < FASD.

SART_R for each ROI (ACC, BA44: Figure 3). BSSS was not a significant contributor to the ANCOVA model and was therefore removed from analyses. Considering we also found a significant group difference in the number of trials retained after artifact rejection (Trial_Count): HC ($M = 598.56$, $SD = 187.60$), FASD ($M = 474.04$, $SD = 168.64$), $t(40) = 2.26$, $p = .03$, we used a 3-way ANCOVA with Trial_Count as covariate. Trial_Count significantly contributed to the model. Therefore, all MEG results are reported with Trial_Count as a covariate. All p-values reported are adjusted p-values. After FDR LSU corrections for multiple comparisons, peak-amplitude in BA44 during SART_R All-Go trials showed a significant 3-way interaction, $F(1,39) = 11.43$, $p = .004$ (Figure 4, left). Additionally, peak-amplitude in ACC during SART_R All-Go trials showed a significant three-way interaction, $F(1,39) = 6.28$, $p = 0.017$ (Figure 4, right). No other three-way interactions survived FDR LSU.

Of the three-way interaction found in BA44, simple interaction tests revealed significant two-way interactions with Trial_Count as a covariate between group and hemisphere at time window 100–200 ms $F(1,39) = 6.54$, $p = .03$, at time window 200–350 ms $F(1,39) = 11.06$, $p = .012$, and at time window 350–550 ms $F(1,39) = 9.02$, $p = .03$. In time window 100–200 ms, simple main effects analysis revealed heightened amplitude in HC ($M = 4.09$, $SD = 2.01$) compared to FASD ($M = 2.57$, $SD = 0.93$) in the left hemisphere $F(1,39) = 5.82$, $p = .04$, Hedges' $g = 1.16$, and in HC ($M = 4.68$, $SD = 2.29$) compared to FASD ($M = 2.80$, $SD = 2.05$) in the right hemisphere $F(1,39) = 5.20$, $p = .048$, Hedges' $g = 0.87$. In time window 200–350 ms, simple main effects analysis revealed heightened amplitude in HC ($M = 5.43$, $SD = 2.09$) compared to FASD ($M = 3.16$, $SD = 1.27$) in the left hemisphere $F(1,39) = 12.26$, $p = .012$, Hedges' $g = 1.36$, and increased amplitude in HC ($M = 5.76$, $SD = 2.21$) compared to

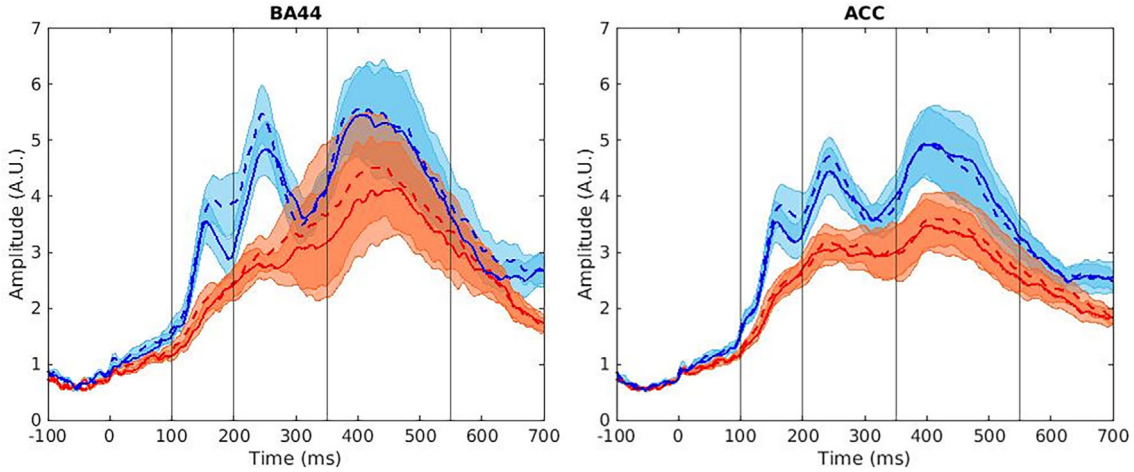

FIGURE 4 Group averaged MEG time courses for bilateral BA44 and ACC. HC averaged time courses are shown in blue and FASD averaged time courses are shown in red. Left hemisphere is denoted by the solid line and right hemisphere is denoted by the dashed line. The blue and red shaded regions denote the standard error.

FASD ($M = 3.52$, $SD = 1.74$) in the right hemisphere $F(1,39) = 7.72$, $p = .032$, Hedges' $g = 1.15$. In time window 350–550 ms, simple main effects analysis showed heightened amplitude in HC ($M = 5.13$, $SD = 1.67$) compared to FASD ($M = 3.39$, $SD = 1.47$) in the left hemisphere $F(1,39) = 8.20$, $p = .032$, Hedges' $g = 1.12$, and increased amplitude in HC ($M = 6.62$, $SD = 3.96$) compared to FASD ($M = 3.66$, $SD = 1.68$) in the right hemisphere $F(1,39) = 6.11$, $p = .042$, Hedges' $g = 1.03$ (Figures 3 and 4).

Of the three-way interaction found in ACC, simple interaction tests revealed significant two-way interactions with Trial_Count as a covariate between group and hemisphere at time window 100–200 ms $F(1,39) = 5.69$, $p = .03$, not at time window 200–350 ms $F(1,39) = 3.44$, $p = .071$, but again at time window 350–550 ms $F(1,39) = 4.84$, $p = .04$. In time window 100–200 ms, simple main effects analysis revealed no significant differences in amplitude in HC ($M = 3.61$, $SD = 1.67$) compared to FASD ($M = 3.11$, $SD = 1.15$) in the left hemisphere $F(1,39) = 2.48$, $p = .15$, Hedges' $g = 0.36$, no significant differences in amplitude in HC ($M = 4.44$, $SD = 1.83$) compared to FASD ($M = 3.04$, $SD = 1.24$) in the right hemisphere $F(1,39) = 3.68$, $p = .08$, Hedges' $g = 0.92$. In time window 200–350 ms, simple main effects analysis revealed no significant differences in amplitude in HC ($M = 4.29$, $SD = 1.81$) compared to FASD ($M = 4.07$, $SD = 1.93$) in the left hemisphere $F(1,39) = 0.75$, $p = .43$, Hedges' $g = 0.12$, no significant differences in amplitude in HC ($M = 5.12$, $SD = 1.33$) compared to FASD ($M = 3.76$, $SD = 1.61$) in the right hemisphere $F(1,39) = 3.71$, $p = .08$, Hedges' $g = 0.91$. In time window 350–350 ms, simple main effects analysis revealed heightened amplitude in HC ($M = 5.60$, $SD = 3.66$) compared to FASD ($M = 3.63$, $SD = 1.40$) in the left hemisphere $F(1,39) = 6.62$, $p = .04$, Hedges' $g = 0.75$, but no significant differences in HC ($M = 5.43$, $SD = 2.73$) compared to FASD ($M = 4.04$, $SD = 2.48$) in the right hemisphere $F(1,39) = 0.38$, $p = .54$, Hedges' $g = 0.53$ (Figures 3 and 4).

Finally, to assess associations between neural activation during SART and child neuropsychological evaluations, caretaker ratings of child behavior, and task-based performance during SART, Pearson's correlations were calculated only on variables found to be significantly different between groups, as reported above. Within the neuropsychological evaluations, these analyses revealed significant associations between MEG and all three WASI measures (Table 6), in that the

TABLE 6 Correlations between MEG and neuropsychological evaluations.

Variable	Pearson's *r*	Adjusted *p*-value
RAG_LH_BA44_100–200 ms		
WASI Full-Scale IQ	0.443	.014
WASI Vocab	0.418	.021
RAG_RH_BA44_100–200 ms		
WASI Full-Scale IQ	0.532	.005
WASI Vocab	0.469	.01
WASI Matrix Reasoning	0.445	.01
DKEFS TMT 4	0.413	.03
RAG_LH_BA44_200–350 ms		
WASI Full-Scale IQ	0.508	.006
WASI Vocab	0.494	.007
RAG_RH_BA44_200–350 ms		
WASI Vocab	0.464	.01
WASI Full-Scale IQ	0.45	.01
RAG_LH_BA44_350–550 ms		
WASI Full-Scale IQ	0.516	.006
WASI Vocab	0.44	.01
WASI Matrix	0.425	.02
DKEFS TMT 4	0.389	.04
RAG_RH_BA44_350–550 ms		
WASI Vocab	0.587	.005
WASI Full-Scale IQ	0.541	.005
DKEFS TMT 4	0.389	.04
RAG_LH_ACC_350–550 ms		
WASI Full-Scale IQ	0.375	.04

TABLE 7 Correlations between MEG and parent/guardian questionnaires.

Variable	Pearson's *r*	Adjusted *p*-value
RAG_LH_BA44_100–200 ms		
C3P_Executive_Function	−0.042	.04
RAG_RH_BA44_100–200 ms		
C3P_Executive_Function	−0.447	.03
RAG_LH_BA44_200–350 ms		
C3P_ADHD_Inattentive	−0.525	.03
C3P_Executive_Function	−0.50	.03
C3P_GI_Total	−0.485	.03
C3P_GI_Restless_Impuslive	−0.476	.03
C3P_Learning_Problems	−0.476	.03
C3P_Inattention	−0.474	.03
C3P_ADHD_Index	−0.473	.03
C3P_Hyperactivity_Impulsivity	−0.446	.03
C3P_ADHD_Hyperactive_Impulsive	−0.427	.05
BRIEF T Metacognition	−0.415	.05
BRIEF T Executive Composite	−0.406	.05
RAG_RH_BA44_200–350 ms		
C3P_ADHD_Inattentive	−0.48	.03
C3P_GI_Restless_Impuslive	−0.477	.04
C3P_Executive_Function	−0.469	.03
C3P_GI_Total	−0.459	.04
C3P_Learning_Problems	−0.451	.03
C3P_Inattention	−0.419	.04
RAG_LH_BA44_350–550 ms		
C3P_Executive_Function	−0.484	.03
C3P_Learning_Problems	−0.48	.03
C3P_ADHD_Index	−0.471	.03
C3P_ADHD_Inattentive	−0.459	.03
C3P_Inattention	−0.408	.05

stronger the peak amplitude in BA44 and ACC, the better the WASI-II performance. Significant associations were seen between BA44 peak amplitude and DKEFS TMT 4 performance (Table 6), in that the stronger the peak amplitude the better the performance. No significant correlations were found with GPB. Significant associations between BA44 peak amplitude and caretaker questionnaires were discovered in multiple variables within BRIEF and C3P (Table 7), showing in general that the stronger the MEG peak amplitude the better the executive functioning and the lower the ratings on inattention/ADHD behaviors. No significant correlations were found in SCTS. Finally, all six significant simple main effects in the BA44 MEG time-points significantly related to the SART behavioral variable, RAG_hit rate, showing that as MEG peak amplitude increases, so does the hit rate in SART Random go trials (Table 8). This relationship was lacking in the ACC. Within-group analyses of these associations revealed consistent directionality of

TABLE 8 Correlations between MEG and SART Random Task Behavior/Performance.

SART random order—all go trials—hit rate		
Variable	**Pearson's *r***	**Adjusted *p*-value**
RAG_LH_BA44_100–200 ms	0.424	.008
RAG_RH_BA44_100–200 ms	0.54	.001
RAG_LH_BA44_200–350 ms	0.528	.001
RAG_RH_BA44_200–350 ms	0.501	.002
RAG_LH_BA44_350–550 ms	0.342	.03
RAG_RH_BA44_350–550 ms	0.421	.008

relationships. Due to decreased sample size when analyzing by group, none of these associations by group survived corrections for multiple comparisons. Further, between-group analyses using Fisher *r*-to-*z* comparisons revealed no group differences in these associations after FDR.

3.6 | Exploratory analysis

To assess if our results in BA44 and ACC could be affected by brain structure, we conducted exploratory analyses to assess group differences in total brain, left frontal lobe, and right frontal lobe volumes. We also assessed if there were any relationships between these volume estimates and MEG peak amplitude. As our MEG results were reported with trial_count as a covariate, we correlated the peak amplitude residuals relative to trial_count with volume. Structural measures were available on 16 healthy control and 16 FASD subjects. Subjects with FASD ($M = 1{,}116{,}644$, $SD = 152{,}836.41$) had significantly smaller total brain volume than HCs ($M = 1{,}241{,}201.78$, $SD = 772{,}77.67$), $t(30) = 2.91$, $p = .008$. Subjects with FASD ($M = 95{,}550.50$, $SD = 11796.50$) had significantly smaller left frontal lobe volume than HCs ($M = 106{,}494.38$, $SD = 6992.38$), $t(30) = 3.19$, $p = .006$). Subjects with FASD ($M = 93{,}355.88$, $SD = 10{,}561.12$) had significantly smaller right frontal lobe volume than HCs ($M = 104{,}572.88$, $SD = 6220.94$), $t(30) = 3.66$, $p = .003$. We utilized FDR LSU for corrections for multiple comparisons and reported adjusted *p*-values.

Utilizing Pearson's correlations, we assessed how these volumetric measures correlated with peak amplitude in variables found to be significantly different between groups, as previously detailed. Total brain volume significantly correlated with right hemisphere BA44 peak amplitude during time window 200–350 ms ($r = 0.48$, $p = .02$). Left frontal volume significantly correlated with left hemisphere BA44 peak amplitude during time windows 200–350 ms ($r = 0.53$, $p = .01$) and 350–550 ms ($r = 0.48$, $p = .02$). Right frontal volume significantly correlated with right hemisphere BA44 peak amplitude during time window 200–350 ms ($r = 0.57$, $p = .01$). No other associations survived corrections for multiple comparisons (see Table 9).

To ensure these relationships were not driven by the natural distribution of our sample (i.e., if all FASD had low volume and low amplitude while all HC had high volume and high amplitude, this could produce strong correlations when assessing combined groups), we

TABLE 9 Correlations between volume measures and MEG peak amplitude residuals.

Structural variable	Peak amplitude residuals with trial count	Pearson's *r*	Adjusted *p*
Total volume	RAG_lh_AC_350_550_peak	0.16	.37
	RAG_lh_BA44_100_200_peak	0.27	.18
	RAG_lh_BA44_200_350_peak	0.41	.0525
	RAG_lh_BA44_350_550_peak	0.39	.057
	RAG_rh_BA44_100_200_peak	0.34	.099
	RAG_rh_BA44_200_350_peak	0.48	.0175*
	RAG_rh_BA44_350_550_peak	0.24	.22
Left frontal volume	RAG_lh_AC_350_550_peak	0.23	.23
	RAG_lh_BA44_100_200_peak	0.33	.11
	RAG_lh_BA44_200_350_peak	0.53	.0105*
	RAG_lh_BA44_350_550_peak	0.48	.018*
Right frontal volume	RAG_rh_BA44_100_200_peak	0.38	.057
	RAG_rh_BA44_200_350_peak	0.57	.0105*
	RAG_rh_BA44_350_550_peak	0.27	.18

*adjusted *p*-value < 0.05.

assessed associations by group only in variables found to be significantly correlated when group data was combined. When assessing by group, all of the relationships between significant variables remained consistent in directionality of correlations. Neither HC ($r = 0.32$, $p = .22$) nor FASD ($r = 0.37$, $p = .16$) revealed significant relationships between total brain volume and right hemisphere BA44 peak amplitude during time window 200–350 ms. When assessing left frontal volume with left hemisphere BA44 during time window 200–350 ms, HC ($r = 0.38$, $p = .14$) did not show a significant relationship, but FASD ($r = 0.53$, $p = .04$) was significantly associated. Conversely, HC ($r = 0.55$, $p = .03$) had a significant correlation between left frontal volume with left hemisphere BA44 during time window 350–550 ms, while FASD did not ($r = 0.28$, $p = .29$). Finally, HC ($r = 0.30$, $p = .26$) did not have a significant relationship between right frontal volume and right hemisphere BA44 during time window 200–350 ms, but FASD did ($r = 0.54$, $p = .03$) (Figure 5). Further, to ensure these by-group relationships were not significantly different from each other, we utilized Fischer *r*-to-*z* transformation. There were no significant differences between groups in any of these associations (*p*'s > .4).

4 | DISCUSSION

In summary, this study demonstrated that children aged 8–12 years with an FASD diagnosis experience deficits in attention and executive function and these deficits are mirrored by alterations in brain function. Specifically, BA44 exhibits a reduced amplitude response in children with an FASD diagnosis relative to HC children. However, the hemisphere related to this effect is dependent on the time window examined. The results further support that children with an FASD diagnosis are not significantly impaired on the less challenging SART_F, again providing evidence that executive functions under high-task demands are most likely to be impaired in this population. The specific results are discussed in more detail below.

4.1 | Neuropsychological evaluations

As expected based on diagnostic criteria, children with an FASD diagnosis showed decreased intellectual functioning, executive control, and fine motor abilities relative to HC children. As measured by the WASI-II, children prenatally exposed to alcohol exhibited lower performance for visual reasoning abilities, verbal abilities, and overall intellectual abilities. This diminished functionality was mirrored by decreased performance on the D-KEFS Trail Making Number-Letter Switching condition. While FASD and HC groups did not differ in baseline conditions assessing visual scanning, number sequencing, letter sequencing, and motor speed, the FASD group showed deficits in response inhibition and working memory as assessed by the more cognitively challenging subtest. This is important as it shows there is not a significant group difference in baseline abilities (visual scanning, number sequencing, and motor speed) required to perform the SART. Results from the GPB revealed bilateral deficiencies in fine motor speed and dexterity abilities in children with an FASD diagnosis consistent with Doney et al. (2014). Overall the results are consistent with a broad literature demonstrating reduced IQ and specific cognitive impairments in children with an FASD (Mattson et al., 2019).

4.2 | Parent/guardian questionnaires

Results from objective assessments of cognitive functioning were further supported by three subjective assessments from the children's caretakers. As indicated by the BRIEF, children with an FASD diagnosis are perceived as having decreased behavioral regulation (BRI) comprised of scores assessing inhibition, flexibility in thought and behavior, and emotional control. Caretakers also rate children with an FASD diagnosis as having decreased metacognition (MI) abilities, as measured by scores of initiation (ability to start a project and work/think/question independently), working memory, planning and

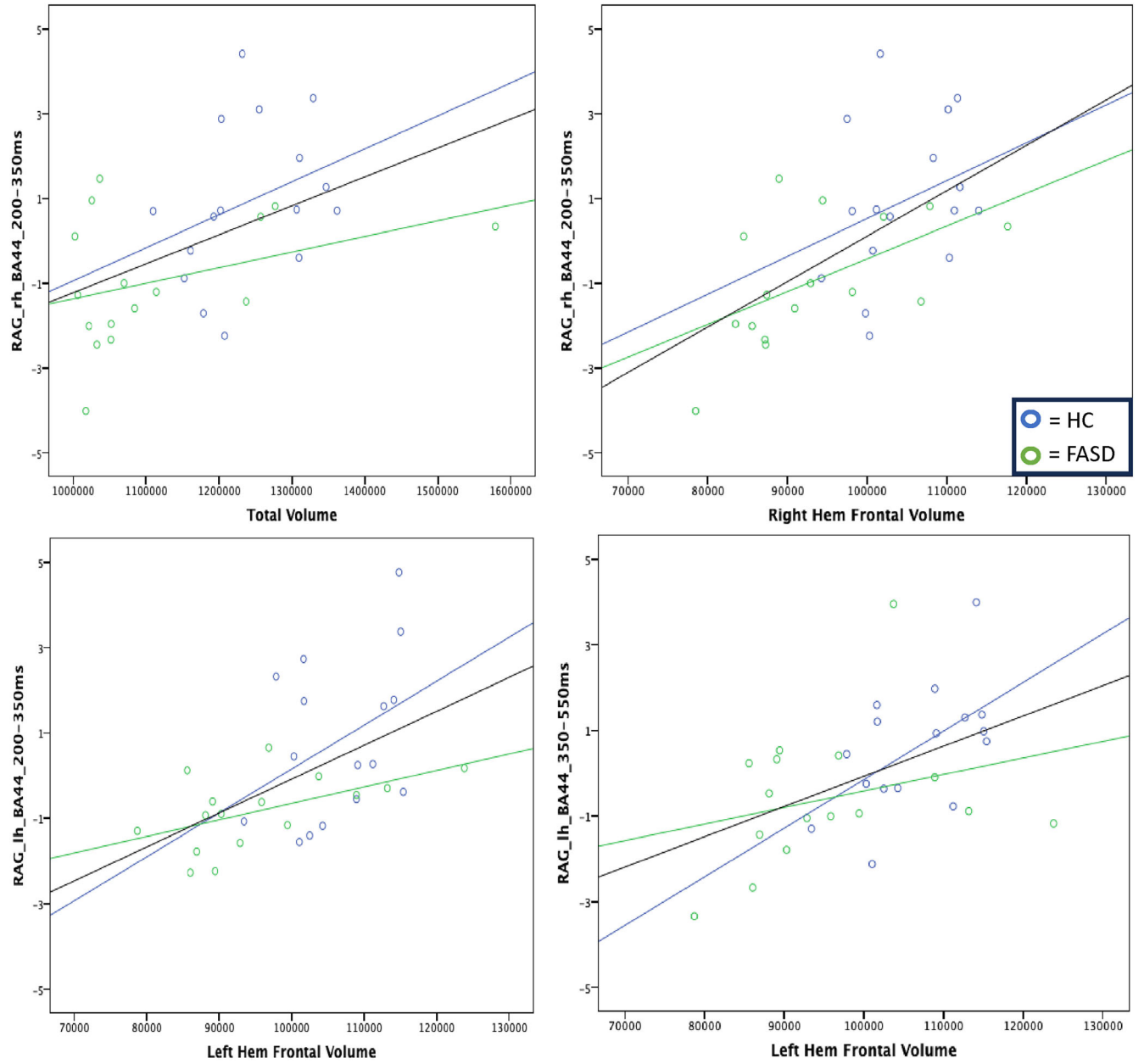

FIGURE 5 Associations between neurophysiologically-assessed neural response amplitude and brain volume measures with regression lines by group and combined (black regression line).

organization, organization of materials, and self-monitoring. Combining these two subscales, BRI and MI, revealed caretaker-observed executive dysfunction in children with an FASD diagnosis. Via the Sluggish Cognitive Tempo Scale, caretakers also rated children with an FASD diagnosis as having increased lack of motivation and slower information processing abilities, which both likely contribute to performance on neuropsychological evaluations and scores on parent/guardian questionnaires. As measured by C3P scale, caretakers evaluated children with an FASD diagnosis as having more hyperactivity, inattentiveness, learning problems, and difficulties in social interactions. These subjective caretaker ratings of executive functioning, motivation, and behavior corresponded to our participants' performances on objective measures of cognitive abilities and executive functioning, and likely contribute to the well-documented academic difficulties experienced by individuals with FASD (Glass et al., 2017; Streissguth et al., 2004).

4.3 | SART behavior

Given the differences in performance on well-established, reliable, and validated neuropsychological assessments and parent/guardian questionnaires, we expected to find differences in performance in the SART tasks. Interestingly, in the easier SART_F version of the task, we

found no significant behavioral differences in performance. However, when analyzing the more cognitively challenging SART_R, we found children with an FASD diagnosis had decreased ability to accurately 'Go' on all numbers. This is perhaps explained by the increased difficulty in the random task, decreased abilities in executive functioning elements that are required to succeed, or a combination of these and other factors. This is consistent with prior findings reporting that children with an FASD struggle on more complex tasks and can perform equivalently to controls on easier tasks (Kodituwakku & Kodituwakku, 2014).

4.4 | Magnetoencephalography

With significant differences found in behavioral performance on SART_R, next we examined the neurophysiological underpinnings of decreased functionality using MEG. Specifically, compared to the HC group, the FASD group had decreased amplitude bilaterally in BA44 during time window 100–200, 200–350, and 350–550 ms during all 'Go' trials of SART_R. Additionally, compared to the HC group, the FASD group had reduced amplitude in left ACC during the 350–550 ms time window. Brodmann Area 44 is classically known to be involved in speech production and processing different language components (Gelfand & Bookheimer, 2003), space perception, action understanding and imitation, hand movements (Rizzolatti et al., 2002), and music perception (Brown et al., 2006). More recent research has implicated BA44 as integral in response inhibition in a Go/No-go task (Forstmann et al., 2008). Specifically, Koechlin and Jubault (2006) found bilateral activation of Broca's area (including BA44) during a Go/No-go task signaling a system of executive processes that control action selection. Multiple studies have implicated the right inferior frontal cortex (IFC), which includes BA44, as being key in not only stopping a prepotent motor response, but also in pausing and braking responses (Aron et al., 2014; Forstmann et al., 2008; Levy & Wagner, 2011). Thus, this increased activation of the right IFC (also known as VLPFC) is thought to reflect not only engagement of motor inhibition, but also attentional orienting processes, updating of action plans, detection of infrequent stimuli (Levy & Wagner, 2011), increased working memory load, attentional monitoring for the stop signal, release of a brake, or application of a brake (Aron et al., 2014). In conjunction with these aforementioned studies, our results in the LH BA44 amplitude between 100 and 200 ms perhaps indicate children with an FASD diagnosis have decreased visual attention abilities to initiate and maintain the selective sensory bias in working memory that in turn reflect sensory information used for perceptual judgments (Hillyard & Anllo-Vento, 1998).

The peaks in the MEG time courses and their associated time windows identified in our BA44 ROI are highly consistent with peaks identified in frontal sensors in prior ERP studies, lending credence to our findings (Hillyard et al., 1995; Mangun & Buck, 1998). Dockree et al. (2005) identified ERP components during SART_F, two that translate to interpretation of our significant results. They highlighted selection negativity (SN) between 120 and 160 ms that aligns with our timing of 100 and 200 ms and cue recognition (CR) occurring ~300 ms that supports our findings between 200 and 350 ms. This SN activity indicates a repetitive deployment of selective attentional reserves during critical processing periods (Dockree et al., 2005). Unlike with SART_F in which critical processing periods are limited to trials 2, 3, and 9 (Dockree et al., 2005), the critical processing periods on the SART_R are present on every trial, as every stimulus has a chance to be the target numeral 3. This suggests children with an FASD diagnosis in our study have decreased selective attention capacity, highlighted by the increased difficulty in SART_R. Likewise, children with an FASD diagnosis exhibited decreased amplitude during cue recognition, discrimination, and implementation of subsequent goal-directed processes to integrate the stimuli with the goal. These dysfunctions in early sensory processing and later cue recognition likely contribute to behavioral differences in execution of the 'Go' response during SART_R. This also sheds light on the idiosyncrasies of neurophysiological activity in the SART_R, implicating bilateral activation in BA44 in action selection and braking.

Our findings that children with FASD have decreased neural activation in the ACC during Go trials perhaps indicate that these children not only have problems with inhibition itself, but also with the real-time interpretation of their action outcomes. This reduction in amplitude is consistent with the results of Gerhold et al. (2017), who reported reduced amplitude of the N2 and P2 peaks during a Go/No-go task in children with PAE. Interestingly, a late positive component also revealed increased amplitude in PAE relative to controls. While our study, with a shorter interstimulus interval (900–1100 ms) relative to the Gerhold study (4500 ms inter-trial interval), did not examine the late potential, the larger amplitude late potential may explain the larger amplitude frontal response seen in the PAE groups in most of the Go/No-go fMRI studies (Fryer et al., 2007; Kodali et al., 2017; Nash et al., 2017; O'Brien et al., 2013; Ware et al., 2015). Despite this amplitude difference between the MEG/EEG versus fMRI results, the fMRI studies revealed activations in similar regions as that examined here (ACC: O'Brien et al., 2013; DLPFC: Kodali et al., 2017). Previous studies have implicated the ACC as integral in target detection (Posner & Petersen, 1990), error detection and decision making (Botvinick, 2007), and conflict monitoring (Yeung et al., 2004). As every presentation of each numeral in SART_R has a chance to be a No-go stimulus, ACC activation is required for accurate execution of response and analysis of performance. Of course, ACC involvement extends beyond Go/No-go paradigms. It is possible our findings in the ACC, can elucidate the neurophysiological underpinnings of common behavioral dysfunctions seen in children with FASD. Inability to detect errors and accurately monitor conflicting stimuli due to non-typical ACC activation might be contributing to the well-documented, generalized executive functioning deficits (Kodituwakku, 2007), including attention (Coles et al., 2002) and inhibition (Mattson et al., 1999), found in children with FASD. Inconsistencies in the regions that reveal significant differences remain in the fMRI studies and may be attributable to differences in task design, PAE group characteristics, or sample size. The current study employed a region of interest analysis based on the literature review indicating regions most likely to be affected

due to PAE, however our cortical maps of MEG responses indicate the importance of employing a whole brain analysis in future studies.

4.5 | Correlations: MEG × neuropsychological assessments/parent/guardian questionnaires/SART behavior

We found significant relationships in MEG peak amplitude during SART_R localized to bilateral BA44 during all trials where a button press is required (RAG) and results from multiple neuropsychological measures, parent/guardian questionnaires, and task-based performance variables. Specifically, we found peak amplitude in left BA44 during time window 100–200 ms post-stimulus and in left and right BA44 200–350 ms was significantly associated with performance on WASI-II vocabulary and WASI-II full-scale IQ scores. The stronger the peak-amplitude, the better the vocabulary scores and the higher the IQ scores. These MEG measures were not associated with WASI-II Matrix Reasoning, GPB, or D-KEFS scores. Results support previously discussed literature implicating BA44 as a multifunctional brain region integral not only in speech production and processing components of language (Gelfand & Bookheimer, 2003), but also in multiple mechanisms of response inhibition (Aron et al., 2014; Forstmann et al., 2008; Koechlin & Jubault, 2006; Levy & Wagner, 2011). This association in neural activity and child performance was also present in the task-based measure, RAG hit rate. MEG peak amplitudes in all three time-windows of significance were correlated with the hit rate variable during all trials where a button press is required, showing that the larger the peak amplitude the better the hit rate performance. This supports a general view that children with higher fidelity cortical responses (greater signal to noise) will perform better than children with lower amplitude responses.

Peak amplitude in BA44 was associated with subjective caretaker ratings of child behavior. Specifically, caretakers rated children with greater peak amplitudes as having lower levels of ADHD-type behaviors and learning problems, assessed by the C3P, and lower levels of deficits in executive functioning, as assessed by the BRIEF. This highlights that not only is neural activity, as assessed by MEG, associated with IQ scores, fundamental cognitive abilities such as vocabulary, and task-based performance measures, but also in subjective ratings of observable behaviors as assessed by caregivers. The decreased amplitude measured with MEG, an electrophysiological technique, related to poor cognitive and behavioral performance may be caused by cell loss or decreases in synaptic density reported in preclinical studies of prenatal alcohol exposure (Berman & Hannigan, 2000). While group differences in behavioral and brain measures may indicate a trivial association between brain and behavioral measures and may not show direct causality, the specificity of the effects (associations were only seen in BA44 not ACC) suggest the association is not simply due to group differences in both variables.

4.6 | Exploratory analysis

In our exploratory analysis, we found group differences in total brain volume, left frontal volume, and right frontal volume, with FASD having significantly smaller volumes than HCs. This is congruent with multiple previous studies assessing structural differences in subjects with FASD versus HCs (Archibald et al., 2001; Johnson et al., 1996; Lebel et al., 2008, 2012; Li et al., 2008; Mattson et al., 1994, 1996; Willoughby et al., 2008). We also found that these variables significantly correlated with the MEG amplitude variables found to be different between groups. It is probable that these structural differences could be affecting the peak amplitude levels we found in our groups consistent with Candelaria-Cook, Solis, et al. (2022).

4.7 | Limitations

Some limitations to this study include non-matched sample sizes with smaller *N* in the HC group compared to the FASD group. Additionally, by limiting our analysis to BA44 and ACC in the MEG, we have potentially missed important group differences in other regions of the brain and a whole brain analysis in a larger sample is warranted. However, we chose to limit our analysis to our hypothesized regions based on the attention deficits experienced by children with an FASD diagnosis. An additional limitation is that we did not use the eyetracker employed during the prosaccade task (Pervin et al., 2021) to confirm compliance with fixation during the SART. The similarity in performance across groups minimizes the likelihood that differences in source amplitude were related to poor fixation compliance. It is also important to collect larger samples to allow for examination of sex effects and potential interactions between age, sex and prenatal exposures in future studies. Future studies will perform whole-brain analysis to allow for an extension of the current work.

5 | CONCLUSIONS

In conclusion, cognitive and behavioral deficits were found in children with an FASD diagnosis as assessed by their caretakers, neuropsychological evaluations, task-dependent performance in SART_R, and MEG activity during SART_R. The lack of significant group differences in SART_F performance and MEG activity supports prior studies indicating that children with an FASD diagnosis struggle with more complex tasks that require executive functions. Conversely, it demonstrates that the SART_R, an easy to administer task that children can understand and perform, is well suited to differentiate between these groups. These findings reveal new insights into the multi-domain deficiencies in functionality in children prenatally exposed to alcohol when compared to non-exposed children, and provide evidence that electrophysiological techniques, specifically MEG, can identify cortical changes directly associated with these cognitive and behavioral deficits. These reported cortical changes suggest a potential target metric for therapeutic interventions for children with an FASD diagnosis.

CONFLICT OF INTEREST STATEMENT

The authors declare no relevant conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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REFERENCES

Amiez, C., Joseph, J. P., & Procyk, E. (2005). Anterior cingulate error-related activity is modulated by predicted reward. *The European Journal of Neuroscience*, *21*, 3447–3452. https://doi.org/10.1111/j.1460-9568.2005.04170.x

Archibald, S. L., Fennema-Notestine, C., Gamst, A., Riley, E. P., Mattson, S. N., & Jernigan, T. L. (2001). Brain dysmorphology in individuals with severe prenatal alcohol exposure. *Developmental Medicine and Child Neurology*, *43*, 148–154.

Aron, A. R., Behrens, T. E., Smith, S., Frank, M. J., & Poldrack, R. A. (2007). Triangulating a cognitive control network using diffusion-weighted magnetic resonance imaging (MRI) and functional MRI. *The Journal of Neuroscience*, *27*, 3743–3752. https://doi.org/10.1523/JNEUROSCI.0519-07.2007

Aron, A. R., & Poldrack, R. A. (2006). Cortical and subcortical contributions to stop signal response inhibition: Role of the subthalamic nucleus. *The Journal of Neuroscience*, *26*, 2424–2433. https://doi.org/10.1523/JNEUROSCI.4682-05.2006

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2014). Inhibition and the right inferior frontal cortex: One decade on. *Trends in Cognitive Sciences*, *18*, 177–185. https://doi.org/10.1016/j.tics.2013.12.003

Barratt. (2006). The Barratt Simplified Measure of Social Status. Available online at http://wbarratt.indstate.edu/socialclass/Barratt_Simplifed_Measure_of_Social_Status.pdf

Benjamini, Y., & Hochberg, Y. (1995). Controlling the false discovery rate – A practical and powerful approach to multiple testing. *The Journal of the Royal Statistical Society, Series B (Statistical Methodology)*, *57*, 289–300.

Berman, R. F., & Hannigan, J. H. (2000). Effects of prenatal alcohol exposure on the hippocampus: Spatial behavior, electrophysiology, and neuroanatomy. *Hippocampus*, *10*, 94–110. https://doi.org/10.1002/(SICI)1098-1063(2000)10:1<94::AID-HIPO11>3.0.CO;2-T

Bertrand, J., Floyd, L. L., Weber, M. K., Fetal Alcohol Syndrome Prevention Team, DOBD Developmental Disabilities, NCOBD Developmental Disabilities, & CFDC Prevention. (2005). Guidelines for identifying and referring persons with fetal alcohol syndrome. *Morbidity and Mortality Weekly Report. Recommendations and Reports*, *54*, 1–14.

Bolanos, A. D., Coffman, B. A., Candelaria-Cook, F. T., Kodituwakku, P., & Stephen, J. M. (2017). Altered neural oscillations during multisensory integration in adolescents with fetal alcohol spectrum disorder. *Alcoholism, Clinical and Experimental Research*, *41*, 2173–2184. https://doi.org/10.1111/acer.13510

Botvinick, M. M. (2007). Conflict monitoring and decision making: Reconciling two perspectives on anterior cingulate function. *Cognitive, Affective, & Behavioral Neuroscience*, *7*, 356–366. https://doi.org/10.3758/cabn.7.4.356

Braver, T. S., Barch, D. M., Gray, J. R., Molfese, D. L., & Snyder, A. (2001). Anterior cingulate cortex and response conflict: Effects of frequency, inhibition and errors. *Cerebral Cortex*, *11*, 825–836. https://doi.org/10.1093/cercor/11.9.825

Brown, S., Martinez, M. J., & Parsons, L. M. (2006). Music and language side by side in the brain: A PET study of the generation of melodies and sentences. *The European Journal of Neuroscience*, *23*, 2791–2803. https://doi.org/10.1111/j.1460-9568.2006.04785.x

Cai, W., & Leung, H. C. (2011). Rule-guided executive control of response inhibition: Functional topography of the inferior frontal cortex. *PLoS One*, *6*, e20840. https://doi.org/10.1371/journal.pone.0020840

Candelaria-Cook, F. T., Schendel, M. E., Flynn, L., Cerros, C., Kodituwakku, P., Bakhireva, L. N., Hill, D. E., & Stephen, J. M. (2022). Decreased resting-state alpha peak frequency in children and adolescents with fetal alcohol spectrum disorders or prenatal alcohol exposure. *Developmental Cognitive Neuroscience*, *57*, 101137. https://doi.org/10.1016/j.dcn.2022.101137

Candelaria-Cook, F. T., Solis, I., Schendel, M. E., Wang, Y. P., Wilson, T. W., Calhoun, V. D., & Stephen, J. M. (2022). Developmental trajectory of MEG resting-state oscillatory activity in children and adolescents: A longitudinal reliability study. *Cerebral Cortex*, *32*, 5404–5419. https://doi.org/10.1093/cercor/bhac023

Carr, J. L., Agnihotri, S., & Keightley, M. (2010). Sensory processing and adaptive behavior deficits of children across the fetal alcohol spectrum disorder continuum. *Alcoholism, Clinical and Experimental Research*, *34*, 1022–1032. https://doi.org/10.1111/j.1530-0277.2010.01177.x

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747–749. https://doi.org/10.1126/science.280.5364.747

Centers for Disease Control and Prevention (CDC) (2002). Fetal alcohol syndrome–Alaska, Arizona, Colorado, and New York, 1995-1997. *MMWR Morb Mortal Wkly Rep*, *51*(20), 433–435.

Chernick, V., Childiaeva, R., & Ioffe, S. (1983). Effects of maternal alcohol intake and smoking on neonatal electroencephalogram and anthropometric measurements. *American Journal of Obstetrics and Gynecology*, *146*, 41–47.

Church, M. W. (1987). Chronic in utero alcohol exposure affects auditory function in rats and in humans. *Alcohol*, *4*, 231–239.

Church, M. W., & Gerkin, K. P. (1988). Hearing disorders in children with fetal alcohol syndrome: Findings from case reports. *Pediatrics*, *82*, 147–154.

Church, M. W., Hotra, J. W., Holmes, P. A., Anumba, J. I., Jackson, D. A., & Adams, B. R. (2012). Auditory brainstem response (ABR) abnormalities across the life span of rats prenatally exposed to alcohol. *Alcoholism, Clinical and Experimental Research*, *36*, 83–96. https://doi.org/10.1111/j.1530-0277.2011.01594.x

Coffman, B. A., Kodituwakku, P., Kodituwakku, E. L., Romero, L., Sharadamma, N. M., Stone, D., & Stephen, J. M. (2013). Primary visual response (M100) delays in adolescents with FASD as measured with MEG. *Human Brain Mapping*, *34*, 2852–2862. https://doi.org/10.1002/hbm.22110

Coles, C. D., Platzman, K. A., Lynch, M. E., & Freides, D. (2002). Auditory and visual sustained attention in adolescents prenatally exposed to alcohol. *Alcoholism, Clinical and Experimental Research*, *26*, 263–271.

Company, L. I. (2002). *Grooved Pegboard Test: User instructions model 32025*. Lafayette, Inc.

Conners, C. K. (1997). *Conners' rating scales - Revised*. Multi-Health Systems Inc.

Curley, L. B., Newman, E., Thompson, W. K., Brown, T. T., Hagler, D. J., Jr., Akshoomoff, N., Reuter, C., Dale, A. M., & Jernigan, T. L. (2018). Cortical morphology of the pars opercularis and its relationship to motor-inhibitory performance in a longitudinal, developing cohort. *Brain Structure & Function*, *223*, 211–220. https://doi.org/10.1007/s00429-017-1480-5

D'angiulli, A., Grunau, P., Maggi, S., & Herdman, A. (2006). Electroencephalographic correlates of prenatal exposure to alcohol in infants and children: A review of findings and implications for neurocognitive development. *Alcohol*, *40*, 127–133. https://doi.org/10.1016/j.alcohol.2006.09.031

Dale, A., Liu, A., Fischl, B., Buckner, R., Belliveau, J., Lewine, J., & Halgren, E. (2000). Dynamic statistical parametric mapping: Combining

fMRI and MEG for high-resolution imaging of cortical activity. *Neuron*, *26*, 55–67.

De Zubicaray, G. I., Zelaya, F. O., Andrew, C., Williams, S. C., & Bullmore, E. T. (2000). Cerebral regions associated with verbal response initiation, suppression and strategy use. *Neuropsychologia*, *38*, 1292–1304. https://doi.org/10.1016/s0028-3932(00)00026-9

Delis, D., Kaplan, E., & Kramer, J. (2001). *Delis-Kaplan executive function system*. Pearson Education, Inc.

Dockree, P. M., Kelly, S. P., Robertson, I. H., Reilly, R. B., & Foxe, J. J. (2005). Neurophysiological markers of alert responding during goal-directed behavior: A high-density electrical mapping study. *NeuroImage*, *27*, 587–601. https://doi.org/10.1016/j.neuroimage.2005.05.044

Doney, R., Lucas, B. R., Jones, T., Howat, P., Sauer, K., & Elliott, E. J. (2014). Fine motor skills in children with prenatal alcohol exposure or fetal alcohol spectrum disorder. *Journal of Developmental and Behavioral Pediatrics*, *35*, 598–609. https://doi.org/10.1097/DBP.0000000000000107

Durston, S., Thomas, K. M., Yang, Y. H., Ulug, A. M., Zimmerman, R. D., & Casey, B. J. (2002). A neural basis for the development of inhibitory control. *Developmental Science*, *5*, F9–F16. https://doi.org/10.1111/1467-7687.00235

Falkenstein, M., Hoormann, J., Christ, S., & Hohnsbein, J. (2000). ERP components on reaction errors and their functional significance: A tutorial. *Biological Psychology*, *51*(2-3), 87–107.

Forstmann, B. U., Van Den Wildenberg, W. P., & Ridderinkhof, K. R. (2008). Neural mechanisms, temporal dynamics, and individual differences in interference control. *Journal of Cognitive Neuroscience*, *20*, 1854–1865. https://doi.org/10.1162/jocn.2008.20122

Fryer, S. L., Tapert, S. F., Mattson, S. N., Paulus, M. P., Spadoni, A. D., & Riley, E. P. (2007). Prenatal alcohol exposure affects frontal-striatal BOLD response during inhibitory control. *Alcoholism, Clinical and Experimental Research*, *31*, 1415–1424. https://doi.org/10.1111/j.1530-0277.2007.00443.x

Garratt, L. C., & Kelly, T. P. (2008). To what extent does bilingualism affect children's performance on the NEPSY? *Child Neuropsychology*, *14*, 71–81. https://doi.org/10.1080/09297040701218405

Gelfand, J. R., & Bookheimer, S. Y. (2003). Dissociating neural mechanisms of temporal sequencing and processing phonemes. *Neuron*, *38*, 831–842. https://doi.org/10.1016/s0896-6273(03)00285-x

Gerhold, M. M., Jacobson, S. W., Jacobson, J. L., Molteno, C. D., Meintjes, E. M., & Andrew, C. M. (2017). An ERP study of response inhibition in the auditory domain in children with fetal alcohol spectrum disorders. *Alcoholism, Clinical and Experimental Research*, *41*, 96–106. https://doi.org/10.1111/acer.13263

Glass, L., Moore, E. M., Akshoomoff, N., Jones, K. L., Riley, E. P., & Mattson, S. N. (2017). Academic difficulties in children with prenatal alcohol exposure: Presence, profile, and neural correlates. *Alcoholism, Clinical and Experimental Research*, *41*, 1024–1034. https://doi.org/10.1111/acer.13366

Green, C. R., Mihic, A. M., Brien, D. C., Armstrong, I. T., Nikkel, S. M., Stade, B. C., Rasmussen, C., Munoz, D. P., & Reynolds, J. N. (2009). Oculomotor control in children with fetal alcohol spectrum disorders assessed using a mobile eye-tracking laboratory. *The European Journal of Neuroscience*, *29*, 1302–1309. https://doi.org/10.1111/j.1460-9568.2009.06668.x

Hamalainen, M., Hari, R., Ilmoniemi, R. J., Knuutila, J., & Lounasmaa, O. V. (1993). Magnetoencephalography – Theory, instrumentation, and applications to noninvasive studies of the working human brain. *Reviews of Modern Physics*, *65*, 413–497.

Hamalainen, M. S., & Lundqvist, D. (2019). MEG as an enabling tool in neuroscience: Transcending boundaries with new analysis methods and devices. In S. Supek & C. Aine (Eds.), *Magnetoencephalography*. Springer.

Hampshire, A., Chamberlain, S. R., Monti, M. M., Duncan, J., & Owen, A. M. (2010). The role of the right inferior frontal gyrus: Inhibition and attentional control. *NeuroImage*, *50*, 1313–1319. https://doi.org/10.1016/j.neuroimage.2009.12.109

Hari, R., & Puce, A. (2017). *MEG-EEG primer*. Oxford University Press.

Havlicek, V., Childiaeva, R., & Chernick, V. (1977). EEG frequency spectrum characteristics of sleep states in infants of alcoholic mothers. *Neuropädiatrie*, *8*, 360–373. https://doi.org/10.1055/s-0028-1091532

Hillyard, S. A., & Anllo-Vento, L. (1998). Event-related brain potentials in the study of visual selective attention. *Proceedings of the National Academy of Sciences of the United States of America*, *95*, 781–787. https://doi.org/10.1073/pnas.95.3.781

Hillyard, S. A., Mangun, G. R., Woldorff, M., & Luck, S. J. (1995). Neural systems mediating selective attention. In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 665–681). MIT Press.

Hoyme, H. E., Kalberg, W. O., Elliott, A. J., Blankenship, J., Buckley, D., Marais, A. S., Manning, M. A., Robinson, L. K., Adam, M. P., Abdul-Rahman, O., Jewett, T., Coles, C. D., Chambers, C., Jones, K. L., Adnams, C. M., Shah, P. E., Riley, E. P., Charness, M. E., Warren, K. R., & May, P. A. (2016). Updated clinical guidelines for diagnosing fetal alcohol spectrum disorders. *Pediatrics*, *138*, e20154256. https://doi.org/10.1542/peds.2015-4256

Hoyme, H. E., May, P. A., Kalberg, W. O., Kodituwakku, P., Gossage, J. P., Trujillo, P. M., Buckley, D. G., Miller, J. H., Aragon, A. S., Khaole, N., Viljoen, D. L., Jones, K. L., & Robinson, L. K. (2005). A practical clinical approach to diagnosis of fetal alcohol spectrum disorders: Clarification of the 1996 institute of medicine criteria. *Pediatrics*, *115*, 39–47. https://doi.org/10.1542/peds.2004-0259

Ioffe, S., Childiaeva, R., & Chernick, V. (1984). Prolonged effects of maternal alcohol ingestion on the neonatal electroencephalogram. *Pediatrics*, *74*, 330–335.

Jahfari, S., Stinear, C. M., Claffey, M., Verbruggen, F., & Aron, A. R. (2010). Responding with restraint: What are the neurocognitive mechanisms? *Journal of Cognitive Neuroscience*, *22*, 1479–1492. https://doi.org/10.1162/jocn.2009.21307

Jirikowic, T. L., Thorne, J. C., Mclaughlin, S. A., Waddington, T., Lee, A. K. C., & Astley Hemingway, S. J. (2020). Prevalence and patterns of sensory processing behaviors in a large clinical sample of children with prenatal alcohol exposure. *Research in Developmental Disabilities*, *100*, 103617. https://doi.org/10.1016/j.ridd.2020.103617

Johnson, V. P., Swayze, V. W., Sato, Y., & Andreasen, N. C. (1996). Fetal alcohol syndrome: Craniofacial and central nervous system manifestations. *American Journal of Medical Genetics*, *61*, 329–339. https://doi.org/10.1002/(SICI)1096-8628(19960202)61:4<329::AID-AJMG6>3.0.CO;2-P

Kalberg, W. O., & Buckley, D. (2007). FASD: What types of intervention and rehabilitation are useful? *Neuroscience and Biobehavioral Reviews*, *31*, 278–285. https://doi.org/10.1016/j.neubiorev.2006.06.014

Kodali, V. N., Jacobson, J. L., Lindinger, N. M., Dodge, N. C., Molteno, C. D., Meintjes, E. M., & Jacobson, S. W. (2017). Differential recruitment of brain regions during response inhibition in children prenatally exposed to alcohol. *Alcoholism, Clinical and Experimental Research*, *41*, 334–344. https://doi.org/10.1111/acer.13307

Kodituwakku, P., & Kodituwakku, E. L. (2014). Cognitive and behavioral profiles of children with fetal alcohol spectrum disorders. *Current Developmental Disorders Reports*, *1*, 149–160.

Kodituwakku, P. W. (2007). Defining the behavioral phenotype in children with fetal alcohol spectrum disorders: A review. *Neuroscience and Biobehavioral Reviews*, *31*, 192–201. https://doi.org/10.1016/j.neubiorev.2006.06.020

Kodituwakku, P. W., Handmaker, N. S., Cutler, S. K., Weathersby, E. K., & Handmaker, S. D. (1995). Specific impairments in self-regulation in children exposed to alcohol prenatally. *Alcoholism, Clinical and Experimental Research*, *19*, 1558–1564.

Koechlin, E., & Jubault, T. (2006). Broca's area and the hierarchical organization of human behavior. *Neuron*, *50*, 963–974. https://doi.org/10.1016/j.neuron.2006.05.017

Koren, G., Zelner, I., Nash, K., & Koren, G. (2014). Foetal alcohol spectrum disorder: Identifying the neurobehavioural phenotype and effective interventions. *Current Opinion in Psychiatry*, *27*, 98–104. https://doi.org/10.1097/YCO.0000000000000038

Lebel, C., Mattson, S. N., Riley, E. P., Jones, K. L., Adnams, C. M., May, P. A., Bookheimer, S. Y., O'Connor, M. J., Narr, K. L., Kan, E., Abaryan, Z., & Sowell, E. R. (2012). A longitudinal study of the long-term consequences of drinking during pregnancy: Heavy in utero alcohol exposure disrupts the normal processes of brain development. *The Journal of Neuroscience*, *32*, 15243-51. https://doi.org/10.1523/JNEUROSCI.1161-12.2012

Lebel, C., Rasmussen, C., Wyper, K., Walker, L., Andrew, G., Yager, J., & Beaulieu, C. (2008). Brain diffusion abnormalities in children with fetal alcohol spectrum disorder. *Alcoholism, Clinical and Experimental Research*, *32*, 1732–1740. https://doi.org/10.1111/j.1530-0277.2008.00750.x

Levy, B. J., & Wagner, A. D. (2011). Cognitive control and right ventrolateral prefrontal cortex: Reflexive reorienting, motor inhibition, and action updating. *Annals of the new York Academy of Sciences*, *1224*, 40–62. https://doi.org/10.1111/j.1749-6632.2011.05958.x

Li, Z., Ma, X., Peltier, S., Hu, X., Coles, C. D., & Lynch, M. E. (2008). Occipital-temporal reduction and sustained visual attention deficit in prenatal alcohol exposed adults. *Brain Imaging and Behavior*, *2*, 39–48. https://doi.org/10.1007/s11682-007-9013-0

Mangun, G. R., & Buck, L. A. (1998). Sustained visual-spatial attention produces costs and benefits in response time and evoked neural activity. *Neuropsychologia*, *36*, 189–200. https://doi.org/10.1016/s0028-3932(97)00123-1

Margret, C. P., Chappell, T. D., Li, C. X., Jan, T. A., Matta, S. G., Elberger, A. J., & Waters, R. S. (2006). Prenatal alcohol exposure (PAE) reduces the size of the forepaw representation in forepaw barrel subfield (FBS) cortex in neonatal rats: Relationship between periphery and central representation. *Experimental Brain Research*, *172*, 387–396. https://doi.org/10.1007/s00221-005-0339-9

Mattson, S. N., Bernes, G. A., & Doyle, L. R. (2019). Fetal alcohol spectrum disorders: A review of the neurobehavioral deficits associated with prenatal alcohol exposure. *Alcoholism, Clinical and Experimental Research*, *43*, 1046–1062. https://doi.org/10.1111/acer.14040

Mattson, S. N., Goodman, A. M., Caine, C., Delis, D. C., & Riley, E. P. (1999). Executive functioning in children with heavy prenatal alcohol exposure. *Alcoholism, Clinical and Experimental Research*, *23*, 1808–1815.

Mattson, S. N., Jernigan, T. L., & Riley, E. P. (1994). MRI and prenatal alcohol exposure: Images provide insight into FAS. *Alcohol Health and Research World*, *18*, 49–52.

Mattson, S. N., Riley, E. P., Sowell, E. R., Jernigan, T. L., Sobel, D. F., & Jones, K. L. (1996). A decrease in the size of the basal ganglia in children with fetal alcohol syndrome. *Alcoholism, Clinical and Experimental Research*, *20*, 1088–1093.

Maxwell, S., & Delaney, H. (2004). *Designing experiments and analyzing data: A model comparison perspective*. Psychology Press.

May, P. A., Baete, A., Russo, J., Elliott, A. J., Blankenship, J., Kalberg, W. O., Buckley, D., Brooks, M., Hasken, J., Abdul-Rahman, O., Adam, M. P., Robinson, L. K., Manning, M., & Hoyme, H. E. (2014). Prevalence and characteristics of fetal alcohol spectrum disorders. *Pediatrics*, *134*, 855–866. https://doi.org/10.1542/peds.2013-3319

May, P. A., Chambers, C. D., Kalberg, W. O., Zellner, J., Feldman, H., Buckley, D., Kopald, D., Hasken, J. M., Xu, R., Honerkamp-Smith, G., Taras, H., Manning, M. A., Robinson, L. K., Adam, M. P., Abdul-Rahman, O., Vaux, K., Jewett, T., Elliott, A. J., Kable, J. A., … Hoyme, H. E. (2018). Prevalence of fetal alcohol spectrum disorders in 4 US communities. *The Journal of the American Medical Association*, *319*, 474–482. https://doi.org/10.1001/jama.2017.21896

May, P. A., & Gossage, J. P. (2001). Estimating the prevalence of fetal alcohol syndrome. A summary. *Alcohol Research & Health*, *25*, 159–167.

May, P. A., Gossage, J. P., Kalberg, W. O., Robinson, L. K., Buckley, D., Manning, M., & Hoyme, H. E. (2009). Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Developmental Disabilities Research Reviews*, *15*, 176–192. https://doi.org/10.1002/ddrr.68

Mazzone, M. (2014). A generative system for intentional action? *Topoi An International Review of Philosophy*, *33*, 77–85. https://doi.org/10.1007/s11245-013-9186-7

Nash, K., Stevens, S., Clairman, H., & Rovet, J. (2017). Preliminary findings that a targeted intervention leads to altered brain function in children with fetal alcohol spectrum disorder. *Brain Sciences*, *8*, 7. https://doi.org/10.3390/brainsci8010007

O'Brien, J. W., Norman, A. L., Fryer, S. L., Tapert, S. F., Paulus, M. P., Jones, K. L., Riley, E. P., & Mattson, S. N. (2013). Effect of predictive cuing on response inhibition in children with heavy prenatal alcohol exposure. *Alcoholism, Clinical and Experimental Research*, *37*, 644–654. https://doi.org/10.1111/acer.12017

O'Malley, K. D., & Nanson, J. (2002). Clinical implications of a link between fetal alcohol spectrum disorder and attention-deficit hyperactivity disorder. *Canadian Journal of Psychiatry*, *47*, 349–354. https://doi.org/10.1177/070674370204700405

Olson, H. C., Morse, B. A., & Huffine, C. (1998). Development and psychopathology: Fetal alcohol syndrome and related conditions. *Seminars in Clinical Neuropsychiatry*, *3*, 262–284.

Paley, B., & O'Connor, M. J. (2009). Intervention for individuals with fetal alcohol spectrum disorders: Treatment approaches and case management. *Developmental Disabilities Research Reviews*, *15*, 258–267. https://doi.org/10.1002/ddrr.67

Paolozza, A., Rasmusse, C., Pei, J., Hanlon-Dearman, A., Nikkel, S. M., Andrew, G., Mcfarlane, A., Samdup, D., & Reynolds, J. N. (2014). Deficits in response inhibition correlate with oculomotor control in children with fetal alcohol spectrum disorder and prenatal alcohol exposure. *Behavioural Brain Research*, *259*, 97–105. https://doi.org/10.1016/j.bbr.2013.10.040

Penny, A. M., Waschbusch, D. A., Klein, R. M., Corkum, P., & Eskes, G. (2009). Developing a measure of sluggish cognitive tempo for children: Content validity, factor structure, and reliability. *Psychological Assessment*, *21*, 380–389. https://doi.org/10.1037/a0016600

Perkins, G. (2016). *Socioeconomic status: Influences, disparities and current issues*. Nova Publishers.

Pervin, Z., Pinner, J. F. L., Flynn, L., Cerros, C. M., Williams, M. E., Hill, D. E., & Stephen, J. M. (2021). School-aged children diagnosed with an FASD exhibit visuo-cortical network disturbance: A magnetoencephalography (MEG) study. *Alcohol*, *99*, 59–69. https://doi.org/10.1016/j.alcohol.2021.12.001

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience*, *13*, 25–42. https://doi.org/10.1146/annurev.ne.13.030190.000325

Puce, A., & Hamalainen, M. S. (2017). A review of issues related to data acquisition and analysis in EEG/MEG studies. *Brain Sciences*, *7*, 58. https://doi.org/10.3390/brainsci7060058

Rae, C. L., Hughes, L. E., Weaver, C., Anderson, M. C., & Rowe, J. B. (2014). Selection and stopping in voluntary action: A meta-analysis and combined fMRI study. *NeuroImage*, *86*, 381–391. https://doi.org/10.1016/j.neuroimage.2013.10.012

Riley, E. P., & McGee, C. L. (2005). Fetal alcohol spectrum disorders: An overview with emphasis on changes in brain and behavior. *Experimental Biology and Medicine*, *230*, 357–365. https://doi.org/10.1177/15353702-0323006-03

Rizzolatti, G., Fogassi, L., & Gallese, V. (2002). Motor and cognitive functions of the ventral premotor cortex. *Current Opinion in Neurobiology*, *12*, 149–154. https://doi.org/10.1016/s0959-4388(02)00308-2

Robertson, I. H., Manly, T., Andrade, J., Baddeley, B. T., & Yiend, J. (1997). 'Oops!': Performance correlates of everyday attentional failures in traumatic brain injured and normal subjects. *Neuropsychologia*, *35*, 747–758.

Roozen, S., Peters, G. J., Kok, G., Townend, D., Nijhuis, J., & Curfs, L. (2016). Worldwide prevalence of fetal alcohol spectrum disorders: A systematic literature review including meta-analysis. *Alcoholism, Clinical and Experimental Research*, *40*, 18–32. https://doi.org/10.1111/acer.12939

Rosen, A. F. G., Roalf, D. R., Ruparel, K., Blake, J., Seelaus, K., Villa, L. P., Ciric, R., Cook, P. A., Davatzikos, C., Elliott, M. A., Garcia De La Garza, A., Gennatas, E. D., Quarmley, M., Schmitt, J. E., Shinohara, R. T., Tisdall, M. D., Craddock, R. C., Gur, R. E., Gur, R. C., & Satterthwaite, T. D. (2018). Quantitative assessment of structural image quality. *NeuroImage*, *169*, 407–418. https://doi.org/10.1016/j.neuroimage.2017.12.059

Sampson, P. D., Streissguth, A. P., Bookstein, F. L., Little, R. E., Clarren, S. K., Dehaene, P., Hanson, J. W., & Graham, J. M., Jr. (1997). Incidence of fetal alcohol syndrome and prevalence of alcohol-related neurodevelopmental disorder. *Teratology*, *56*, 317–326. https://doi.org/10.1002/(SICI)1096-9926(199711)56:5<317::AID-TERA5>3.0.CO;2-U

Segonne, F., Dale, A. M., Busa, E., Glessner, M., Salat, D., Hahn, H. K., & Fischl, B. (2004). A hybrid approach to the skull stripping problem in MRI. *NeuroImage*, *22*, 1060–1075. https://doi.org/10.1016/j.neuroimage.2004.03.032

Sirin, S. R. (2005). Socioeconomic status and academic achievement: A meta-analytic review of research. *Review of Educational Research*, *75*, 417–453. https://doi.org/10.3102/00346543075003417

Sowell, E. R., Thompson, P. M., Mattson, S. N., Tessner, K. D., Jernigan, T. L., Riley, E. P., & Toga, A. W. (2001). Voxel-based morphometric analyses of the brain in children and adolescents prenatally exposed to alcohol. *Neuroreport*, *12*, 515–523.

Sowell, E. R., Thompson, P. M., Mattson, S. N., Tessner, K. D., Jernigan, T. L., Riley, E. P., & Toga, A. W. (2002). Regional brain shape abnormalities persist into adolescence after heavy prenatal alcohol exposure. *Cerebral Cortex*, *12*, 856–865. https://doi.org/10.1093/cercor/12.8.856

Stephen, J., Kodituwakku, P., Kodituwakku, E. L., Romero, L., Peters, A. M., Sharadamma, N. M., Caprihan, A., & Coffman, B. A. (2012). Delays in auditory processing identified in preschool children with FASD. *Alcoholism: Clinical and Experimental Research*, *36*, 1720–1727.

Stephen, J. M., Aine, C. J., Christner, R. F., Ranken, D., Huang, M., & Best, E. (2002). Central versus peripheral visual field stimulation results in timing differences in dorsal stream sources as measured with MEG. *Vision Research*, *42*, 3059–3074.

Stratton, K., Howe, C., & Battaglia, F. P. (Eds.). (1996). *Institute of medicine. Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment*. National Academy Press.

Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental and Behavioral Pediatrics*, *25*, 228–238. https://doi.org/10.1097/00004703-200408000-00002

Taulu, S., & Kajola, M. (2005). Presentation of electromagnetic multichannel data: The signal space separation method. *Journal of Applied Physics*, *97*, 124905. https://doi.org/10.1063/1.1935742

Uusitalo, M. A., & Ilmoniemi, R. J. (1997). Signal-space projection method for separating MEG or EEG into components. *Medical & Biological Engineering & Computing.*, *35*, 135–140.

Verbruggen, F., & Logan, G. D. (2009). Models of response inhibition in the stop-signal and stop-change paradigms. *Neuroscience and Biobehavioral Reviews*, *33*, 647–661. https://doi.org/10.1016/j.neubiorev.2008.08.014

Wang, R., Martin, C. D., Lei, A. L., Hausknecht, K. A., Turk, M., Micov, V., Kwarteng, F., Ishiwari, K., Oubraim, S., Wang, A. L., Richards, J. B., Haj-Dahmane, S., & Shen, R. Y. (2022). Prenatal ethanol exposure impairs sensory processing and habituation to visual stimuli, effects normalized by enrichment of postnatal environmental. *Alcoholism, Clinical and Experimental Research*, *46*, 891–906. https://doi.org/10.1111/acer.14818

Ware, A. L., Infante, M. A., O'Brien, J. W., Tapert, S. F., Jones, K. L., Riley, E. P., & Mattson, S. N. (2015). An fMRI study of behavioral response inhibition in adolescents with and without histories of heavy prenatal alcohol exposure. *Behavioural Brain Research*, *278*, 137–146. https://doi.org/10.1016/j.bbr.2014.09.037

Wechsler, D. (2011). *Wechsler abbreviated scale of intelligence*. Pearson.

Willoughby, K. A., Sheard, E. D., Nash, K., & Rovet, J. (2008). Effects of prenatal alcohol exposure on hippocampal volume, verbal learning, and verbal and spatial recall in late childhood. *Journal of the International Neuropsychological Society*, *14*, 1022–1033. https://doi.org/10.1017/S1355617708081368

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*, 931–959. https://doi.org/10.1037/0033-295X.111.4.939

Zordan, L., Sarlo, M., & Stablum, F. (2008). ERP components activated by the "GO!" and "WITHHOLD!" conflict in the random sustained attention to response task. *Brain and Cognition*, *66*, 57–64. https://doi.org/10.1016/j.bandc.2007.05.005

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