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RESEARCH ARTICLE

Increased alpha power in autistic adults: Relation to sensory behaviors and cortical volume

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Abstract
Alpha-band (~10 Hz) neural oscillations, crucial for gating sensory information, may offer insights into the atypical sensory experiences characteristic of autism spectrum disorder (ASD). We investigated alpha-band EEG activity in autistic adults ($n = 29$) compared with a nonautistic group ($n = 23$) under various stimulus-driven and resting-state conditions. The autistic group showed consistently higher alpha amplitude across all time points. In addition, there was proportionally more suppression of alpha at stimulus onset in the autistic group, and alpha amplitude in this stimulus-onset period correlated with sensory behaviors. Recent research suggests a link between subcortical structures' volume and cortical alpha magnitude. Prompted by this, we explored the association between alpha power and the volume of subcortical structures and total cortical volume in ASD. Our findings indicate a significant correlation with total cortical volume and a group by hippocampal volume interaction, pointing to the potential role of anatomical structural characteristics as potential modulators of cortical alpha oscillations in ASD. Overall, the results highlight altered alpha in autistic individuals as potentially contributing to the heightened sensory symptoms in autistic compared with nonautistic adults.

Lay Summary
Our study explores how individuals with autism process sensory information differently, focusing on alpha-band oscillations—brain waves thought to help filter and prioritize sensory inputs. We found that people with ASD have heightened levels of these oscillations, suggesting their brains may filter sensory information in a unique way and contribute to increased sensitivity or avoidance of sensory stimuli. Additionally, by linking this brain activity to the structure of specific brain areas like the hippocampus, our research suggests a structural foundation for these distinctive sensory processing patterns in ASD.

KEYWORDS
alpha, autism spectrum disorder, EEG, sensory processing, vision

INTRODUCTION

Autism spectrum disorder (ASD) is a neurodevelopmental condition marked by a broad spectrum of symptoms, including challenges in social communication and repetitive behaviors. In addition, altered sensory processing in ASD, manifesting as hypersensitivity or hyposensitivity to sensory stimuli, is a clinically significant aspect that can significantly affect quality of life (Costa-López et al., 2021; Leader et al., 2021). Identifying the neurophysiological bases for these sensory processing differences has been challenging. One potential basis are alpha-band (8–12 Hz) oscillations as they are known to shape sensory experiences (Foxe & Snyder, 2011; Jensen & Mazaheri, 2010; Klimesch et al., 2007; Liu et al., 2016; K. Mathewson et al., 2011; Sherman et al., 2016).

Alpha oscillations are the most prominent and easily detectable brain rhythms in the human EEG, particularly pronounced when individuals close their eyes. These oscillations, typically in the 8–12 Hz range, are most evident over posterior brain regions, where they are thought to reflect the interaction between cortical and subcortical networks. Among these, the thalamus is known to have a significant role, as evidenced by studies showing that thalamic lesions can significantly diminish alpha oscillations (Ohmoto et al., 1978). Moreover, electrophysiological recordings have demonstrated strong coherence between alpha oscillations in the visual cortex and thalamus, underscoring the thalamus's role as a central hub in alpha rhythm generation (Lőrincz et al., 2009). In addition to subcortical contributions, cortical activity, especially in the deeper layers of the visual cortex, is integral to the origin and modulation of alpha oscillations (Bollimunta et al., 2008, 2011).

Research on the functional role of alpha oscillations in perception and cognition in nonautistic populations has revealed a variety of potential functions. Most notably, alpha oscillations are widely recognized as an active inhibitory mechanism, selectively suppressing irrelevant sensory information to optimize cognitive processing (Clayton et al., 2015; Jensen & Mazaheri, 2010). For example, increases in alpha amplitude are typically linked to worse performance in tasks requiring visual processing, reflecting a withdrawal of attention from visual stimuli (Ergenoglu et al., 2004; K. Mathewson et al., 2011; van Dijk et al., 2008). Conversely, when visual attention is increased, alpha power in the corresponding cortical regions decreases, facilitating enhanced processing of relevant stimuli (Rihs et al., 2007; Thut et al., 2006; Yuasa et al., 2023). This modulation underscores alpha oscillations' role in balancing sensory input and cognitive demands (Fries et al., 2008).

Beyond their inhibitory role, alpha oscillations also appear to directly influence perceptual processes. For example, certain visual illusions that seem to flicker do so within the alpha frequency range, with the intensity of the illusion correlating with the power of alpha oscillations in the EEG (Sokoliuk & VanRullen, 2013). This relationship suggests that alpha rhythms may act as a temporal sampling mechanism. Supporting this idea, several studies have demonstrated that visual stimuli are more effectively processed when they coincide with the trough, rather than the peak, of the alpha cycle, highlighting alpha's role as a temporal sampling mechanism. Additionally, alpha oscillations are linked to stability in visual perception. Research on multistable perception, such as the Necker cube illusion, shows that individuals are more likely to experience perceptual shifts during periods of reduced alpha power (Isoglu-Alkaç et al., 2000; Mathes et al., 2010). These reductions are believed to destabilize ongoing perceptual interpretations, thereby facilitating transitions to alternative interpretations (Piantoni et al., 2017; Strüber & Herrmann, 2002).

Notably the dimensions over which processing differences have been identified in autism align with many of the known functions of alpha oscillations. First, an often cited (though controversial) hypothesis is that autism is characterized by a generally disruption to neural inhibition (Antoine et al., 2019; Christ et al., 2011; Dickinson et al., 2016; Rubenstein & Merzenich, 2003). Second, there is evidence that autism is associated with disrupted attentional filtering of sensory information (Dawson et al., 2004; Keehn et al., 2013; Loveland & Landry, 1986; Maestro et al., 2002; Toth et al., 2006), though some recent findings suggest that attention may be intact (Fischer et al., 2014, 2016; Grubb et al., 2013a, 2013b). Third, compelling evidence demonstrates altered perceptual switch rates during multistable perception tasks, such as binocular rivalry, in autistic individuals (Robertson et al., 2016; Spiegel et al., 2019). Taken together, the behavioral and perceptual alterations observed in autism align well with the known functions of alpha oscillations, suggesting the simple hypothesis that alpha signaling may be disrupted or attenuated in autism.

However, to date, research on alpha oscillations in autism has yielded a complex and sometimes conflicting set of findings. Both an extensive review (Wang et al., 2013) and recent meta-analysis (Neo et al., 2023) suggests that resting-state alpha magnitude may be reduced in autistic individuals; however, the variability across studies is considerable with a few showing increased alpha amplitude (Cornew et al., 2012; K. J. Mathewson et al., 2012; Sutton et al., 2005). This inconsistency may be partly due to the fact that alpha oscillations are highly modulated by various stimulus and cognitive factors, complicating the interpretation of results.

To address these challenges, we designed an experiment that systematically examined multiple stimulus-based factors known to influence alpha oscillations. We measured several features of alpha, including baseline alpha during eyes-open rest, alpha response at the onset of visual stimulation, during sustained visual stimulation, at the offset of stimulation, and the return to baseline. Mullinger et al. (2017) examined poststimulus alpha dynamics in nonautistic individuals, particularly the postevent rebound of alpha (PERS). Their findings, combined with our previous work showing a reduction in the fMRI BOLD undershoot in autism (Murray et al., 2020), led us to hypothesize that the PERS may also be altered in autism. Drawing from this framework, we adopted a similar experimental approach, manipulating stimulus contrast to explore alpha dynamics during both the primary response and the poststimulus phase in autism. Finally, we took into account potential confounding factors that potentially impact alpha amplitude, such as sleep quality (Tran et al., 2020) and medication use (Aiyer et al., 2016; Clarke et al., 2002).

Recent research by Ghafari et al. (2024), which focused on non-autistic adults, has provided compelling

evidence that the structure of subcortical regions, such as the thalamus and basal ganglia, plays a critical role in modulating cortical alpha oscillations, particularly in the context of spatial attention. Their study demonstrated that hemispheric asymmetries in the volumes of the thalamus, caudate nucleus, and globus pallidus were predictive of the modulation of alpha-band power during attention tasks in neurotypical individuals. In contrast, Edgar et al. (2015) investigated the relationship between thalamic volume and resting-state alpha activity in children with ASD and found that these associations were absent in the autistic group, suggesting that atypical thalamic contributions may underlie the abnormalities in resting-state alpha oscillations observed in autism. These structural findings in nonautistic and autistic populations suggest that differences in subcortical regions, such as the thalamus and hippocampus, may modulate alpha oscillations. Our study examined alpha oscillations across both stimulus-driven and resting-state conditions in autistic adults, particularly focusing on the relationship between alpha amplitude, sensory behaviors, and structural brain characteristics.

METHODS

Participants

Participants included a total of 55 adults (ages 18–31), 30 of whom were autistic[1] and 25 nonautistic participants that served as a comparison group. All participants gave informed written consent to participate in the study, which was approved by the Institutional Review Board of the University of Washington, and received monetary compensation. All participants had normal or corrected-to-normal vision. Exclusion criteria for all participants included a diagnosis of epilepsy or other neurological disorders, a past serious head injury, motor impairments, or sensory impairments. To be included in the study, all participants were required to be stable on their medication for at least 3 months. Medication use included 10 autistic subjects using an SSRI, 8 using a stimulant, and 3 using both. Nonautistic participants did not report a history of neurodevelopmental or learning disorders and were required to score <70 (de Vries et al., 2023) on the Autism Quotient-28 (AQ-28; Baron-Cohen et al., 2001).

Autistic participants were required to (a) have a prior ASD community diagnosis; (b) have clinician observed autistic behaviors/symptoms based on a telehealth adaptation of the Autism Diagnostic Observation Schedule- second edition (ADOS-2; Lord et al., 2012) (Module-4 calibrated severity score; Hus & Lord, 2014; Hus et al., 2014), the Childhood Autism Rating Scales-High Functioning (CARS-HF) (Schopler et al., 2010), and the DSM-5 checklist. Note, recruitment and assessment occurred during the Covid-19 pandemic thus our clinical procedures were adapted to minimize in-person contact. For the ADOS, all interview-based question tasks (i.e., *Conversation and Reporting*, *Emotions*, *Social Difficulties and Annoyances*, *Friends and Marriage*, *Loneliness*, as well as Module 4's *Current Work or School*, *Daily Living*, and *Plans and Hopes* tasks) were administered as usual. *Description of a Picture*, *Telling a Story from a Book*, and *Cartoons* were administered via screenshare. *Demonstration Task* was administered as described in the manual, with the examiner tilting their screen down to draw a scene and explaining that the goal was to pretend like the examiner and participant were sitting on opposite sides of the table. Three tasks were adapted for telehealth. For the *Create a Story* task, participants were asked to have available three generic items (e.g., cord/string, leaf, and tissue) and two choice items approximately the size of their hand (no electronics). Items were scored using the ADOS-2 scoring system and protocol sheets. More information about the protocol can be provided upon request and publications are under-review establishing the reliability and validity of the adaptation.

TABLE 1 Demographic, phenotypic, and data quality measures of the autistic and nonautistic participants. (with this caption added, I would delete that text in the second row that says "Demographic and Phenotypic measures").

	Autistic	Nonautistic
Demographic and phenotypic measures	Mean (SD); range	Mean (SD); range
Age	24 (3); 18–29	23 (3); 19–30
IQ	118 (13); 92–146	114 (9); 100–137
Sex (m/f/no report)	12/17/1	12/11
SRS	71 (10); 52–97	47.6 (6); 36–63
ADOS CSS	6.4 (1.97); 2–9	na
Cooccurring conditions (self-report)	*n*	*n*
ADHD	14	0
Anxiety	20	0
Depression	16	3
OCD	5	0
PTSD	2	0
EEG quality metrics	Mean (SD); range	Mean (SD); range
Artifact-rejected trials/30; high contrast	2.83 (4.72); 0–18	2.52 (3.23); 0–9
Artifact-rejected trials/30; low contrast	2.67 (4.77); 0–23	2.39 (3.85); 0–12
Number of removed ICA components	1.4 (0.13); 1–4	1.8 (0.21); 1–4
Number of bad recording channels	5.5 (0.96); 1–20	9.0 (1.7); 1–26

[1]Identity-first language will be used based on a survey of our participants' preference.

The final sample included 52 participants. The EEG recordings from one autistic and two nonautistic participants were of insufficient quality to include in any analyses and were excluded. One subject (nonautistic) was excluded for having more than 20% bad channels. Bad channels were designated by consensus of three experts after visual inspection. One nonautistic subject was excluded for having only 2 out of 30 "clean" (using a ±75 mV rejection threshold) trials for the eyes-open, high-contrast condition. One subject (autistic) was excluded due to persistent, repetitive blinking. There was a trend-level difference in the number of ICA components removed with more removed components in the nonautistic subjects ($t_{50} = -1.83$, $p = 0.073$). There was no statistical difference between groups in the number of bad recording channels or number of artifact-rejected trials ($p > 0.10$). Demographic, diagnostic, and co-occurring diagnoses information for the remaining 52 participants is in Table 1.

IQ was assessed via the Wechsler Abbreviated Scales of Intelligence, 2nd Edition (WASI-2) (Wechsler, 2011). Three subtests (vocabulary, matrix reasoning, and similarities) were adapted to be administered via HIPAA-complaint Zoom and one subtest (Block Design) was administered in-person. The Full-Scale IQ based on four subtests was utilized for the current analysis.

Behavioral measures

The Adolescent/Adult Sensory Profile (Brown, C. & Dunn, W. Adolescent-Adult Sensory Profile: User's Manual) was used to assess whether our neural measures are associated with broad disruptions in sensory behavior. The questionnaire consists of 60 items that gauges an individual's sensory experiences in everyday situations, including responses to auditory, visual, tactile, and olfactory stimuli. It measures sensory processing patterns across four quadrants (registration, seeking, sensitivity, and avoiding). Dunn's model of sensory processing (Dunn, 1997) suggests that individuals can be characterized along two dimensions: a neurological threshold (e.g., a high threshold would require a stronger or more intense input to elicit a behavioral response) and a person's reaction (passive or active) to a sensory stimulus. For the purposes of the analyses presented here, we assumed that alpha amplitude—a relatively low-level sensory/perceptual neural response—would be most related to the "threshold" dimension. Thus, we combined "low registration and sensory seeking" scores as they indicate a high sensory threshold, and combined "sensory sensitivity and sensory avoiding" scores as they indicate a low sensory threshold.

We used a brief survey to assess general sleep quality. Participants responded to, "Considering the past week, how would you rate your sleep quality?" and could respond with: (1) Very good (wake up feeling refreshed every morning) (2) fairly good (wake up feeling refreshed more than 3 days a week), (3) fairly bad (wake up unrefreshed more than 3 days a week), and (4) very bad (wake up feeling unrefreshed every morning). Responses were converted to a 1–4 numerical scale.

Stimulus and experimental design

Stimuli were circular counterphase flickering checkerboards (Figure 1a). Each check subtended 1° of visual angle. The radius of each checkerboard was 6° and each of two checkerboards was centered 8° to the left and right of the central fixation cross. A small Gaussian blur (SD = 0.05°) was added to the edges. Two luminance contrasts were used, high (100%) and low (2%; which is just barely visible). Each phase of the checkerboard was presented for 10 frames which, at the 60 Hz refresh rate, corresponded to a flicker rate of 3 Hz (i.e., one complete cycle of each checkerboard phase occurred three times in 1 s). The flicker was used to induce a strong visual response to the stimuli. A single trial contained 10 s of stimulus (low or high contrast) and 20 s of a blank-screen fixation period. Stimuli of a particular contrast were confined to a single run of six trials. At the end of a run of trials, text was presented to "shut your eyes." The eyes-closed condition lasted 20 s. Each contrast run was done five times for a total of 30 trials per contrast condition. Runs of different contrast were alternated. Breaks of up to 3 min were provided between blocks and total acquisition time was 40 min. The stimuli were presented on a luminance-calibrated LCD monitor positioned 57 cm in front of the participants.

Procedure

The protocol included 5–7 in-person experimental visits. Consent was obtained prior to any assessments or procedures, which took approximately 15 min. IQ and autism assessments were administered remotely in a standalone visit that took approximately 60–120 min. A 60 min EEG session was either done on its own visit or occasionally grouped with other experimental procedures. The MRI data were collected as part of its own visit that included fMRI scanning (not reported here).

For EEG, subjects were seated at a table that had the monitor and eye-tracker. Room lights were turned off during the experiment. A chin rest was used to help maintain subject position throughout the duration of the experiment. Subjects were asked to maintain central fixation throughout the duration of the trials. Eye tracking was used to monitor fixation. Due to the particular table/monitor/eye-tracker constraints of our setup, we were able to record eye tracking data from approximately 70% of our subjects (the camera angle of the tracker was such that for the other subjects we could not reliably

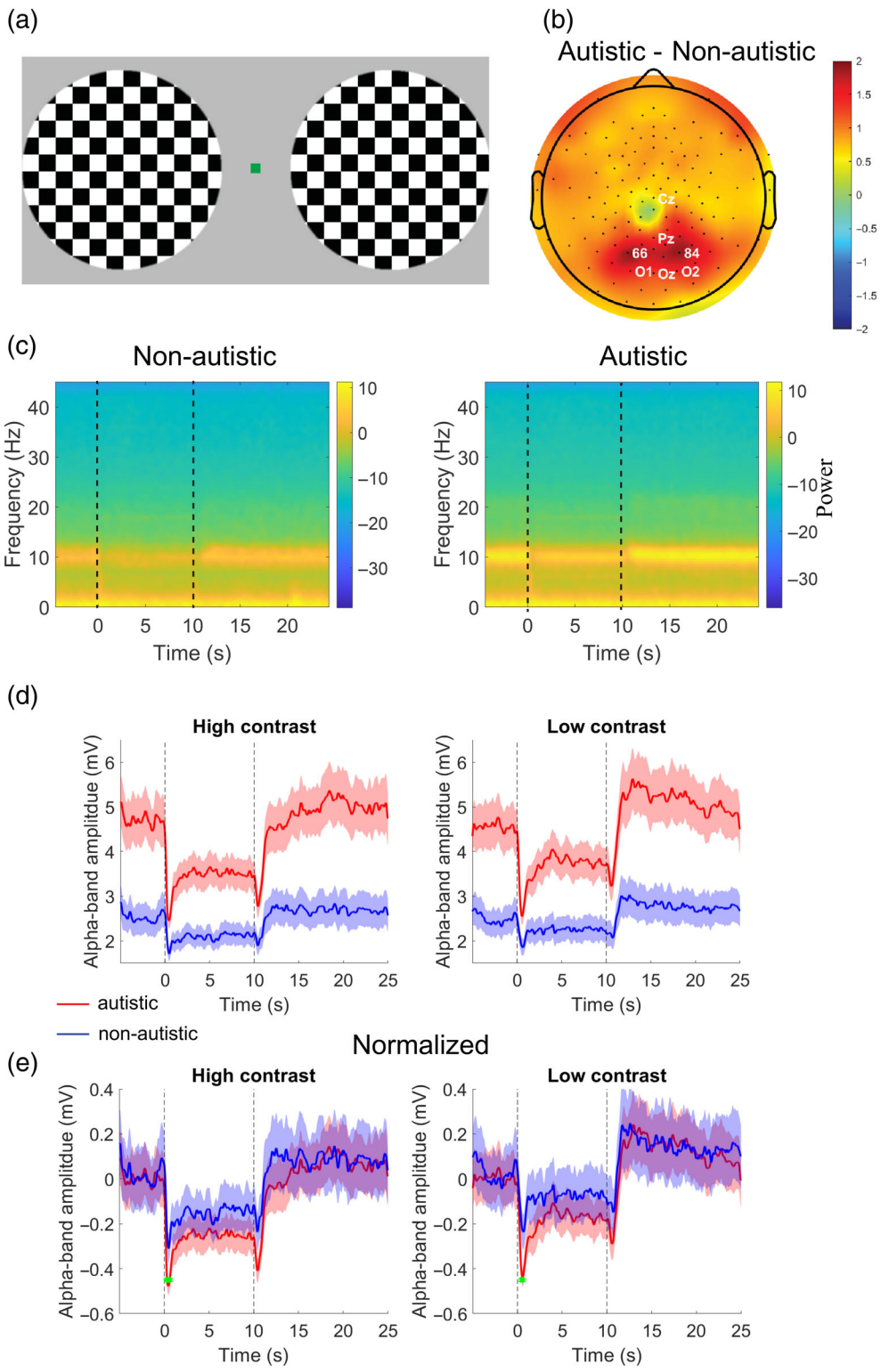

FIGURE 1 (a) Schematic of the stimulus configuration which varied between fixation-only, high contrast flickering checkerboards (shown) and low-contrast flickering checkboards. (b) Scalp topography of differences in alpha amplitude comparing autistic minus nonautistic. The largest differences were observed in channels 66 and 84. (c) Time-frequency plots of spectral power for the nonautistic and autistic groups averaged in channels 66 and 84. (d) Alpha-filtered timeseries of responses comparing autistic (red) and nonautistic responses averaged in channels 66 and 84. (e) The mean value of the prestimulus period was used to normalize responses for each group. This effectively equated alpha amplitude at all timepoints except at stimulus onset. A two-sample t-test of the normalized response performed at each timepoint shows a significant difference in a brief window just after stimulus onset, an effect replicated in both the high and low contrast conditions (green $*p < 0.05$).

calibrate). Specifically, 18 out of 29 autistic subjects and 19 out of 23 nonautistic subjects had usable eye tracking data. Setting an arbitrary threshold of 1° around fixation as "maintaining fixation," the autistic group maintained fixation an average of 86% (sd = 0.18) of the time (over the entire 30 s trial duration) and the nonautistic group 84% (sd = 0.22).

There were no substantive Covid-19-related changes to our standard EEG procedures and all procedures were consistent across groups. However, to minimize in-person contact, we arranged the EEG experimental room to be adjacent to a control room that had a one-way window and a two-way, baby-monitor style communication device. This way the experimenter was able to remain in

the control room for nearly the entire duration of the experiment and was able to control the start/stop of experimental stimulus display computer. Masks were used by participants and experimenters.

EEG recordings and processing

Scalp potentials were measured with Geodesic EEG System 300 (Electrical Geodesics, Inc.) using 128-channel hydrocell sensor nets. After application of the sensor net, impedances were measured and adjusted to 50 kΩ, wherever possible. The acquisition sampling rate was 250 Hz. Acquired data were processed using EEGLab Toolbox (Delorme & Makeig, 2004) to detect and mitigate artifacts, tabulate statistics, and summarize results. EEGLab functions were used to downsample (to 100 Hz) and band-pass filter the continuous scalp potential time series. The low frequency cutoff was 0.25 Hz with a transition band of 0.5 Hz; the high frequency cutoff was 45 Hz with a transition band of 10 Hz. The continuous recording was then divided into 30-second trial segments beginning 5 s before the onset of each visual stimulus. These trial segments were concatenated and inspected for channels containing bad signals due to poor scalp contact or other persistent non-physiological artifacts. The signals on these bad channels were excluded from analysis, and when needed, replaced by spatial interpolation of signals from neighboring sensor locations. Signals from the remaining channels were then processed using independent component analysis (ICA) (Makeig et al., 1996). Descriptive statistics were used to estimate each component's probability of belonging to seven possible categories (Pion-Tonachini et al., 2019). Components with high probabilities in the "Eye" category were inspected and removed. Remaining components were remixed to create new trial segments free from large oculomotor artifacts.

For time sequence analysis of alpha signal, the trial segments were additionally filtered by multiplying the frequency spectrum with a Gaussian window function centered on 10 Hz, with SD of 1.25 Hz. This filtered spectrum was inverse-Fourier transformed back to the time domain. The Hilbert transform was then applied to determine the amplitude of the alpha band signal as a function of time.

To identify spatial differences in alpha amplitude between groups, we compared topographies (autistic—nonautistic) and observed the most pronounced group differences in lateral parietal electrodes, particularly channels 66 and 84 (Figure 1b). Specifically, we subtracted the mean alpha amplitude across the 30 s trial and identified the two electrodes with the largest difference between the autistic and nonautistic groups. While our primary analyses are centered on these channels (66 and 84), similar patterns were noted in adjacent occipital-parietal electrodes.

MRI scanning and analysis

On a separate day, subjects participated in an MRI experiment, which included the collection of a T1-weighted anatomical scan that served as the basis of the data presented here. Data were acquired using a Philips Achieva 3-tesla scanner with a 32-channel high-resolution head coil. T1-weighted MPRAGE structural MRI data were acquired in each session at 1 mm isotropic resolution. FreeSurfer's (ver. 6.0.1) recon-all function was used to automatically segment and reconstruct cortical and subcortical volumes from the structural MRI data (Fischl et al., 2002, 2004; Jovicich et al., 2009). This process involved intensity normalization, skull stripping, and the segmentation of white matter to identify cortical and subcortical structures. The cortical surface was reconstructed to measure total cortical volume. Subcortical structures, including the amygdala, nucleus accumbens, putamen, pallidum, hippocampus, thalamus, caudate nucleus, were segmented to obtain volumetric measures.

Statistical analysis

To examine changes in alpha amplitude across the course of the experiment, mixed-effects models were employed to account for individual variability in alpha oscillation trends over time. Alpha amplitude was averaged across time points for each trial. First, linear mixed-effects models were used to assess whether there was a significant linear increase in alpha amplitude over the duration of the experiment within each group (autistic and nonautistic). For each group, a separate model was fit with *trial number* as a fixed effect, and random intercepts and slopes for *subjects*. To compare the rate of increase in alpha amplitude between the two groups, an additional mixed-effects model was fit including an *interaction term* between *group* and *trial number*. This interaction model was designed to test for any differences in the slopes (i.e., the rate of change in alpha amplitude across trials) between the autistic and nonautistic groups.

To investigate the relationship between alpha amplitude and neuroanatomical features, we first assessed whether total cortical volume was a significant predictor of alpha power. Given that alpha amplitude represents a global neural signal, we hypothesized that it might be related to the overall amount of cortical tissue. Using the Statsmodels package in Python (Seabold & Perktold, 2010) we performed a multiple regression analysis to examine the association between total cortical volume and alpha amplitude, controlling for age, sex, and group status (autistic vs. nonautistic) as covariates. This model aimed to account for demographic and diagnostic factors that might confound the relationship between cortical volume and alpha power.

Next, we extended the analysis to explore the specific contributions of subcortical nuclei to alpha power. We performed separate multiple regression analyses for seven subcortical structures: the amygdala, nucleus accumbens, putamen, pallidum, hippocampus, thalamus, and caudate nucleus. The mean volumes of the left and right hemispheres for each subcortical structure were used, given strong bilateral symmetry. Partial correlation coefficients, controlling for total cortical volume, confirmed bilateral symmetry (with correlations ranging from 0.62 to 0.89), so mean values were used for simplicity.

Each regression model included age, sex, group status, and the volume of the subcortical structure as covariates. In addition, an interaction term between group status and subcortical structure volume was included to examine whether the relationship between subcortical volume and alpha power differed between the autistic and nonautistic groups. This interaction term allowed us to assess group-specific differences in the structural-functional relationship. Each subcortical structure was modeled separately in its own regression, resulting in a total of seven models. The models aimed to determine whether subcortical volume contributed to alpha amplitude beyond the influence of total cortical volume and whether any interactions between group status and subcortical volume predicted alpha power. Lastly, *p*-values from the multiple regression models were corrected for multiple comparisons using the Bonferroni correction to control for familywise error across the seven models.

RESULTS

To validate the focus on alpha-band oscillations, we conducted a full-spectrum inspection of ongoing oscillations, visualized through spectrograms of spectral power (Figure 1c). This analysis highlighted two key findings: the dominance of power within the alpha-band (~10 Hz) and additional low-frequency activity (~1–2 Hz). No notable responses were observed at any other frequency band. The spectrograms reveal the characteristic alpha suppression during checkerboard-induced visual stimulation (the onsets and offsets indicated by vertical dashed lines), and demonstrate increased overall increased alpha power in the autistic group, that we next characterize in more detail.

Alpha amplitude increase in ASD

To analyze the time course of ongoing alpha activity, we segmented the timeseries into 30-second intervals, starting 5 s before each visual stimulus onset. As shown in Figure 1d, alpha amplitude was consistently higher in autistic individuals compared with nonautistic individuals across all time points in both low and high contrast conditions. This pattern suggested a baseline elevation in the autistic group's alpha response. To quantify this observation, we used the average of the 5-second prestimulus period to normalize the timecourses. Normalizing resulted in comparable alpha responses between the groups, with the notable exception of a brief period after stimulus onset (approximately 170–730 ms, noted in green in Figure 1e and Figure 2b). In this window, the autistic group showed more pronounced alpha suppression, a reduction in alpha amplitude beyond what normalizing could account for.

We further investigated alpha amplitude variations during eyes-closed conditions, which typically show an increase in alpha activity. Trials instructing participants to close their eyes (with visually displayed instructions) were included at the end of each run, following 20 s of a blank screen with fixation target only. We observed a significant elevation in alpha amplitude during the eyes-closed periods in both groups (average of 5–15 s versus preeyes-closed baseline; autistic, $p < 0.01$; nonautistic, $p = 0.01$), as expected, and this response was heightened in the autistic group compared with the nonautistic group (Figure 2). Notably, normalizing equated the alpha timecourses for nearly all timepoints. The only exception was the alpha suppression response to the visual instruction to close the eyes. This finding mirrors the earlier observed failure of normalizing in the eyes-open condition, specifically during the brief period after stimulus onset, thereby replicating the pattern of differential alpha suppression response in both eyes-open and eyes-closed scenarios in autistic individuals.

Control analyses

A number of state factors, such as fatigue may influence alpha amplitude (Tran et al., 2020). We observed no apparent relationship between sleep quality and alpha amplitude (Figure S1). We then examined alpha power amplitude across successive trials, potentially indicative of changing state during the experiment (Figure 3). Using a mixed-effects model to account for individual variability, both groups exhibited a significant linear increase in alpha amplitude over the duration of the experiment (autistic, $p < 0.0001$; nonautistic, $p = 0.0004$). However, the comparison of the rate of increase between groups, indicated by the interaction term, was not statistically significant ($p = 0.2915$). This implies that there is no evidence to suggest that the rate of this increase differs between the two groups. That is, both groups experienced a similar trend of increased alpha amplitude over time.

Lastly, we consider the use of psychotropic medications among young adults with ASD. Specifically, we evaluated the impacts of SSRIs and stimulant use, as these were the two most commonly prescribed medication classes in our sample. Our findings indicate that neither SSRIs nor stimulants usage can explain the increased alpha observed in the ASD group; in fact, they

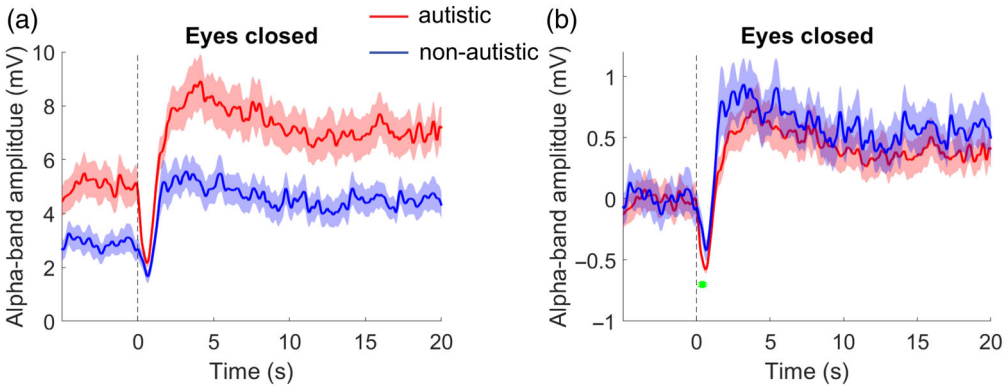

FIGURE 2 (a) Alpha-filtered timecourses during the eyes-closed condition. Vertical dashed bar indicates the onset of the visual cue to close eyes. (b) Normalizing the timecourses effectively equated for magnitude differences (except for the alpha-suppression in response to the visual cue, green $*p < 0.05$).

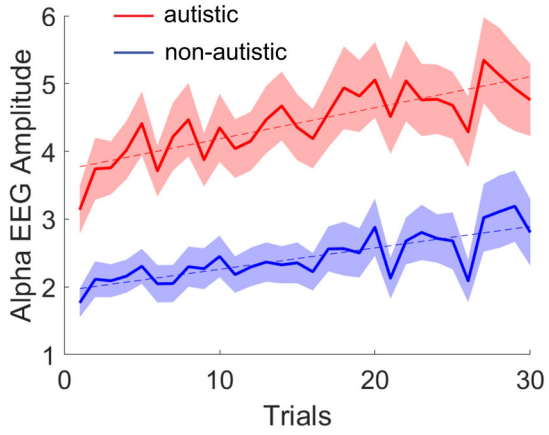

FIGURE 3 Average alpha amplitude over the course of the experimental session.

were both associated with a reduction of alpha when compared with autistic participants who are not using these medications (Figure S2).

Sensory symptoms

The difference in the alpha suppression response to visual onset transients (Figure 1e) in the autistic group may have behavioral relevance, as assessed with Sensory Profile (see Table 2 for score breakdown). We used the normalized magnitude of alpha in channels 66 and 84 in an onset window (defined as the temporal window of significant difference between groups—green points in Figure 1e—from 340 to 720 post stimulus onset) in each autistic subject to characterize "onset alpha." Within the autistic group, our analysis revealed distinct associations between EEG alpha responses at visual onset and behavioral patterns on the Sensory Profile (Figure 4). Specifically, a higher onset alpha amplitude (less suppression) was associated with a decrease in "Low Registration/Seeking" behaviors ($r = -0.41$, $p < 0.04$), indicating that individuals with stronger postonset alpha responses may be less inclined to seek out sensory experiences or may not notice sensory stimuli as readily. On the other hand, the same increase in onset alpha amplitude was positively trend-level correlated with "Sensitivity/Avoiding" behaviors ($r = 0.36$, $p = 0.07$), indicating that individuals with more pronounced alpha responses may also be more sensitive to sensory stimuli and more likely to avoid them.

Neuroanatomical correlates

We investigated the neuroanatomical correlates to alpha power increases in ASD beginning with a consideration of the potential influence of total cortical volume. The model significantly predicted alpha power, $F_{(4,47)} = 6.03$, $p = 0.001$, adj. $R^2 = 0.283$. Total cortical volume ($t_{(47)} = 3.167$, $p = 0.003$) and group status ($t_{(47)} = 2.627$, $p = 0.012$) were each separately significant predictors of alpha amplitude while age ($t_{(47)} = -1.047$, $p = 0.30$) and sex ($t_{(47)} = -1.505$, $p = 0.139$) were not. The relationship between total cortical volume and alpha amplitude is unique to the autistic subjects (Figure 5a). We note that total cortical volume did not differ by group ($t_{(49)} = 1.3$, $p = 0.18$).

Our analyses also sought to identify which subcortical nuclei, if any, were associated with alpha amplitude beyond the influence of total cortical volume. Alpha magnitude was significantly explained by each of these models, with F-statistics ranging from 3.93 to 7.60 (all Bonferroni adjusted p values were <0.05) and adjusted R^2 values ranging from 0.26 to 0.44. Of the subcortical structure volume predictors, we found that only the model that included the amygdala showed that the subcortical structure volume term significantly predicted alpha power ($t_{45} = -2.045$, $p = 0.047$). We also found that alpha magnitude was significantly predicted from the group-by-structure-volume interaction term for the model that included the hippocampus ($t_{45} = 3.853$, $p < 0.001$), and the model that included the thalamus ($t_{45} = 2.016$, $p = 0.0498$). Nevertheless, following correction for multiple comparisons, only the interaction term including hippocampal volume remained statistically significant. These results suggest that a group difference in the relationship between alpha magnitude and

TABLE 2 Sensory profile measures in the autistic and nonautistic participants. (with this caption added, delete the text "Sensory Profile" in the top left cell).

	"High threshold"		"Low threshold"	
Sensory profile	**Q1; mean (SD)**	**Q2; mean (SD)**	**Q3; mean (SD)**	**Q4; mean (SD)**
Autistic	40.9 (7.9)	42.5 (8.8)	48.9 (10.1)	49.7 (9.0)
Nonautistic	27.5 (6.6)	45.6 (6.6)	30.6 (6.9)	33.9 (8.9)

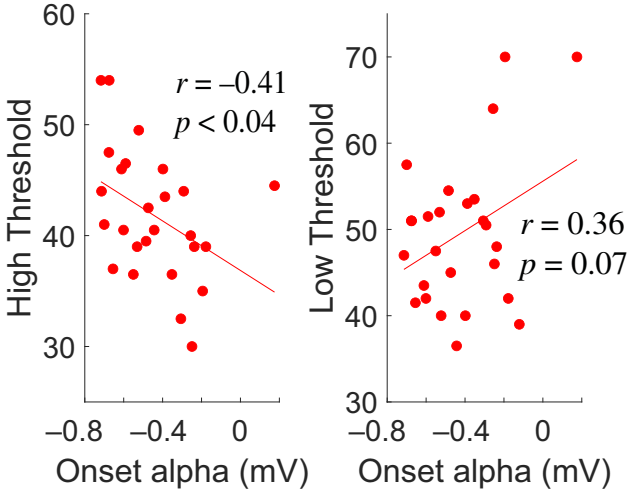

FIGURE 4 The relationship between normalized alpha amplitude at stimulus onset and scores on the Sensory Profile. Left: a combination of scores related to "low registration/low seeking." Right: a combination of scores related to "Sensitivity/Sensory-avoiding."

hippocampal volumes significantly contribute to the prediction of alpha (Figure 5b).

DISCUSSION

This study characterized alpha-band oscillations in autistic individuals in comparison to nonautistic individuals across a variety of passive viewing conditions, including blank-screen fixation, and low and high contrast flickering checkerboards, as well as during eyes-closed conditions. Our objective was to establish baseline measurements of alpha activity, which could subsequently inform research on task-based modulations of alpha through attention manipulations. Contrary to our initial expectations, we observed a substantial and uniform increase in alpha amplitude in the autistic group across all conditions. This finding suggests a consistent alteration in baseline neural activity in autistic adults with average or higher cognitive functioning.

The striking increase in alpha amplitude observed in our autistic individuals was surprising given the widespread use of EEG as a measurement tool in autism research and the prominent, easily detectable nature of alpha responses. However, a recent systematic review and meta-analysis (Neo et al., 2023), which synthesized findings from 41 studies involving 1246 autistic and 1455 neurotypical individuals, while contrasting with our results, points to varied findings. The review found that autistic individuals exhibited reduced relative alpha power ($g = -0.35$) and increased gamma power, but similar delta, theta, alpha, and beta power compared with comparison groups (primarily neurotypical individuals). Work from our own group across two larger consortiums of children ($n = 399$) (Webb et al., 2023) and youth ($n = 280$) (Neuhaus et al., 2021, 2023) demonstrates this inconsistency in alpha findings, although both generally are suggestive of decreases in alpha in autistic youth. Notably, the review highlighted substantial heterogeneity in effect sizes across all frequency bands, including alpha, and identified factors, such as resting-state paradigm type and recording duration as moderators for some EEG power differences.

These findings, in contrast to our own, suggest that the increased alpha amplitude observed in our study might not be a universal characteristic of ASD but could be influenced by specific experimental conditions, participant characteristics, or methodological variations. Of interest, in the meta-analysis, only one paper was included that featured an adult autistic sample (Mathewson et al., 2012); in this paper of 15 autistic adults with average or higher IQ, EEG alpha power was higher in the autistic group than controls during eyes open. Other findings have shown increases in alpha (and other frequency bands) in children, the magnitude of which correlated with symptom severity (Cornew et al., 2012). While our results, alongside the meta-analytic evidence, support the potential of resting-state alpha as reflecting altered neural functioning in autism, they also emphasize inherent challenges of characterizing a heterogeneous condition. Finally, although we aimed to recruit as large a sample as was practically feasible given the time and resource constraints of the project, the possibility that our limited sample size and specific sample characteristics may have contributed to the discrepant results cannot be ruled out. Future studies with larger and more diverse samples will be essential to validate and extend our findings.

Our observation of increased alpha amplitude in the autistic group seems potentially counterintuitive in the context of current understanding of sensory processing in autism. Alpha oscillations are typically linked to sensory inhibition, with a decrease in alpha power often seen during active sensory processing or attentional engagement. Since autism is often characterized by sensory hypersensitivity, one might expect alterations in alpha activity that mirror these sensory processing

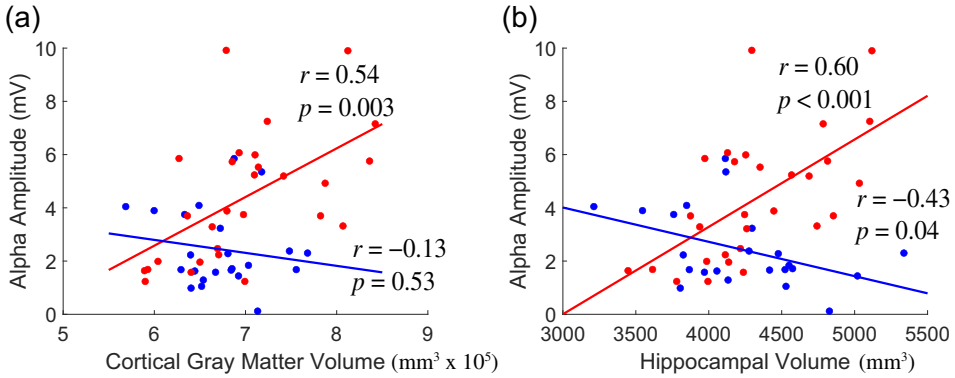

FIGURE 5 Correlations between mean alpha amplitude and total cortical gray matter volume (a) and hippocampal volume (b).

differences—possibly reduced alpha power indicative of a heightened sensory state or sensory over-responsiveness. However, our findings are potentially consistent with a recent finding in a mouse model of autism that found that perceptual deficits may stem from elevated inhibitory and diminished excitatory neuronal activity in the primary visual cortex (del Rosario et al., 2021). This study observed that during visual perceptual tasks, knockout mice not only exhibited increased firing from inhibitory neurons and reduced excitatory neuron activity but also demonstrated reduced neural sensitivity to contrast. In particular, they observed a marked increase in LFP firing in the 3–10 Hz range. This pattern of neural activity aligns closely with the heightened alpha responses we observed in autistic individuals. A notable exception to the baseline difference in alpha was identified in the brief ~500 ms window following stimulus onset, a period typically characterized by alpha suppression (also known as event-related desynchronization, ERD). In this window, autistic individuals exhibited more pronounced alpha suppression after normalizing by prestimulus alpha amplitude. This suggests an alteration in the neural response to sensory stimuli, particularly during the initial processing stage following stimulus presentation. The possibility that this heightened alpha suppression observed in autistic individuals may reflect unique aspects of sensory processing was further suggested by correlations with responses on the Sensory Profile.

Several studies have explored the relationship between alpha power and behavioral measures of sensory processing in both autistic and neurotypical populations, though this area remains relatively under-explored. Pierce et al. (2021) investigated sensory processing in children with autism and found reduced resting-state alpha power compared with neurotypical controls. Interestingly, while differences in alpha power were observed between groups, no direct association between alpha levels and sensory symptoms was found in the autistic children ($n = 31$). Instead, the authors suggested that hemispheric asymmetries in alpha activity may be more sensitive to sensory processing differences than absolute alpha power. Simon et al. (2017) examined sensory hypo-responsiveness in high-risk toddlers and identified correlations between sensory hypo-responsiveness and patterns of frontal alpha asymmetry. Their results indicated that relative right frontal alpha dominance was associated with increased sensory hypo-responsiveness, suggesting that variations in alpha oscillations may reflect underlying neural mechanisms contributing to altered sensory responsiveness in autism.

Similarly, Damiano-Goodwin et al. (2018) reported that elevated sensory seeking behavior in elevated-risk-for-autism infants ($n = 20$) was associated with atypical frontal alpha asymmetry. Not only was sensory seeking linked to reduced social orienting, but this atypical sensory behavior also predicted later social symptomatology. These findings suggest that alpha asymmetry, particularly in frontal regions, may serve as a neural signature for certain patterns of sensory responsiveness in autism and could have cascading effects on social development.

In the current study, we observed relationships between sensory thresholds and alpha oscillations, with distinct correlations between increased alpha power at stimulus onset and both low- (sensitivity/avoiding) and high-threshold (low registration/seeking) behaviors. However, we acknowledge the possibility that these correlations may reflect more complex patterns of sensory modulation. Specifically, the positive association between alpha amplitude and low-threshold behaviors may be driven in part by a few high scores, underscoring the need for caution in interpreting this pattern. Additionally, it is possible that low and high threshold behaviors cooccur within individuals, reflecting variability across sensory domains rather than distinct profiles of hypo- versus hyper-responsiveness. Future research should explore how these behaviors interrelate within individuals and whether they correspond to different neural mechanisms, such as frontal asymmetries, as suggested by prior studies. While our findings contribute to the growing evidence for links between neural oscillations and sensory behaviors in autism, they also highlight the challenges inherent in characterizing sensory modulation within a heterogeneous population.

Similar to our observation of increased overall alpha amplitude in our autistic subjects, our finding of greater

alpha desynchronization at stimulus onset generally runs counter to previous findings. For example, Ewen et al. (2016) found that children with high-functioning autism (HFA, $n = 25$) exhibited significantly less alpha and beta ERD in response to a praxis task compared with typically developing controls. This blunted ERD was particularly evident in the left parietal and central regions and was associated with greater impairments in imitation and increased autism severity. This contrasts with our findings of more pronounced alpha suppression in autistic adults following stimulus onset, suggesting that while there may be a general alteration in alpha dynamics in autism, the direction and magnitude of these changes may vary depending on the specific task and population being studied.

Similarly, Keehn et al. (2017) observed that children with ASD ($n = 19$) showed reduced posterior alpha desynchronization in response to behaviorally relevant targets during an attentional task, coupled with decreased resting alpha power. This lack of alpha desynchronization was linked to poorer target detection and increased autism symptomatology. In contrast, our study found heightened alpha suppression following stimulus onset in autistic adults, which may suggest that the neural mechanisms underlying alpha modulation in response to stimuli are different in adults with autism compared with children. The discrepancy between our findings and those of Ewen et al. (2016) and Keehn et al. (2017) could reflect differences in the age of participants, the cognitive demands of the tasks, or the specific aspects of alpha modulation being measured.

Our finding of a strong association between total cortical volume and alpha amplitude in autistic participants suggests a potential neuroanatomical basis for the observed differences in alpha dynamics. Specifically, within the autistic group, individuals with larger cortical volumes exhibited higher alpha amplitudes. Notably, this correlation was only present in the autistic participants. Additionally, there was no significant difference in total cortical volume between the autistic and nonautistic groups, suggesting that other factors beyond cortical volume alone must be contributing to the differences in alpha amplitude observed in autism.

We analyzed the volumes of subcortical structures, following two recent observations in the literature. First, Edgar et al. (2015) demonstrated that MEG eyes-closed alpha amplitude in nonautistic children correlated with thalamic volume, a relationship that was absent in autistic children. However, interpreting the association between the volume of a single subcortical region, such as the thalamus, is challenging because subcortical structure volumes are strongly correlated with total brain size and cortical volume. For example, in our data, the volume of thalamus and total cortical volume were highly correlated ($r = 0.77$, $p < 10^{-10}$). This association complicates the isolation of specific subcortical volumetric contributions to alpha oscillations. Second, in nonautistic adults, Ghafari et al. (2024) found that hemispheric asymmetries in the volumes of the thalamus, caudate nucleus, and globus pallidus were predictive of MEG alpha-band power modulation during attention tasks. In contrast, our study did not find a similar association between the volume of these subcortical structures and alpha amplitude, except for a significant interaction between group and hippocampal volume. Given that the hippocampus is a relatively large subcortical structure, this relationship might parallel the association between total cortical volume and alpha amplitude, suggesting that larger brain structures in general may be linked to increased alpha power in autism.

What is clear from our findings is the need for future studies examining any attention-mediated effects on alpha in autism to account for individual and group differences in baseline alpha amplitude. Current existing evidence suggests that the deployment of attention-mediated, alpha-band suppression may be disrupted in autism (Keehn et al., 2017; Murphy et al., 2014). Thus, if a higher baseline alpha amplitude is a characteristic feature in some individuals with ASD, this could potentially influence the magnitude or direction of alpha modulation during attentional tasks. Our results raise the question of whether the patterns of alpha suppression or enhancement in response to cognitive demands are altered in the context of this elevated baseline.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

ETHICS STATEMENT

All subjects provided written, informed consent prior to participating. Research protocols were approved by the Institutional Review Board of the University of Washington, and conformed to the ethical principles of the Declaration of Helsinki for research involving human subjects.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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