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# Altered aperiodic EEG spectral power during speech perception task is associated with verbal communication in youths with Autism Spectrum Disorder

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**Most children with Autism Spectrum Disorder (ASD) have co-occurring language impairment, but its neural mechanisms are not well known. Excitation (E) / inhibition (I) imbalance is considered as a key neurobiological mechanism of ASD, and several electroencephalography (EEG)-based E/I balance metrics have been proposed in the previous studies. The goal of the present research was to focus on these metrics abstracted from the speech perception task to investigate their relation to language/communication in autistic youths. We used a high-density 128-channel EEG to register neural responses during speech perception task in the sex- and age-matched groups of youths with ASD (*N* = 162) and typically developing (TD) controls (*N* = 144), aged 7–18 years old. The results revealed alterations in the E/I measures in the ASD group hypothetically associated with a higher level of excitation or neural 'noise' in the cortex as well as broadband reduction of spectral power during speech perception. A greater neural 'noise' reflected in the reduction of aperiodic exponent and offset was related to lower verbal communication but not language skills in youths with ASD. The findings suggested that the higher 'noisiness' in the cortical systems may be a relevant marker to monitor in relation to verbal communication in ASD.**

**Keywords** Autism Spectrum Disorder, Gamma oscillations, Periodic and aperiodic EEG, Excitation/inhibition imbalance, Language impairment, Neural 'noise'

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition associated with the difficulties in social interaction/communication and restricted and repetitive behaviors[1]. It is known that there is a high heterogeneity in language abilities in individuals with ASD, from above average language skills (~25%) to severe language impairments with minimal or even no verbal skills[2–5]. Although language impairment is not a diagnostic criterion of ASD, previous studies have shown that difficulties in language/verbal communication are one of the

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first signs of ASD at early stages of development and can be related to later ASD outcomes[6,7]. However, neural mechanisms of these difficulties are still unknown, and there is a need to develop a set of biomarkers of language/verbal communication impairments in ASD for using as objective measures in clinical trial studies.

Excitation (E)/inhibition (I) imbalance in different brain networks is considered an important neurobiological mechanism in ASD[8–13]. At the system level, multiple studies have suggested that altered interplay between glutamatergic (excitatory) and gamma-aminobutyric acidergic (GABAergic; inhibitory) neurotransmission causes a reduction in signal-to-noise ratio in key neural circuits[12]. This, in turn, may cause an enhanced (or reduced) "noise" in the cortex, which impacts synaptic plasticity during development and results in less effective information processing[12,14,15]. Although alterations in E/I balance can have different causes (elevated E over I as well as reduced E over I, see[8,16–18], most studies reported increased excitatory activity in the autistic brain[11,12]. Several electro-/magnetoencephalography (EEG/MEG)-based E/I balance biomarkers have been proposed to index language and verbal communication impairments in ASD and related disorders[19–25].

Gamma-band neural activity (30–80 Hz) in EEG/MEG is one of the most frequently reported non-invasive measures of E/I balance in the cortex, and it is generated mostly by GABAergic interneurons, expressing calcium-binding protein parvalbumin (PV + inhibitory cells)[12,26–31]. Previous studies have shown atypicalities in gamma activity and relationships between gamma power and language/verbal communication skills in autistic individuals and their first-degree relatives[19,21,32–41]. For example, atypical gamma activity was detected in response to low-level auditory stimuli, such as 40 Hz amplitude-modulated tones and sweeps, and those atypicalities were associated with lower language skills[19,36]. Similar abnormalities in gamma activity were detected in autistic individuals and their first-degree relatives when presenting linguistic stimuli, such as syllables, words, and sentences[21,33,34]. In addition, resting-state EEG gamma oscillations were also associated with language skills in toddlers with ASD and their siblings and/or parents as well as in individuals with Fragile X Syndrome, a single-gene disorder related to ASD[25,41,42].

Development of newer methods for EEG/MEG power analysis has allowed parametrization of spectral power into periodic and aperiodic components to provide more accurate estimation of both oscillatory and non-oscillatory (broadband) activity associated with different neurobiological mechanisms[43–45]. The aperiodic component of the signal can be defined by the 1/f power law distribution that underlies the absolute power spectra[43,45–48] and consists of aperiodic exponent and aperiodic offset. Different modeling studies have shown that the exponent reflects E/I balance and its characteristics can indicate whether the circuit has increased excitatory or inhibitory activity[46], while offset reflects broadband neuronal firing[47]. Investigation of the aperiodic signal together with the oscillatory part of the spectra may significantly contribute to our understanding of the mechanisms at the system level[24].

Although this approach is relatively novel, there is evidence of a relationship between the aperiodic component and language/verbal communication skills in ASD and related conditions. For example, Wilkinson and Nelson[25] found that the aperiodic component of spectral power in the gamma frequency range was altered in Fragile X Syndrome and was related to communication skills in this population. Using MEG, Arutiunian and colleagues[21] demonstrated that aperiodic offset in the auditory cortex during pre-stimulus/baseline period was associated with language abilities in children with ASD. Finally, Wilkinson et al.[24] observed that developmental changes in aperiodic offset and exponent (from 3 to 12 months) predict lower language skills at 18 months in infants with a family history of ASD.

To summarize, previous studies using different hypothetical EEG/MEG measures of E/I balance have demonstrated its alterations in ASD. Findings have largely suggested the E/I profile shifted toward excitation in the autistic brain in response to auditory and speech stimuli, with increased excitation related to lower language and/or communication skills. The aims of the present study were to: 1) comprehensively investigate these neural measures in response to speech stimuli in a large, sex-balanced group of children with and without ASD, and 2) explore associations between these measures and language/communication in autistic youth. *First,* for between-group comparisons we applied a parameterization approach to calculate periodic and aperiodic components of power spectra. We abstracted measures related to E/I balance used in the previous studies: oscillatory gamma power, aperiodic exponent, and aperiodic offset. Based on findings from previous studies, we hypothesized that E/I balance would be altered in ASD, with the profile shifted toward excitation. *Second,* we hypothesized that this could potentially be related to language/communication in the ASD group.

## Methods

### Participants

In total, 306 English-speaking participants (7 to 18 years) took part in the study: 162 youth with ASD (90 male, 72 female) and 144 age- and sex-matched TD controls (77 male, 67 female). Sex was based on parent report of sex assigned at birth. Data were collected from four sites as a part of the GENDAAR Autism Center for Excellence Network, including Seattle Children's Research Institute, Boston Children's Hospital, the University of California in Los Angeles, and Yale University.

The study was approved by the Yale Institutional Review Board, the UCLA Office of Human Research Protection Program, Boston Children's Hospital Institutional Review Board, USC Office for the Protection of Research Subjects, and the University of Virginia Institutional Review Board for Health Sciences Research. All performed procedures were in accordance with the Declaration of Helsinki. All minor children provided verbal assent to participate in the study and were informed that they can withdraw from the study at any time. A written consent form was obtained from a parent of each child participating in the study.

### Behavioral assessment

All participants from the ASD group had a clinical diagnosis based on the DSM-4–TR or DSM-5[1,49] and the diagnosis was confirmed with the Autism Diagnosis Observation Schedule – Second Edition[50], Autism Diagnostic

Interview – Revised[51], caregiver-reported developmental history, and expert clinical judgment. Adaptive skills of all youths were measured with the Vineland Adaptive Behavior Scales – Second Edition (VABS-2), and standard scores (SS) in communication, socialization, and daily living skills domains were calculated with higher scores reflecting better adaptive skills[52]. Verbal and nonverbal IQ were estimated based on the Differential Ability Scales – Second Edition (DAS-II) School Aged Cognitive Battery[53]. Language abilities were measured with the Clinical Evaluation of Language Fundamentals – Fourth Edition (CELF-4), a standardized assessment tool that covers basic structural language skills at different linguistic levels (vocabulary, morphosyntax, semantics, and pragmatics) in both production and comprehension[54]. Participants were administered only those subtests necessary to calculate the CELF-4 Core Language SS, which was used as a measure of overall language skills. Parents were asked to report on their child's autistic traits and social skills using the Social Responsiveness Scale – Second Edition (SRS-2); T-scores for social cognition, social communication, and restricted and repetitive behaviors were calculated for all youths with the higher scores reflecting greater severity[55]. Descriptive statistics are presented in Table 1.

Exclusion criteria were twin status, history and/or presence of known chromosomal syndromes/single-gene conditions related to autism (e.g., Fragile X Syndrome), co-occurring neurological conditions (e.g., epilepsy), significant visual and auditory impairments, or sensory-motor difficulties that would prevent completion of study procedures. TD children had no first or second degree family members diagnosed with ASD, and no elevation of autism traits according to parent report on the SRS-2 (total T-score < 60) or the Social Communication Questionnaire[56], raw score < 11.

## Experimental paradigm and procedure

We used the implicit word segmentation paradigm described in detail in[21] but see also[57–62]. In brief, the experiment consisted of two phases. During the first (exposure) phase, youths heard three-syllabic pseudowords generated from the set of 12 different phonemes (e.g., *pa-bi-ku*), resulting in 180 exposures over ~ 2.5 min. The second (test) phase consisted of 96 trials; duration was 2 min 16 s. Analyses were restricted to the second (test) phase of the experiment. Each trial consisted of a three-syllabic pseudoword with an average duration of ~ 900 ms, followed by a 500–750 ms intertrial interval. A random half of these trials ($N = 48$) used the same pseudowords presented in the first phase during exposure (e.g., *pa-bi-ku*), i.e., 'familiar' items. The remaining random trials ($N = 48$) were constructed by combining the last syllable of each familiar pseudoword with the first two phonemes of other pseudowords (e.g., *pa-bi-ku* and *go-la-tu* became *ku-go-la* and *tu-pa-bi*), i.e., 'unfamiliar' items. The auditory stimuli were presented using a speaker (Logitech speaker system X320) with the same loudness across all participants and sites (65 dB). All participants were instructed to look at the screen and to listen carefully to the 'robot language'. A static robot was presented on the screen simultaneously with the auditory stimuli during the experiment. The speaker was a native American English-speaking female.

## EEG data acquisition and processing

At all four sites, EEG data were collected with EGI 128-channel Net Amps 300 system with HydroCel nets (Magstim EGI Inc., Eugene OR), using Net Station 4.4.2, 4.5.1, or 4.5.2 with a standard Net Station acquisition template. Nets were available without outriders (eye electrodes 125, 126, 127, and 128) for participants with facial sensory sensitivities. The participant's behavior was video recorded during EEG collection. Data were collected at 500 Hz sampling rate, referenced to Cz electrode (vertex), and impedances were < 50 kΩ. To account for an EGI trigger timing error, we incorporated StimTracker event markers, which use auditory and photodiode sensors and 5v inputs to the digital inputs of the amplifiers to assess ground-truth timing accuracy.

To calculate power spectral density (PSD) we used the Batch EEG Automated Processing Platform, BEAPP1.0[63] in MATLAB 2021a, consisting of following steps: (1) formatting the MFF file for Matlab; (2) band-

| Characteristics | Group | | Statistics |
|---|---|---|---|
| | ASD ($N = 162$) | TD ($N = 144$) | |
| Age, months | 151.2 (34.9) | 156.5 (35.3) | $t = -1.32$, $p = 0.18$ |
| Sex, *N* of female participants | 72 | 67 | $\chi^2(1) = 0.13$, p = 0.71 |
| CELF-4 Core Language SS | 91.43 (20.90) | 110.62 (11.26) | $t = -8.74$, $p < \mathbf{0.001}^{***}$ |
| Vineland-2 | | | |
| Communication SS | 76.1 (11.8) | 99.3 (13.2) | $t = -15.9$, $p < \mathbf{0.001}^{***}$ |
| Socialization SS | 72.6 (12.1) | 101.2 (12.6) | $t = -19.9$, $p < \mathbf{0.001}^{***}$ |
| Daily living skills SS | 76.4 (13.7) | 97.7 (14.2) | $t = -13.1$, $p < \mathbf{0.001}^{***}$ |
| Verbal IQ | 100.6 (20.2) | 113.1 (15.6) | $t = -6.0$, $p < \mathbf{0.001}^{***}$ |
| Nonverbal IQ | 100.2 (17.5) | 109.1 (15.1) | $t = -4.7$, $p < \mathbf{0.001}^{***}$ |
| SRS-2 T-score | | | |
| Social cognition | 71.6 (10.9) | 43.8 (5.2) | $t = 27.9$, $p < \mathbf{0.001}^{***}$ |
| Social communication | 74.5 (12.0) | 43.9 (5.2) | $t = 28.2$, $p < \mathbf{0.001}^{***}$ |
| Restricted and repetitive behavior | 73.7 (12.6) | 44.4 (3.5) | $t = 27.0$, $p < \mathbf{0.001}^{***}$ |

**Table 1**. Behavioral characteristics of participants, M(SD). Significance is labeled with $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ and highlighted in bold.

pass filter 1–100 Hz; (3) down sampling from 500 to 250 Hz; (4) implementation of the Harvard Automated Preprocessing Pipeline for EEG (HAPPE1.0) module for artifact detection and rejection[64], including removal of 60 Hz line noise, rejection of bad channels, wavelet enhanced thresholding, Independent Component Analysis (ICA) with automated component rejection, bad channel interpolation, and re-referencing to average; (5) segmentation of the continuous file into 1 s epochs (each epoch consisted of one three-syllabic pseudoword); (6) rejection of bad segments (±40 μV); (7) calculation of the PSD using Hanning window on clean segments.

### EEG variables calculation

To model the periodic and aperiodic components of PSD[43], we used the *specparam 1.1* toolbox[45] in Python v3.10 with the following settings: *peak_width_limit* = [1.5, 8], *n_peaks* = 6, *peak_height* = 0.10, *peak_threshold* = 2, and *frequency_range* = [2, 55]. For each participant, the model provided two parameters (aperiodic offset and aperiodic exponent) to describe the aperiodic 1/f signal. To calculate periodic power (or aperiodic-adjusted power), the aperiodic signal from the model was subtracted from the raw power spectrum, resulting in a flattened spectrum. Three measures were used for statistical analysis: aperiodic exponent, aperiodic offset, and periodic gamma power (35–54.99 Hz). The measures were calculated for nine regions of interest (ROIs; see Fig. 1A).

The ASD and TD groups did not differ in the number of artifact-free epochs in any condition: 'familiar' items or pseudowords, $M_{ASD} = 43.2(1.9)$ vs. $M_{TD} = 43.2(3.6)$, $t(217.1) = -0.21$, $p = 0.82$; 'unfamiliar' items, $M_{ASD} = 43.0(2.0)$ vs. $M_{TD} = 43.4(3.3)$, $t(228.6) = -1.37$, $p = 0.17$.

### Statistical analysis

Statistical analysis was performed in R 2023.03.1 + 446[65] with the *lme4 v1.1.35.5* package[66]. The tables for model outcomes (Tables 2, 3, 4) were created with the *sjPlot v.2.8.17* package[67], data were plotted with *ggplot2 v3.5.2*[68], and the figures, representing neural responses, were created with python data visualization library *matplotlib v3.1.1*[69]. The structure of the models will be specified further in the Results section. A correction for multiple comparisons (false discovery rate, FDR) was applied to the models, and p-values for significant predictors were corrected with *p.adjust.method* in R.

## Results

### Sample characterization

As expected, youths with ASD differed from the TD group in all behavioral measures, having lower skills in language/communication and non-verbal cognition as well as higher presence of autistic traits (see Table 1). There was no difference in age and sex between groups.

### Group, condition, sex differences and age-related changes in neural measures

Figure 1B represents the example of absolute, aperiodic, and periodic spectral power in response to speech stimuli for one ROI (central left). In order to investigate between-group differences in EEG neural measures in response to speech stimuli, we fitted linear mixed-effect models with the EEG metrics as dependent variables and included the main effects of group ('TD' as a reference), condition ('familiar' as a reference), sex ('female' as a reference), region ('frontal' as a reference), laterality ('left' as a reference), age, and group × condition interaction as fixed effects and participants as random intercept. The structure of the models was as follows: *lmer(EEG metric ~ group × condition + sex + region + laterality + age + (1 | ID), data = data, control = lmerControl(optimizer = "Nelder_Mead"))*.

*Region of interest (ROI) and laterality effects in EEG neural measures*

We indicated significant region and laterality effects for all neural measures (see Table 2 for summary).

Specifically, when comparing to frontal ROI, aperiodic exponent was significantly higher in central ROI, $\beta = 0.03$, $t = 7.49$, FDR-corrected $p < 0.001$; aperiodic offset was significantly lower in central and parietal ROIs, $\beta = -0.18$, $t = -34.79$, FDR-corrected $p < 0.001$ and $\beta = -0.03$, $t = -5.45$, FDR-corrected $p < 0.001$; and periodic gamma power was significantly lower in central ROI, $\beta = -0.01$, $t = -8.65$, FDR-corrected $p < 0.001$.

Regarding laterality effects, when comparing to the left EEG subchannels, aperiodic exponent was significantly higher in the midline area, $\beta = 0.08$, $t = 23.28$, FDR-corrected $p < 0.001$; aperiodic offset was significantly lower in the midline and right areas, $\beta = -0.05$, $t = -10.03$, FDR-corrected $p < 0.001$ and $\beta = -0.05$, $t = -9.18$, FDR-corrected $p < 0.001$; and periodic gamma power was significantly lower the midline and right areas, $\beta = -0.00$, $t = -5.40$, FDR-corrected $p < 0.001$ and $\beta = -0.00$, $t = -4.24$, FDR-corrected $p < 0.001$.

*Group, condition, and sex effects in EEG neural measures*

Between-group comparisons revealed significant differences in all the three EEG neural metrics in response to speech stimuli (see Table 2, Fig. 1C). Autistic youths had reduced aperiodic exponent, $\beta = -0.06$, $t = -3.51$, FDR-corrected $p < 0.001$; reduced aperiodic offset, $\beta = -0.05$, $t = -2.11$, FDR-corrected $p = 0.035$; and elevated periodic gamma power, $\beta = 0.01$, $t = 2.60$, FDR-corrected $p = 0.01$.

Condition, nonverbal IQ, as well as group × condition effects were not significant for all EEG measures (see Table 2).

In male participants in comparison to females, it was observed a significantly higher aperiodic offset, $\beta = 0.10$, $t = 4.00$, FDR-corrected $p < 0.001$ (Table 2, Fig. 1D).

*Age-related changes of EEG neural measures*

Linear age-related changes were revealed in the strength of all neural measures (see Table 2, Fig. 1E): decrease in aperiodic exponent, $\beta = -2.29 \times 10^{-3}$, $t = -10.33$, FDR-corrected $p < 0.001$; decrease in aperiodic offset, $\beta = -4.82 \times 10^{-3}$, $t = -14.00$, FDR-corrected $p < 0.001$; and increase in periodic gamma power, $\beta = 9.45 \times 10^{-5}$,

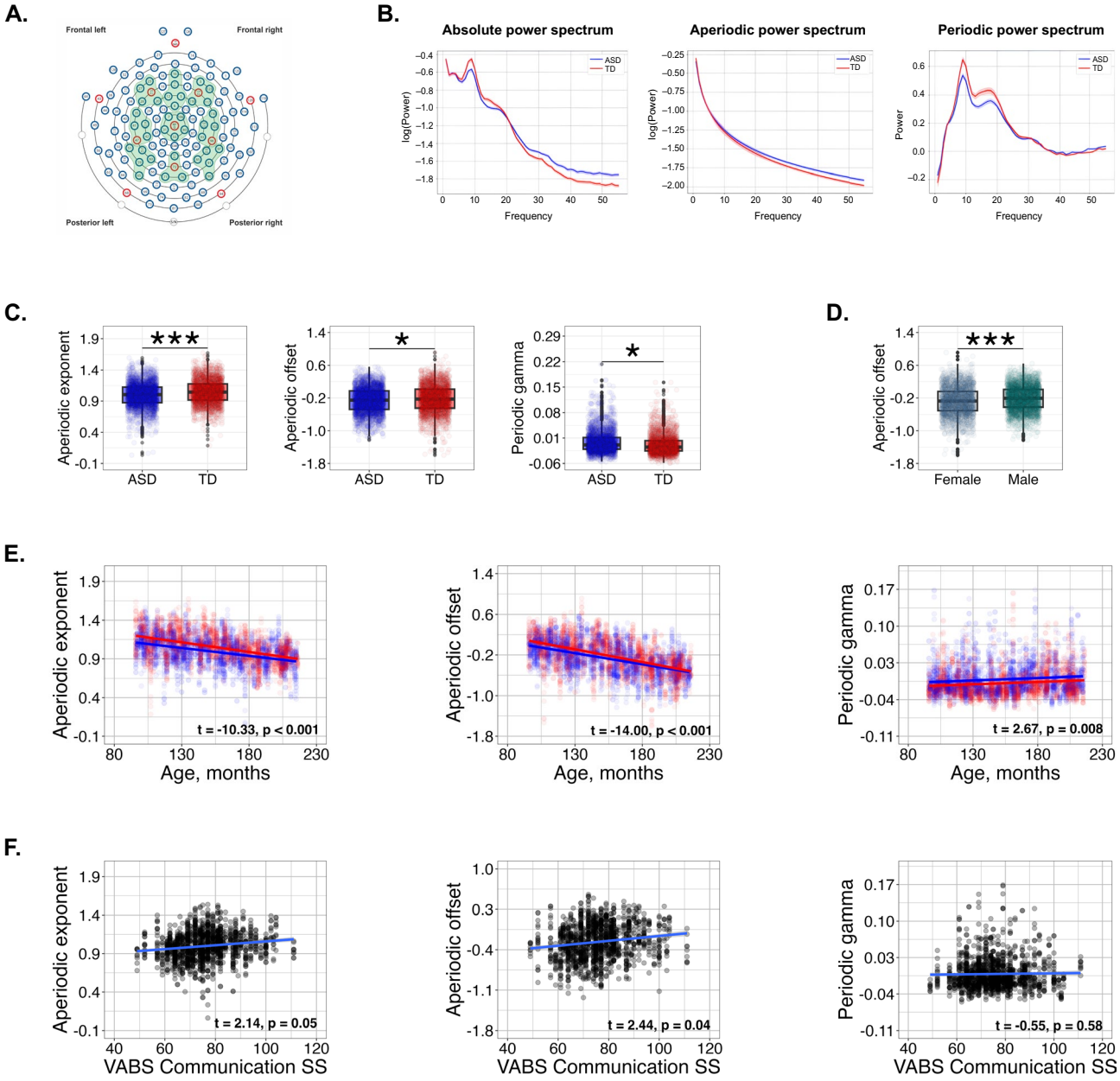


**Fig. 1**. EEG neural measures in response to speech stimuli in youths with and without Autism Spectrum Disorder: (**A**) EEG montage with channels indicated. Channel numbers for regions are frontal left (20, 23, 24, 27, 28), frontal midline (5, 6, 11, 12, 16), frontal right (3, 117, 118, 123, 124), central left (35, 36, 41, 42, 47), central midline (7, 31, 55, 80, 106), central right (93, 98, 103, 104, 110), posterior left (51, 52, 59, 60, 65), posterior midline (62, 71, 72, 76), (9) posterior right (85, 90, 91, 92, 97). (**B**) Absolute, aperiodic and periodic power spectral density (for central left ROI 'familiar' items, for visualization purposes). (**C**) Boxplots representing between-group differences in neural measures. (**D**) Boxplots representing sex differences in aperiodic offset. (**E**) Relationships between EEG neural responses and age. (**F**) Relationships between EEG neural responses and verbal communication. ASD = Autism Spectrum Disorder, TD = typically developing, VABS = Vineland Adaptive Behavior Scales. Significance is labeled with $*p < 0.05$, $**p < 0.01$, $***p < 0.001$; $p$-values are FDR-corrected.

$t = 2.67$, FDR-corrected $p = 0.008$. The mean trajectories demonstrated similar age-related changes in neural measures in both groups of youths.

## The relationships between neural measures and clinical phenotype in youths with ASD

This analysis aimed to investigate whether the EEG measures in response to speech stimuli were associated with language and communication scores in the ASD group when considering a sex effect. Six linear mixed-effects models with EEG metrics as dependent variables, behavioral measures as main effects (CELF Core Language

| Predictors | Aperiodic exponent | | | | Aperiodic offset | | | | Periodic gamma power | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *β* | *SE* | *t* | *p* | *β* | *SE* | *t* | *p* | *β* | SE | *t* | *P* |
| (Intercept) | 1.29 | 0.06 | 20.19 | **< 0.001***** | 0.52 | 0.10 | 5.19 | **< 0.001***** | −0.01 | 0.01 | −0.78 | 0.436 |
| Group, ASD | −0.06 | 0.02 | −3.51 | **< 0.001***** | −0.05 | 0.03 | −2.11 | **0.035*** | 0.01 | 0.00 | 2.60 | **0.01*** |
| Condition, unfamiliar | −0.00 | 0.00 | −0.01 | 0.989 | 0.00 | 0.01 | 0.31 | 0.759 | −0.00 | 0.00 | −0.36 | 0.721 |
| Nonverbal IQ | 0.00 | 0.00 | 1.16 | 0.245 | 0.00 | 0.00 | 0.78 | 0.438 | −0.00 | 0.00 | −1.10 | 0.271 |
| Sex, male | 0.02 | 0.02 | 1.53 | 0.127 | 0.10 | 0.02 | 4.00 | **< 0.001***** | −0.00 | 0.00 | −1.84 | 0.099 |
| Region, central | 0.03 | 0.00 | 7.49 | **< 0.001***** | −0.18 | 0.01 | −34.79 | **< 0.001***** | −0.01 | 0.00 | −8.65 | **< 0.001***** |
| Region, parietal | 0.00 | 0.00 | 0.84 | 0.399 | −0.03 | 0.01 | −5.45 | **< 0.001***** | 0.00 | 0.00 | 1.78 | 0.112 |
| Laterality, midline | 0.08 | 0.00 | 23.28 | **< 0.001***** | −0.05 | 0.01 | −10.03 | **< 0.001***** | −0.00 | 0.00 | −5.40 | **< 0.001***** |
| Laterality, right | 0.00 | 0.00 | 1.04 | 0.298 | −0.05 | 0.01 | −9.18 | **< 0.001***** | −0.00 | 0.00 | −4.24 | **< 0.001***** |
| Age | −0.00 | 0.00 | −10.30 | **< 0.001***** | −0.00 | 0.00 | −13.97 | **< 0.001***** | 0.00 | 0.00 | 2.67 | **0.008**** |
| Group × condition | −0.00 | 0.01 | −0.21 | 0.837 | −0.01 | 0.01 | −0.95 | 0.341 | 0.00 | 0.00 | 0.27 | 0.790 |
| Random effects | | | | | | | | | | | | |
| $\sigma^2$ | 0.01 | | | | 0.02 | | | | 0.00 | | | |
| $\tau_{00\ ID}$ | 0.02 | | | | 0.04 | | | | 0.00 | | | |
| ICC | 0.62 | | | | 0.64 | | | | 0.56 | | | |
| $N_{ID}$ | 306 | | | | 306 | | | | 306 | | | |
| Observations | 5508 | | | | 5508 | | | | 5508 | | | |
| Marginal $R^2$/Conditional $R^2$ | 0.230/0.709 | | | | 0.353/0.767 | | | | 0.045/0.582 | | | |

**Table 2**. The outputs of the models. Significance is labeled with *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ and highlighted in bold. All significant *p*-values are FDR-corrected.

| Predictors | Aperiodic exponent | | | | Aperiodic offset | | | | Periodic gamma power | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *β* | *SE* | *t* | *p* | *β* | *SE* | *t* | *p* | *β* | *SE* | *t* | *p* |
| (Intercept) | 0.75 | 0.12 | 6.35 | **< 0.001***** | −0.74 | 0.20 | −3.75 | **< 0.001***** | 0.01 | 0.02 | 0.53 | 0.598 |
| VABS Communication | 0.00 | 0.00 | 2.14 | **0.05*** | 0.01 | 0.00 | 2.44 | **0.04*** | −0.00 | 0.00 | −0.55 | 0.583 |
| Sex, male | 0.14 | 0.16 | 0.84 | 0.402 | 0.29 | 0.27 | 1.09 | 0.275 | −0.02 | 0.02 | −0.82 | 0.414 |
| VABS Communication × Sex | 0.00 | 0.00 | −0.72 | 0.473 | −0.00 | 0.00 | −0.88 | 0.381 | 0.00 | 0.00 | 0.71 | 0.477 |
| Random effects | | | | | | | | | | | | |
| $\sigma^2$ | 0.01 | | | | 0.02 | | | | 0.00 | | | |
| $\tau_{00\ ID}$ | 0.02 | | | | 0.06 | | | | 0.00 | | | |
| $\tau_{00\ region}$ | 0.00 | | | | 0.01 | | | | 0.00 | | | |
| $\tau_{00\ laterality}$ | 0.00 | | | | 0.00 | | | | 0.00 | | | |
| ICC | 0.70 | | | | 0.76 | | | | 0.55 | | | |
| $N_{ID}$ | 158 | | | | 158 | | | | 158 | | | |
| $N_{region}$ | 3 | | | | 3 | | | | 3 | | | |
| $N_{laterality}$ | 3 | | | | 3 | | | | 3 | | | |
| Observations | 1422 | | | | 1422 | | | | 1422 | | | |
| Marginal $R^2$/Conditional $R^2$ | 0.025/0.708 | | | | 0.036/0.772 | | | | 0.004/0.553 | | | |

**Table 3**. Relationships between EEG neural measures and verbal communication in youths with ASD. The outputs of the models. Significance is labeled with *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ and highlighted in bold. All significant *p*-values are FDR-corrected.

SS, VABS Communication SS) and behavioral measure × sex interaction as fixed effects were fitted. As we did not have a-priory hypothesis on region/laterality, we included it as a random intercepts (with the random effect of participants) in the models. The structure of the models was as follows: *lmer(EEG metric ~ behavioral measure × sex + (1 | ID) + (1 | region) + (1 | laterality), data = data, control = lmerControl(optimizer = "Nelder_Mead"))*. As there was a very high correlation between 'familiar' and 'unfamiliar' items for all neural measures, brain-behavior relationship analysis was performed only for 'unfamiliar' items to avoid redundancy in the analysis: ASD group, aperiodic exponent, $r = 0.91$, $p < 0.001$; aperiodic offset, $r = 0.95$, $p < 0.001$; periodic gamma power, $r = 0.83$, $p < 0.001$.

The output of the models can be seen in Table 3. The results showed that a reduction of the aperiodic components (both exponent and offset) of the EEG spectral power during speech perception was associated with lower verbal communication scores in ASD: aperiodic exponent, $\beta = 0.00$, SE = 0.00, $t = 2.14$, FDR-corrected $p = 0.05$; aperiodic offset, $\beta = 0.01$, SE = 0.00, $t = 2.44$, FDR-corrected $p = 0.04$; no relationship was detected for

| Predictors | Aperiodic exponent | | | | Aperiodic offset | | | | Periodic gamma power | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *β* | *SE* | *t* | *p* | *β* | *SE* | *t* | *p* | *β* | *SE* | *t* | *p* |
| (Intercept) | 1.02 | 0.11 | 9.42 | **<0.001***** | −0.25 | 0.18 | −1.45 | 0.147 | −0.01 | 0.02 | −0.38 | 0.703 |
| CELF | −0.00 | 0.00 | −0.29 | 0.773 | −0.00 | 0.00 | −0.20 | 0.842 | 0.00 | 0.00 | 0.38 | 0.701 |
| Sex, male | −0.02 | 0.14 | −0.16 | 0.876 | −0.01 | 0.22 | −0.05 | 0.962 | −0.01 | 0.02 | −0.53 | 0.598 |
| CELF × Sex | 0.00 | 0.00 | 0.31 | 0.755 | 0.00 | 0.00 | 0.40 | 0.689 | 0.00 | 0.00 | 0.43 | 0.670 |
| Random effects | | | | | | | | | | | | |
| $\sigma^2$ | 0.01 | | | | 0.02 | | | | 0.00 | | | |
| $\tau_{00\ ID}$ | 0.02 | | | | 0.06 | | | | 0.00 | | | |
| $\tau_{00\ region}$ | 0.00 | | | | 0.01 | | | | 0.00 | | | |
| $\tau_{00\ laterality}$ | 0.00 | | | | 0.00 | | | | 0.00 | | | |
| ICC | 0.69 | | | | 0.75 | | | | 0.53 | | | |
| $N_{ID}$ | 118 | | | | 118 | | | | 118 | | | |
| $N_{region}$ | 3 | | | | 3 | | | | 3 | | | |
| $N_{laterality}$ | 3 | | | | 3 | | | | 3 | | | |
| Observations | 1062 | | | | 1062 | | | | 1062 | | | |
| Marginal $R^2$/Conditional $R^2$ | 0.003/0.694 | | | | 0.015/0.759 | | | | 0.008/0.532 | | | |

**Table 4**. Relationships between EEG neural measures and language skills in youths with ASD. The outputs of the models. Significance is labeled with $^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ and highlighted in bold. All significant *p*-values are FDR-corrected.

periodic gamma power, $\beta = -0.00$, SE = 0.00, $t = -0.55$, FDR-corrected $p = 0.58$ (Fig. 1F, Table 3). To test whether the significant relationships could be driven by 'noisiness' in the EEG signal, we fitted a linear model between the number of artifact-free epochs and verbal communication and did not show a significant effect: $\beta = 0.00$, SE = 0.01, $t = 0.20$, $p = 0.83$.

No significant relationships were detected between CELF Core Language SS and neural responses (Table 4). The results did not reveal any interaction with the sex (Table 3, 4).

## Discussion

The present study investigated neural mechanisms of speech perception in a large sample of youths with ASD and relation of these mechanisms to behavioral measures of language and verbal communication. Three potential EEG E/I balance measures have been abstracted from the EEG during speech perception task: aperiodic exponent, aperiodic offset, and periodic gamma power. In general, we revealed altered E/I balance in the ASD group compared to TD controls, and variability in these E/I measures was associated with verbal communication in youths with ASD.

Between-group comparisons of EEG E/I balance measures during speech perception task demonstrated significant differences all three variables: autistic youths had reduced aperiodic exponent and aperiodic offset and elevated periodic gamma power. Although those are separate neural measures abstracted from the EEG signal, all between-group differences had similar functional direction: youths with ASD had neural profiles associated with increased excitation in comparison to the TD group. Although we used updated parameterization methods, our results are consistent with the previous studies[11,12,14,15].

Aperiodic exponent, one of the primary EEG measures of E/I balance, was significantly reduced in the ASD group, and research in humans and animals as well as computational modeling have revealed that this may reflect elevated excitatory activity in the cortex and, subsequently, elevated neural 'noise' in the brain networks[43,45,46]. Remarkably, the ASD group had also a reduced aperiodic offset which is associated with a broadband neural firing and population spiking[47,48]. These two EEG measures are tightly related to each other. At the system level, greater excitation in the neural circuits (reduced exponent) can lead to a reduction of broadband neural activity (thus, to a higher 'noisiness' of the cortex), so that higher E/I ratio is related to greater randomness in the patters of neural firing[70–74]. Likewise, elevated periodic gamma power in youths with ASD reflected the same atypical functioning of neural populations as the aperiodic component of neural spectra with the evidence of increased E/I ratio. This is consistent with previous studies that have reported increased gamma power[21,25] in response to auditory and language stimuli in individuals with ASD.

Although we did not conduct source estimation and abstracted our neural measures from the EEG scalp-level ROIs, we hypothesized that the main generators of neural activity were widely-distributed networks of the primary and secondary temporal regions in the left and right hemispheres given the nature of the task (perception of speech stimuli presented auditorily). This corresponds to previous studies that showed alterations in gamma power and aperiodic component of the spectra in the auditory cortex of autistic individuals[19–21,33,34,36]. All of these alterations observed in the ASD group (e.g., altered E/I balance and broadband reduction of neural firing that may lead to increased random neural 'noise' in the circuit) can affect the precise timing and coordination of neural activity, which is essential for information processing[71,73]. Thus, this alteration in neural activity can impact temporal cortex 'functions' in youths with ASD, including not only language and verbal communication but also more general social communication[75,76].

Indeed, we found relationships between atypical neural activity in response to speech stimuli and behavioral measures in youths with ASD. Reduction of both aperiodic exponent and aperiodic offset was related to lower communication in autistic individuals; this is consistent with the limited number of existing studies on aperiodic neural activity in neurodevelopmental disorders[21,24,25]. It is important to note that there were no associations between neural responses and language scores measures with the CELF. The main difference between VABS Communication and CELF Core Language SS is that they assess different abilities: while CELF measures specifically structural language skills at the levels of grammar/vocabulary, VABS assesses more general adaptive verbal communication, which at the neural level involves wider and less-specialized networks[77]. Presumably, the relationship between verbal communication (not structural language) and aperiodic components of the EEG spectral power could hypothetically be explained by the fact that these neural measures reflect broadband neuronal firing and represent general neural network properties[43,45,46].

Although we showed between-group differences in the neural measures, we identified similar age-related changes in the EEG metrics in both groups of participants. Periodic gamma power in response to speech stimuli increased with age, similar to previous findings[78]. This age-related increase of gamma strength was attributed to developmental changes in E/I balance in local circuits, mostly by maturation of the inhibitory GABA system[78]; this development starts early in neonate brain by switching from a depolarizing to hyperpolarizing action of GABA receptors, i.e., excitatory-to-inhibitory shift of GABA receptors[79]. The decline in aperiodic exponent and offset observed in this study is also consistent with previous reports[45,74,80,81]. These age-related declines may reflect the overall broadband change of background neural firing and may account for a normative redistribution of spectral power from lower to higher levels over development[45]. However, it is still largely unexplored how age-related changes in these two types of neural activity – aperiodic power spectra and gamma-band oscillations – relate to each other. Future studies would benefit from combining these EEG measures with magnetic resonance spectroscopy to identify how concentrations of specific neurotransmitters (GABA vs. glutamate) and their age-related maturation are associated with functioning of EEG-based E/I balance metrics[80].

## Limitations

This work has some limitations that should be highlighted. First, although the study included a large sample size, most individuals with ASD had average or above average verbal skills. To generalize the findings and assess the validity of identified neural markers, it is necessary to include nonverbal or minimally verbal individuals with autism. Second, the study speculated on E/I balance and GABA/glutamate ratio based on non-invasive EEG measures. To note these measures represent indirect, system-level correlates of E/I balance but not direct measurements of synaptic E/I ratios. Future studies would benefit from combining these EEG measures with magnetic resonance spectroscopy to identify how concentration of specific neurotransmitters (GABA vs. glutamate) is related to EEG measures.

## Conclusion

The present study aimed to investigate a set of non-invasive E/I balance measures derived from EEG speech perception task and their relation to clinical phenotype in a large group of youths with ASD. The main finding is that aperiodic component of spectral power was reduced in autistic youths compared to the TD group and this reduction was related to lower verbal communication in autistic youths. The findings hypothetically suggested increased excitation and the reduction of broadband neural firing/spiking activity and, thus, higher 'noisiness' in the cortical systems responsible for speech perception. This E/I imbalance pattern, therefore, may be relevant for monitoring verbal communication in individuals with ASD across development. However, more studies addressing these metrics are needed to clarify their stability, validity, and generalizability.

## Data availability

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request. This also includes codes for statistical analysis and auditory/visual materials from the experimental paradigm.

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## Author contributions

VA, MS, EN, HB, RAB, SYB, MD, ARG, AJ, SJ, JCM, AN, JDVH, KAP, and SJW participated in project conceptualization and writing, including editing and final approval. MS, HB, SJ, AN, and SJW were involved in EEG data acquisition. VA, MS, HB, and SJW completed the analysis. VA and SJW drafted the manuscript.

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## Declarations

### Competing interests

James C. McPartland consults or has consulted with Customer Value Partners, Bridgebio, Determined Health, Apple, Neumarker, and BlackThorn Therapeutics, has received research funding from Janssen Research and Development, serves on the Scientific Advisory Boards of Pastorus and Modern Clinics, and receives royalties from Guilford Press, Lambert, Oxford, and Springer. The remaining authors have no conflict of interest to declare.

### Ethics approval and consent to participate

The study was approved by the Yale Institutional Review Board, the UCLA Office of Human Research Protection Program, Boston Children's Hospital Institutional Review Board, USC Office for the Protection of Research Subjects, and the University of Virginia Institutional Review Board for Health Sciences Research. All procedures performed were in accordance with the Declaration of Helsinki. All minor children provided verbal assent to participate in the study and were informed that they can withdraw from the study at any time during the experiment. A written informed consent form was obtained from a parent of each child participating in the study.

### Additional information

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