Title: Change in aperiodic activity over first year of life is associated with later autism diagnosis and 18-month language development in infants with family history of autism.

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Conflict of Interest
The authors declare that they have no conflict of interest.

Author Contributions

Carol Wilkinson: conceptualization, methodology, software, formal analysis, data curation, writing – original draft, and visualization. Haerin Chung: methodology, software, formal analysis, data curation, writing – original draft, and visualization. Amy Dave: formal analysis, data curation, writing – review & editing. Helen Tager-Flusberg: investigation, resources, writing – review & editing, supervision, project administration, and funding acquisition. Charles A. Nelson: investigation, resources, writing – review & editing, supervision, project administration, and funding acquisition.

Data availability: Consents obtained from human participants at our institution for the studies described prohibit sharing of identifiable and de-identified individual data without a data use agreement in place. Please contact the corresponding author with data requests.

1   **Abstract**

2   Delays in language often co-occur among toddlers diagnosed with autism. Despite the high

3   prevalence of language delays, the neurobiology underlying such language challenges remains

4   unclear. Prior research has shown reduced EEG power across multiple frequency bands in 3-to-

5   6-month-old infants with an autistic sibling, followed by accelerated increases in power with age.

6   Here we apply new methods to decompose the power spectra into aperiodic (broad band neural

7   firing) and periodic (oscillations) activity to explore possible links between aperiodic changes in

8   the first year of life and later language outcomes. Combining EEG data across two longitudinal

9   studies of infants with and without autistic siblings, we assessed whether infants with an

10   elevated familial likelihood (EFL) exhibit altered changes in both periodic and aperiodic EEG

11   activity at 3 and 12 months of age, compared to those with a low likelihood (LL), and whether

12   developmental change in activity is associated with language development. At 3-months of age,

13   we observed that EFL infants have significantly lower aperiodic activity from 6.7-55Hz ($p<0.05$).

14   However, change in aperiodic activity from 3 to 12 months was significantly increased in infants

15   with a later diagnosis of autism, compared to EFL infants without an autism diagnosis. In

16   addition, greater increases in aperiodic offset and slope from 3-to12-months were associated

17   with worse language development measured at 18 months. Findings suggest that early age-

18   dependent changes in EEG aperiodic power may serve as potential indicators of autism and

19   language development in infants with family history of autism.

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1 **Introduction**
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3 Early delays in language have been shown to have long lasting impacts on behavioral
4 functioning, academic success, vocational outcomes and quality of life(Law et al., 2009;
5 Snowling et al., 2006; Young et al., 2002). Language delays are highly prevalent in young
6 children diagnosed with autism. At the time of diagnosis, as many as 40% of autistic toddlers
7 have co-occurring language delays(Reetzke et al., 2022), and the degree of later gains in
8 language development is variable, with 30% of autistic children remaining minimally
9 verbal(Tager-Flusberg & Kasari, 2013). Notably, younger siblings of autistic children are at
10 elevated likelihood for *both* autism and language delays, suggesting shared underlying biology
11 (Marrus et al., 2018). Despite the high prevalence of language impairments in autism, our
12 understanding of the neurobiology underlying impairments is limited. Pinpointing neural markers
13 that are specifically associated with language delays in infant-siblings may shed light on neural
14 mechanisms of early language development and facilitate earlier detection and intervention.
15
16 Electroencephalography (EEG), which measures network level brain activity at the scalp, has
17 proven to be a promising tool for identifying early neural markers of altered development in
18 infancy. In a number of studies resting (baseline) EEG data have been collected longitudinally in
19 infants with an elevated familial likelihood of developing autism (EFL, infants with an older
20 sibling with autism). These studies have observed a global reduction in frontal EEG power
21 across multiple frequency bands at early ages (3-6 months), as well as differences in EEG
22 trajectories from 6 to 24 months in EFL infants regardless of later autism diagnosis (Huberty et
23 al., 2021; Levin et al., 2017; Tierney et al., 2012; Wilkinson et al., 2020). While EEG power is
24 reduced in early infancy in EFL infants, differences are less pronounced at 12 and 24 months,
25 and studies of older autistic children have observed either no differences, or *increases* in
26 absolute gamma power(Arutiunian et al., 2024; Huberty et al., 2021; Mukerji et al., 2024; Rojas
27 & Wilson, 2014; van Diessen et al., 2015). This suggests that *change* in power spectral features
28 early in development may be an important biomarker for autism or other developmental delays.
29
30 Studies of EEG power and language development have had mixed findings depending on age
31 and likelihood group. In infancy, lower frequencies (delta, theta, and alpha) have been
32 associated with language development in both low likelihood infants (LL, infants without an older
33 sibling with autism) and EFL infants(Levin et al., 2017; Pierce et al., 2021). In contrast, in
34 toddlers (16-36 months) frontal gamma power has been associated with language development
35 (Benasich et al., 2008; Gou et al., 2011; Wilkinson et al., 2019). Interestingly, the direction of
36 association between brain activity and language often differs between EFL versus LL toddlers.
37 Gamma power is positively associated with language in LL toddlers, yet negatively associated in

1 EFL toddlers, suggesting possible differences in factors impacting language development

2 between groups (Lombardo et al., 2015; Swanson et al., 2017; Wilkinson et al., 2019). The

3 increased gamma power in autistic children has been hypothesized to reflect imbalance in

4 neural excitation/inhibition (E/I)(Brunel & Wang, 2003; Buzsáki & Wang, 2012; Economo &

5 White, 2012). Thus, while higher gamma power in typically developing toddlers and

6 preschoolers may be a beneficial indicator of brain activity for language development, in autistic

7 children it may instead serve as a marker of E/I imbalance impeding processes important for

8 language and cognitive development.

9

10 While past EEG studies in autism have largely focused on either absolute or relative measures

11 of power, newer methods utilize parametrization of the spectrum into aperiodic and periodic

12 components to provide a more accurate estimate of oscillatory and non-oscillatory

13 activity(Donoghue et al., 2020; Ostlund et al., 2022). The aperiodic signal is defined by the 1/f

14 power law distribution that underlies the absolute power spectra(Gao et al., 2017; Manning et

15 al., 2009; Miller et al., 2009), and can be described by the offset and slope (defined as the χ in

16 the $1/f^{\chi}$ formula). The aperiodic offset is thought to reflect broad band, non-oscillatory neuronal

17 firing(Manning et al., 2009; Miller, 2010), and, growing evidence suggests that the slope of the

18 aperiodic signal may, in part, reflect the excitatory-inhibitory (E/I) balance of the brain, with a

19 flatter slope associated with increased excitation over inhibition(Chini et al., 2022; Gao et al.,

20 2017; McKeon et al., 2024). Together, these components of aperiodic activity may inherently

21 reflect broader neural dynamics such as cortical excitability or developmental maturation. In

22 addition, modulation in aperiodic activity, such as flatter slopes, can influence absolute power in

23 higher frequencies, like gamma. Thus, previous associations between absolute gamma power

24 and language development could instead reflect associations in aperiodic activity and language

25 development. By accounting for individual differences in aperiodic activity, parametrization of

26 the absolute spectrum also provides more accurate measurement of peak frequency and

27 amplitude of oscillatory, or periodic components of the spectra(Buzsáki et al., 2013; Donoghue

28 et al., 2020; Ostlund et al., 2022). Notably, both aperiodic and periodic components change with

29 development, with dynamic changes occurring during the first year after birth(Cellier et al., 2021;

30 Hill et al., 2022; Rayson et al., 2023; Rico-Picó et al., 2023; Wilkinson et al., 2024).

31

32 In this study, we aimed to investigate developmental changes in aperiodic and periodic

33 components in EFL vs LL infants and assess how alterations may be associated with later

34 autism and language outcomes. Leveraging data collected across two infant-sibling studies with

35 3- and 12- month time points, we first assessed whether there are differences in frontal

36 aperiodic and periodic power in EEG data collected in LL and EFL infants at 3 and 12 months of

1   age. Next, we determined whether EFL-ASD infants exhibit distinctive patterns of change in

2   EEG aperiodic or periodic power from 3 to 12 months of age. Finally, we assessed whether

3   developmental changes in EEG features are associated with language development.

4

5   **Methods**

6

7   **Participants:** This analysis includes infants who were recruited as part of two consecutive

8   studies in the same lab: The Infant Sibling Project (IRB-X06-08-0374), and the Infant Screening

9   Project (IRB-P00018377). Institutional review board (IRB) approval was obtained from Boston

10   Children's Hospital and IRB protocol numbers are provided for each study. Secondary analysis

11   for this paper was also approved (IRB-P00037531). Written informed consent was obtained

12   from a parent or guardian prior to each child's participation in the study. Both studies were

13   prospective, longitudinal studies, enrolling infants with and without first degree family history of

14   ASD starting as early as 3-months of age. However, both studies initially started recruiting at

15   later ages, and added the 3-month enrollment option part way through enrollment. The Infant

16   Screening Project also recruited a group of infants with elevated social communication concerns

17   at 12 months of age, but because of the small sample size, they were excluded from this

18   analysis. Exclusion criteria included gestational age less then 36 weeks, history of prenatal or

19   postnatal medical or neurological problems, as well as identified genetic disorders. Sample

20   characteristics are shown in Table 1.

Table 1: Sample characteristics

		3-month		12-month	
		LL N = 59 (11, 48)	EFL N = 51 (26, 25)	LL N = 153 (62, 91)	EFL N = 137 (77, 60)
Sex		36 M, 23 F	31 M, 29 F	81 M, 72 F	76 M, 61 F
Ethnicity, *n (%)*					
	Not Hispanic or Latino	57 (97)	42 (82)	147 (96)	126 (92)
	Hispanic or Latino	2 (3)	8 (16)	4 (3)	11 (8)
	Not Answered	0 (0)	1 (2)	2 (1)	0 (0)
Race, *n (%)*					
	White	50 (85)	40 (78)	123 (80)	122 (89)
	Black or African American	1 (2)	0 (0)	4 (3)	1 (1)
	Asian	1 (2)	2 (4)	6 (4)	8 (6)
	Mixed Race	7 (12)	8 (16)	18 (12)	6 (4)
	Not answered	0 (0)	1 (2)	2 (1)	0 (0)
Household income, *% (n)*					
	<$35,000	1 (2)	3 (6)	7 (4.5)	3 (2)
	$35,000 - $75,000	2 (3)	3(6)	7 (4.5)	15 (11)
	>$75,000	54 (92)	39 (76)	127 (83)	99 (72)
	Not answered	2(3)	6 (12)	12 (8)	19 (14)
EEG quality metrics					
	Number of Segments	103.2 ± 39.1	97.8 ± 37.4	89.3 ± 39.0	93 ± 53.1
	Percent Good Channels	92.2 ± 0.05	92.8 ± 0.05	92.4 ± 0.05	92.4 ± 0.04
	Percent ICs Rejected	0.34 ± 0.10	0.37 ± 0.13	0.39 ± 0.10	0.37 ± 0.12
	Mean Artifact Probability of Kept ICs.	0.15 ± 0.05	0.14± 0.04	0.12 ± 0.05	0.12 ± 0.05

Developmental and autism assessments: Participants in both studies were seen longitudinally for developmental assessments using the Mullen Scales of Early Learning (MSEL). As the Infant Screening Project spanned the COVID-19 pandemic, in person assessments for a number of participants at 24 and 36 months was not possible and remote evaluations took place. Therefore, to maximize the sample size of analyses related to language development, the MSEL Verbal Developmental Quotient at 18 months (available for all in person) was used instead of later ages. For the Infant Sibling Study, ASD outcomes were determined using the Autism Diagnostic Observation Schedule (ADOS) and parent-child interaction administered at 24 and/or 36 months of age. During the COVID-19 pandemic, remote autism evaluations for the Infant Screening Project included the Brief Observation of Symptoms of Autism (BOSA), parent-child interaction, and Vineland Adaptive Behavioral Scales – Third Edition, Parent Interview Form (Vineland-3). In both studies, for toddlers meeting criteria on the ADOS/BOSA or coming within three points of cutoffs, a licensed clinical psychologist reviewed scores and video recordings of concurrent and previous behavioral assessments and using DSM-5 criteria provided a best estimate of clinical judgement.

EEG data collection: In both studies, resting-state, non-task-related, EEG data were collected while the infant was held by their seated caregiver in a dimly lit, sound attenuated room with low-electrical signal background. In the Infant Sibling Study, a research assistant ensured the infant remained calm by blowing bubbles and/or showing toys. Continuous EEG data was collected using 64-channel Geodesic Sensor (<10% of data) or a 128-channel Hydrocel Geodesic Sensor Nets (Electrical Geodesics, Inc., Eugene, OR), connected to either a NetAmps 200 or 300 amplifier (Electrical Geodesic Inc.) and sampled at either 250 or 500Hz. In the Infant Screening Project study, a video of abstract moving objects was shown and EEG data was collected with the 128-channel Hydrocel Geodesic Sensor Net, connected to a NetAmps 300 amplifier and sampled at 500Hz. For both studies, data were referenced online to a single vertex electrode (Cz). Electrooculographic electrodes were removed to improve the child's comfort.

EEG pre-processing and rejection criteria:

1 Raw Netstation (Electrical Geodesics, Inc) files were exported to MATLAB (version R2017a) for

2 preprocessing and absolute power calculations using the Batch Automated Processing Platform

3 (BEAPP(Levin et al., 2018)) with integrated Harvard Automated Preprocessing Pipeline for EEG

4

5 (HAPPE(Gabard-Durnam et al., 2018)). For each EEG, a 1Hz high-pass and 100Hz low-pass

6 filter were applied, data sampled at 500Hz were resampled to 250Hz, and then run through the

7 HAPPE module consisting of 60Hz line noise removal, bad channel rejection, and artifact

8 removal using combined wavelet-enhanced independent component analysis (ICA) and Multiple

9 Artifact Rejection Algorithm (MARA[5,6]). The following channels, in addition to the 10-20

10 electrodes, were used for MARA: 64-channel net – 16, 9, 8, 3, 58, 57, 21, 25, 18, 30, 43, 50, 53,

11 32, 33, 38, 41, 45; and 128-channel net - 28, 19, 4, 117, 13, 112, 41, 47, 37, 55, 87, 103, 98, 65,

12 67, 77, 90, 75. After artifact removal, channels removed during bad channel rejection were then

13 interpolated, data were referenced to the average reference, detrended to the signal mean, and

14 segmented into 2-second segments. Any segments with retained artifact were rejected using

15 HAPPE's amplitude and joint probability criteria. EEG recordings were rejected using the

16 following HAPPE data quality measures: Fewer than 20 segments (40 seconds of total EEG),

17 percent good channels < 80%, percent independent components rejected >80%, mean artifact

18 probability of components kept > 0.3, and percent variance retained < 25%. Table 1 shows

19 quality metrics across LL and EFL groups. There were no significant differences between

20 groups.

21

22 **EEG Power Spectra Analysis:**

23

24 For each 2-second segment, the power spectral density at each electrode was calculated in the

25 BEAPP Power Spectral Density (PSD) module using a multitaper spectral analysis(Babadi &

26 Brown, 2014) and three orthogonal tapers. For each electrode, the PSD was averaged across

27 segments, and then averaged across all frontal electrodes [4, 11, 19, 24, 28, 117, 124]. The

28 PSD was then further analyzed using a modified version of SpecParam v1.0.0(Donoghue et al.,

29 2020) (https://github.com/fooof-tools/fooof; in Python v3.6.8; modifications described in

30 (Wilkinson et al., 2024) with available code at osf.io/u3gp4. The SpecParam model was used in

31 the fixed mode (no spectral knee) evaluating spectra between 2-55Hz, with *peak_width_limits*

32 set to [0.5, 18.0], *max_n_peaks* = 7, and *peak_threshold* = 2). Mean R^2 for the full sample using

33 this modified version of SpecParam was 0.997 (STD 0.009; range 0.875-0.9998).

34

35 Here we define aperiodic offset as aperiodic power at 2.5Hz, as there are high levels of error in

36 the SpecParam estimates at frequencies below 2.5Hz. Aperiodic slope, defined by χ in the 1/fχ

37 model fit, is provided by SpecParam. Total aperiodic activity is defined as the integral of the

1 aperiodic spectra from 2.5-55Hz. To characterize the periodic power spectra, the SpecParam

2 estimated aperiodic signal was subtracted from the absolute power spectrum. Periodic gamma

3 power was calculated using the integral of the periodic spectra from 30-45Hz.

4

5 **Statistical Analyses:**

6 Group differences in the absolute, aperiodic, and periodic power spectra were assessed using a

7 non-parametric clustering method controlling for multiple comparisons using Monte Carlo

8 estimation with 10,000 permutations(Maris & Oostenveld, 2007), employed with MNE-

9 Python(Gramfort et al., 2014). Group differences in EEG measures were statistically compared

10 using Mann Whitney U, or ANCOVA with sex included as covariate.

11

12 Study data were collected and managed using REDCap electronic data capture tools hosted at

13 Boston Children's Hosptial(Harris et al., 2009, 2019).

14
15 **Results**
16
17 Table 1 shows sample characteristics for participants providing EEG data either 3-months or 12-

18 months of age. Fewer EEGs were available at 3 months of age for both studies, as the early

19 time point was not included until part-way through each study. Forty-one LL-NoASD, 16 EFL-

20 ASD, and 16 EFL-NoASD infants had EEG data at both 3 and 12 months.

21

22 *Power spectra differences at 3 months of age.*

23

24 Frontal absolute, aperiodic, and periodic power spectra for LL and EFL infants at 3 months are

25 shown (Figure 1A). We used a non-parametric clustering method, controlling for multiple

26 comparisons, to assess for group differences in the power spectra. Within the 3-month absolute

27 power spectra a significant cluster was identified in the gamma range (43-55Hz, $p<0.05$). Within

28 the 3-month aperiodic component, a significant cluster spanning alpha through gamma

29 frequencies was identified (6.7-55Hz, $p < 0.05$). No significant clusters were identified in the 3-

30 month periodic spectra. The combination of these findings suggest that differences observed in

31 absolute gamma power are largely driven by differences in aperiodic activity. Statistically

32 significant group differences were observed for both absolute gamma power and total aperiodic

33 activity, with the EFL group exhibiting reduced activity (Mann Whitney U; Absolute Gamma: U =

34 1850.0, $p < 0.05$; Total Aperiodic Activity: U = 1962.0, $p < 0.01$; Figure 1B, C, D) compared to

35 the LL group. No significant difference was observed for periodic gamma power, aperiodic slope

36 or offset.

Figure 1
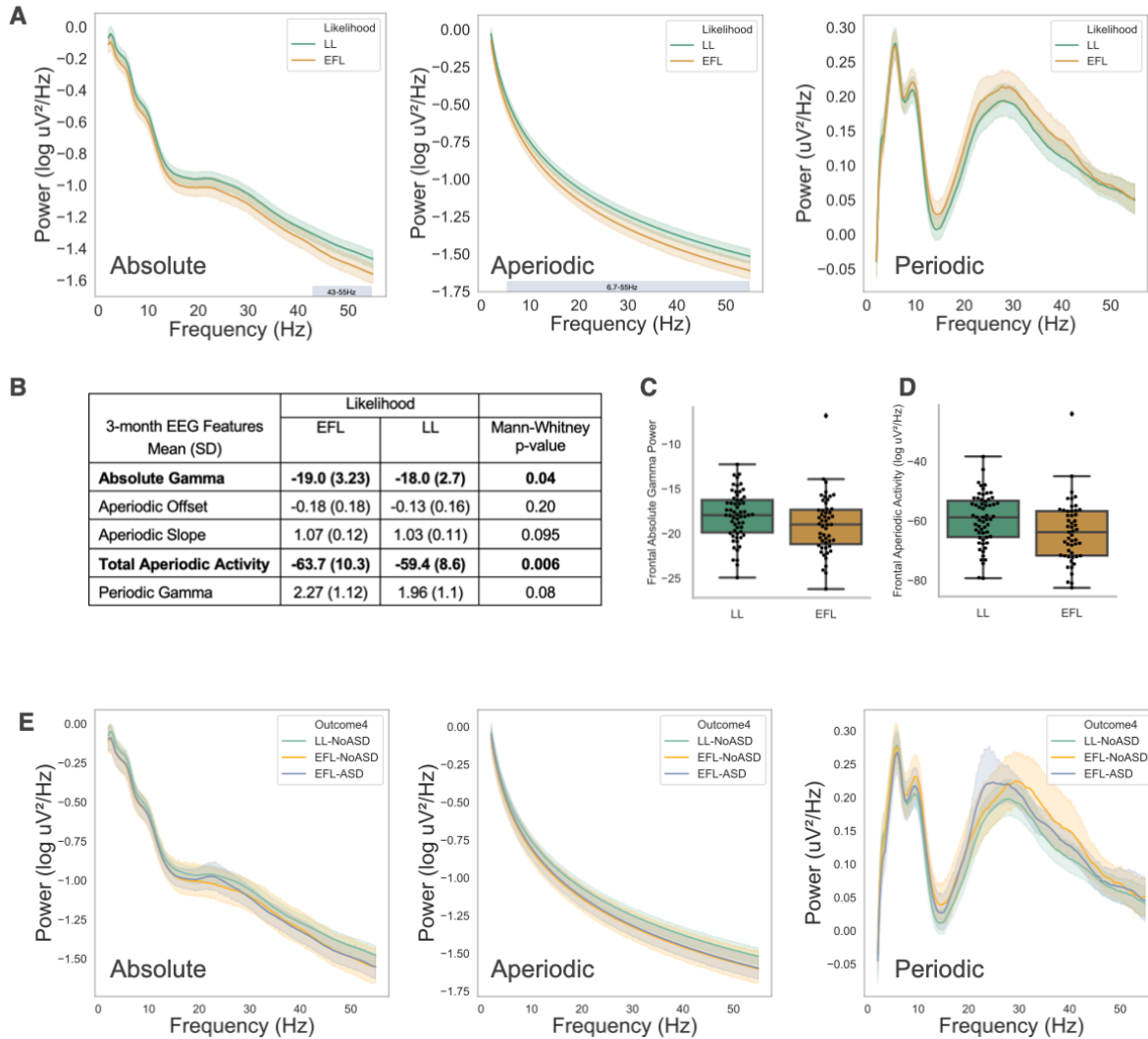
Figure 1. (A) Absolute, Aperiodic, and Periodic spectra at 3 months by Likelihood group. Colored shading around spectra represents 95% confidence intervals. Identified statistically significant clusters are shown as horizontal bars defined by the cluster's frequency band. **(B)** Mean comparison of EEG measures by Likelihood group. **(C, D)** Box plot of group differences in absolute gamma power and total aperiodic activity. **(E)** Absolute, aperiodic, and periodic spectra at 3 months by Outcome group. Shading around spectra represents 95% confidence intervals.

Next, we assessed whether differences in aperiodic activity at 3 months were driven by those infants later diagnosed with autism. Power spectra from EEG collected at 3 months of age are shown in Figure 1E based on later autism outcome (LL-noASD n = 48, EFL-noASD n = 23, EFL-ASD n = 22). No significant differences were observed between outcome groups on non-parametric clustering methods across the spectrum and no significant differences were observed for specific aperiodic or gamma power measures (ANCOVA with sex included as covariate, Supplemental Table 1)
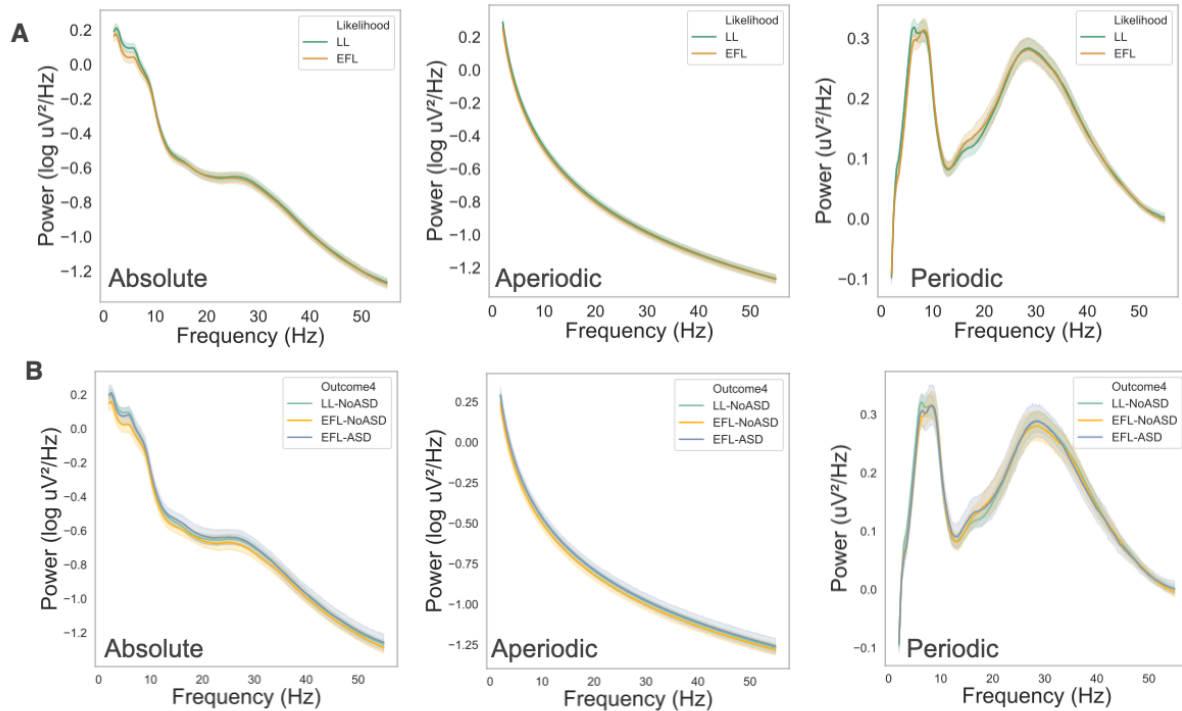
1   *Power spectra differences at 12 months.*

2

3   Power spectra for LL and EFL infants at 12 months are shown in Figure 2A, and 12-month

4   spectra group by later autism outcomes are shown in Figure 2B. Unlike at 3 months, there were

5   no significant differences between groups on any measures.

Figure 2

Figure 2. Absolute, Aperiodic, and Periodic spectra at 12 months groups by (A) Likelihood and (B) Outcome groups. Shading represent 95% confidence intervals.

6
7   *Change in EEG aperiodic and periodic activity from 3 to 12 months.*

8

9   Our 3- and 12-month-old findings suggests that EFL infants with significantly lower aperiodic

10   activity early in infancy have greater increases aperiodic activity between 3 and 12 months,

11   leading to similar EEG aperiodic spectra at 12 months. This finding might reflect greater

12   developmental increases in aperiodic activity exhibited by the EFL group, regardless of ASD

13   outcome. Alternatively, it could be primarily driven by more pronounced changes in aperiodic

14   activity among the EFL-ASD infants. To test this, we limited our analysis to those infants with

15   both 3- and 12-month-old data, and confirmed that outcome groups were similar in the number

16   of days between visits. 41 LL-NoASD, 16 EFL-ASD, and 16 EFL-NoASD infants had EEG data

17   at both 3 and 12 months.

18

1   Mixed-effects linear regression models were performed to test whether change in aperiodic
2   features (slope, offset, total aperiodic activity) were different between outcome groups (LL-
3   noASD, EFL-noASD, EFL-ASD). Age (months) was treated as a within-subjects factor and
4   outcome groups were included as between-subjects factor. Sex was included as a covariate in
5   analyses as there were differences in sex distribution between outcome groups (with the EFL-
6   ASD group having higher proportion of males). A priori pairwise comparisons revealed
7   significant outcome group differences in the change in EEG measures from 3 and 12 months
8   (Table 2 and Figure 3; see Supplementary Table 2 for full model outputs). Specifically, EFL-
9   ASD infants exhibited greater change in aperiodic total activity when compared to LL-NoASD
10 infants, and greater change in aperiodic offset when compared to both EFL-noASD and LL-
11 noASD infants. Given prior literature on absolute gamma activity in autism, we also tested
12 whether outcome groups differed in change of absolute gamma power. No significant
13 differences were observed.

Figure 3
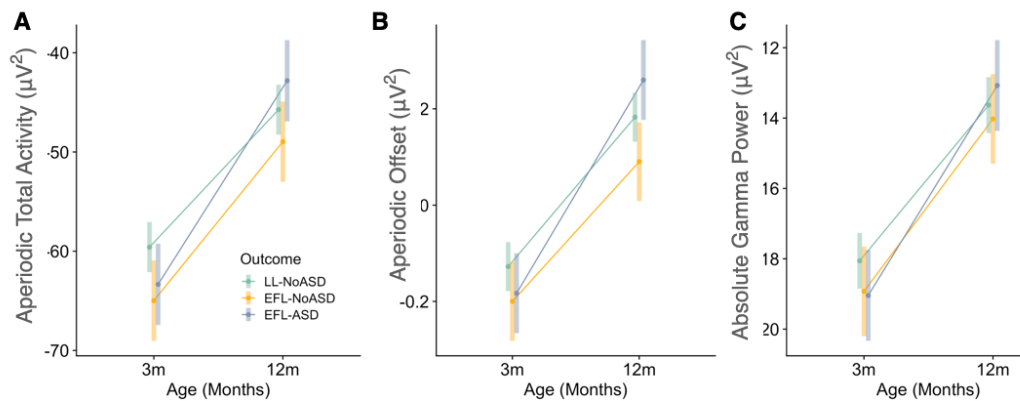
Figure 3. Change in EEG measures from 3 to 12 months. (A) Aperiodic Total Activity, (B) Aperiodic Offset, and (C) Absolute Gamma Power.

|	Outcome Comparison	estimate	SE	df	t.ratio	p.value
Aperiodic Total Activity	(EFL-ASD) - (EFL-NoASD)	-4.50	3.93	70	-1.147	0.255
	(EFL-ASD) - (LL-NoASD)	-6.67	3.27	70	-2.037	**0.046**
	(EFL-NoASD) - (LL-NoASD)	-2.17	3.27	70	-0.661	0.511
Aperiodic Offset	**(EFL-ASD) - (EFL-NoASD)**	-0.153	0.077	70	-1.997	**0.049**
	(EFL-ASD) - (LL-NoASD)	-0.133	0.064	70	-2.079	**0.041**
	(EFL-NoASD) - (LL-NoASD)	0.020	0.064	70	0.316	0.753
Absolute Gamma Power	(EFL-ASD) - (EFL-NoASD)	-1.065	1.165	70	-0.914	0.364
	(EFL-ASD) - (LL-NoASD)	-1.539	0.971	70	-1.585	0.118
	(EFL-NoASD) - (LL-NoASD)	-0.474	0.971	70	-0.488	0.627

Table 2: A priori pairwise comparisons of EEG changes between outcome groups (3-to-12 months)

1   *Exploratory analysis of EEG features associated with language development.*
2

3   Language and cognitive impairments often co-occur in autistic toddlers, and therefore EEG

4   findings associated with autism outcomes may not be specific to the diagnosis, but rather

5   reflective of developmental delays. In addition, while we have previously found that increased

6   absolute gamma power measured at both 18 and 24 months is associated with reduced

7   language scores in EFL infants, our current analyses, now parametrizing the power spectra into

8   aperiodic and periodic components, suggest that associations between absolute gamma power

9   and language in autism may instead be driven by alterations in underlying aperiodic activity.

10   Given our above observation that increased change in aperiodic offset and total aperiodic

11   activity are associated later ASD diagnosis, we next explored whether change in aperiodic

12   activity is associated with future language impairment across the EFL group (n = 25) when

13   controlling for either autism outcome or nonverbal cognitive ability. Linear regressions were

14   performed with 18-month verbal developmental quotients as the dependent variable,

15   developmental change from 3-to 12-months in aperiodic measures as the independent variable

16   (Model 1), and either 18m nonverbal developmental quotients (Model 2) or ASD diagnosis

17   included as a covariate (Model 3). Results are shown in Table 3. Increased changes in

18   aperiodic offset and slope were significantly associated with reduced 18 month verbal

19   developmental quotients (Figure 4). Notably, associations remained significant when accounting

20   for differences in 18-month nonverbal developmental quotient or for autism diagnosis. No

21   significant associations were observed for aperiodic total activity or absolute gamma power.

Figure 4
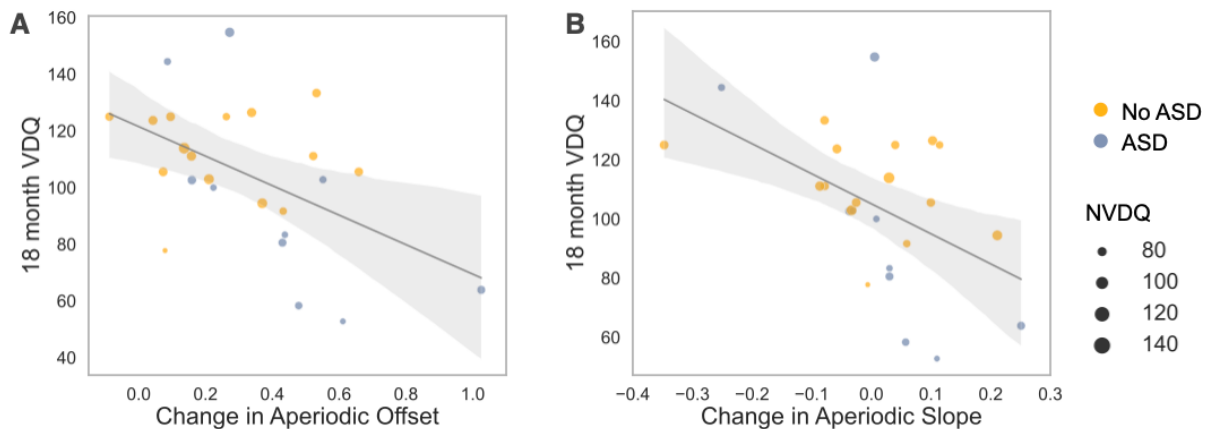
Figure 4. Regression plots from Model 1 comparing (A) change in aperiodic offset and 18-month MSEL verbal developmental quotient (VDQ) and (B) change in aperiodic slope and 18-month MSEL VDQ. Size of dots indicated individual's MSEL nonverbal developmental quotient (NVDQ). Color represents autism outcome.

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Table 3: Linear regressions assessing associations between developmental change EEG features and 18-month MSEL VDQ

	Model 1	Model 2	Model 3
Adjusted R^2	0.223	0.325	
Variables [B coefficient (SE)]			
Δ Aperiodic Offset	**-52.1 (18.5)***	**-44.4 (17.7)***	**-45.6 (19.8)***
18m NVDQ		**0.71 (0.34)***	
ASD			-9.3 (9.8)
Adjusted R^2	0.246	0.385	0.272
Variables [B coefficient (SE)]			
Δ Aperiodic Slope	**-101.6 (37.1)***	**-98.9 (32.8)****	**-96.4 (35.8)***
18m NVDQ		**0.85 (0.31)***	
ASD			-15.1 (8.9)
Adjusted R^2	0.058	0.181	0.078
Variables [B coefficient (SE)]			
Δ Aperiodic Total Activity	-0.71 (0.45)	-0.51 (0.43)	-0.50 (0.48)
18m NVDQ		0.78 (0.37)	
ASD			-13.1 (10.6)
Adjusted R^2	0.044	0.164	0.057
Variables [B coefficient (SE)]			
Δ Absolute Gamma Power	-2.6 (1.8)	-1.66 (1.76)	-1.60 (2.0)
18m NVDQ		0.78 (0.38)	
ASD			-13.1 (11.3)

* $p<0.05$; ** $p<0.10$, ***$p<0.005$

Discussion

Leveraging two longitudinal data sets with a prospective infant-sibling design, we compared early developmental changes in EEG power spectra from 3 to 12 months between infants with low and elevated likelihood based on family history of autism. To do this, we both used traditional assessment of absolute EEG power, but also separately analyzed aperiodic and periodic activity. We observe two major findings: First we observe early differences in aperiodic activity in infants with family history of autism regardless of later autism outcome. Second, we observe that greater developmental increases in aperiodic activity are associated with both later autism diagnosis and reduced language development in EFL infants.

Early differences in EEG activity at 3 months associated with autism family history.

At 3-months of age, EFL infants exhibited significantly reduced absolute gamma power as well as overall reduced aperiodic activity compared to LL infants. Interestingly, these differences were not driven by those infants with later diagnoses of autism, suggesting that while early differences in EEG activity may be driven by genetic/familial factors associated with autism,

1   reduced 3-month aperiodic activity alone is not predictive of later diagnosis. We, and others

2   have previously reported reduced EEG power in EFL infants early in infancy(Huberty et al.,

3   2021; Levin et al., 2017; Tierney et al., 2012; Wilkinson et al., 2020), but until now, studies have

4   not examined whether this is due differences in aperiodic or periodic activity. Our observations

5   suggest that prior reported differences in absolute power, could be largely driven by reduced

6   aperiodic activity in EFL infants.

7

8   Aperiodic activity represents non-oscillatory, broad band neuronal spiking activity(Manning et

9   al., 2009). The most dramatic increased in aperiodic activity occur in the first months after birth,

10   likely reflecting increases in neuronal number, neuronal connections and

11   synaptogenesis(Wilkinson et al., 2024). In addition, growing evidence suggests that aperiodic

12   slope is an indirect measurement of network excitatory and inhibitory (E/I) balance(Chini et al.,

13   2022; Gao et al., 2017; McKeon et al., 2024). While it is hypothesized that alterations in E/I

14   balance play a role in the development of autism and other neurodevelopmental disorders, we

15   did not observe significant differences in aperiodic slope based on familial likelihood or later

16   outcomes. Thus, reductions in aperiodic activity observed in EFL infants may reflect early

17   hypoconnectivity and/or delays in synaptogenesis.

18

19   *Change in aperiodic EEG activity associated with later autism diagnosis.*

20

21   Consistent with previous studies of absolute EEG power, at 12-months, we observed no

22   significant group differences (likelihood or outcome group) for either aperiodic or periodic

23   activity. However, group differences were observed when assessing the *change* in aperiodic

24   activity from 3- to 12-months of age. Here we found infants with later ASD diagnoses exhibited

25   *greater* developmental increases in aperiodic total power and offset. These findings are aligned

26   with longitudinal MRI studies of infant-sibling design which find that EFL-ASD infants exhibit

27   increased growth rates in cortical surface area during the first year of life and increased total

28   brain volume growth rates in the second year compared to both EFL-noASD and LL

29   infants(Hazlett et al., 2017).

30

31   Again, changes in aperiodic slope were not different between likelihood or outcome groups.

32   However, it is important to note that these findings do not directly conflict with prior literature

33   finding altered E/I balance in children or adults with autism. Our observations are specific to

34   EEG activity across the first year after birth, when networks are being established and some of

35   the most significant changes in aperiodic and periodic activity occur. It is still unclear whether

36   aperiodic slope measured during the first year of life is an accurate proxy for E/I balance.

1

2 *Greater early developmental change in aperiodic activity associated with reduced language*

3 *development.*

4

5 Finally, exploratory analysis found that for EFL infants, increased change in aperiodic offset and

6 slope from 3-to-12months was associated with lower language ability at 18 months. Importantly

7 this association remained significant after accounting for either differences in nonverbal

8 development or autism diagnosis.

9

10 This is the first study to examine change in EEG activity between these specific ages. Levin et

11 al 2017, focusing on 3-month EEG data (using an overlapping data set to this paper), showed

12 that increased high alpha power at 3 months was associated with better 12-month expressive

13 language skills. Huberty et al. (2023), specifically investigated whether change in absolute EEG

14 spectral power from 6 to 36 months is associated with language development in infant-siblings.

15 In Huberty et al., significant associations were limited to 6-month alpha power and *concurrent*

16 language ability, with increased 6-month alpha power associated with better expressive

17 language skills at 6-months, while controlling for concurrent nonverbal skills. In contrast to our

18 findings, there were no significant associations between change in EEG power from 6 to 36

19 months and later language abilities. Differences in both the age range analyzed, and EEG

20 processing methods (absolute vs aperiodic activity) may explain the difference in findings.

21

22 A recent study by Piazza et al 2023(Piazza et al., 2023), evaluated EEG data collected at 6 and

23 12 month of age in infants with either elevated likelihood for autism or language impairment.

24 They observed reduced absolute power in low frequency bands (delta, theta, low alpha) at both

25 ages in infants with elevated autism likelihood compared to those with low autism likelihood.

26 Evaluation based on later outcomes observed that infants with later autism diagnosis also

27 showed reduced delta and theta power at 6 months, and in theta at 12 months, and infants with

28 later language delays showed increased high frequency power at 12 months of age. However,

29 authors did not specifically assess *within subject change* in EEG power, and it is possible that

30 the observed differences at 6 and 12 months, reflect greater changes in aperiodic activity in

31 those infants with later autism diagnoses.

32

33 Why might greater changes in aperiodic offset and slope be associated with reduced language

34 development in infants with family history of autism? EFL infants as a group also exhibited

35 significantly lower aperiodic activity at 3 months compared to LL infants. While it is still unclear

36 what neurobiological mechanisms underly this reduced activity, it is possible that EFL infants

1   who overcompensate for these early differences have altered processing of sensory information

2   critical for language development. For example, infants undergoing a rapid shift in connectivity

3   or synaptogenesis in response to underlying aberrant circuitry may not make appropriate

4   connections in response to the sensory environment. In addition, early alterations in aperiodic

5   activity maybe impact the development of neural circuits important for rhythmic activity that play

6   a critical role in language processing and therefore language acquisition.

7

8   *Limitations*

9

10   While we combined data from two longitudinal studies, increasing our 3- and 12-month samples

11   sizes, fewer than 73 infants provided data at both time points. Larger prospective longitudinal

12   studies starting early in infancy are required to replicate our findings. Our sample was also

13   largely comprised of infants from white families with higher levels of income, reducing

14   generalizability. We have previously observed that lower household income is also associated

15   with reduced EEG absolute power at 2-3 months of age, with greater increases in power from 3

16   to 9 months(Wilkinson et al., 2023). Larger datasets with greater variability in SES measures

17   are needed to test whether SES factors such as income, parental education, and stress,

18   moderate our observed associations between EEG aperiodic activity and autism or language

19   outcomes.

20

21   *Conclusions*

22

23   Leveraging data from two longitudinal infant sibling studies with data collected as early as 3

24   months of age, we found that EFL infants regardless of later autism diagnosis have reduced

25   aperiodic activity compared to LL infants at 3 months, but not at 12 months of age. Further, EFL

26   infants with later ASD diagnosis exhibited significantly greater increases in aperiodic activity

27   from 3- to 12-months compared those without ASD diagnosis, and change in aperiodic slope

28   and offset were associated with language development at 18 months of age. Given the close

29   association between ASD and language development, larger longitudinal studies that include

30   EEG data in early infancy are needed to further tease apart the complex interactions between

31   brain and behavior associations.

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