Is frontal EEG gamma power a neural correlate of language in toddlerhood? An examination of late talking and expressive language ability

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Abstract

Few studies have examined neural correlates of late talking in toddlers, which could aid in understanding etiology and diagnosis of developmental language disorder (DLD). Greater frontal gamma activity has been linked to better language skills, but findings vary by risk for developmental disorders, and this has not been investigated in late talkers. This study examined whether frontal gamma power (30-50 Hz), from baseline-state electroencephalography (EEG), was related to DLD risk (categorical late talking status) and a continuous measure of expressive language in 124 toddlers. Frontal gamma power was significantly associated with late talker status over and above demographic factors and receptive language (β=1.96, McFadden's Pseudo R^2=0.21). No significant association was found among subgroups of late talkers with greater likelihood of DLD (McFadden's Pseudo R^2=0.22), or overall expressive language in continuous analysis (Pearson's r=-0.07). Findings suggest that frontal gamma activity may discriminate between groups of children that differ in DLD risk.

Keywords

late talkers; developmental language disorder; toddlers; electroencephalography (EEG); neural oscillations; Bayes factor

1. Introduction

Toddlerhood is a critical period of language development when children undergo rapid growth in their expressive language skills, typically acquiring over 300 spoken words by 2 years of age (Fenson et al., 1994). However, 10-20% of children are late talkers (Dale et al., 2003; Klee et al., 1998; Rescorla, 1989; Rescorla et al., 1993; Rescorla & Achenbach, 2002; Rescorla & Alley, 2001; Zubrick et al., 2007), typically defined as having a vocabulary of less than 50 words and/or not using word combinations by 2 years old, but without another condition such as autism spectrum disorder or deafness to explain the delay (Rescorla, 1989). Late talkers are at higher risk for developmental language disorder (DLD, defined by marked difficulties with speaking, using, and/or understanding language despite no overt causes) – a disorder which has life-long impact (Carpendale & Young, 2002; Durkin & Conti- Ramsden, 2007; Young et al., 2002).

Various risk and protective factors affect the likelihood that a child will be a late talker (e.g., males are more likely to be late talkers) and the likelihood for late talkers to have persistent language difficulties consistent with DLD. Currently, it is not known why most late talkers (estimated at more than 60%) catch up (Dale et al., 2003), whereas others continue to struggle. In a community-based sample of over 1,720 two-year-olds, demographic factors associated with language delay, such as biological sex and family history of speech and language difficulties, only explained 7% of variance in children's vocabulary level (Reilly et al., 2007). Thus, differences in toddlerhood vocabulary remain largely unexplained, hindering scientific and clinical advancement toward identifying correlates that robustly differentiate those toddlers with language delays who will exhibit sustained impairment and those that will naturally catch up. Specifically, studying biological correlates of language development in the brain may uncover

biomarkers to aid in early life identification of neurobiological substrates of DLD (Rescorla, 2011; Siu, 2015) and developmental differences in children's language skills more broadly (Kuhl, 2010; McWeeny & Norton, 2020; Sanchez-Alonso & Aslin, 2022; Zuk et al., 2021).

1.1. Baseline EEG Studies of Language Development

Despite the need to better understand the neurobiology of DLD and heterogeneity of language development, very few brain studies have examined late talkers. To our knowledge, only four studies are published in the literature to date that collect brain measures from late talking children (Chen et al., 2016; Dynak et al., 2021; Grossheinrich et al., 2010; 2019). Three of these studies examined a specific type of neural response to an infrequent versus frequent sound thought to reflect automatic processing of auditory input, two of which pool from the same sample of children (Chen et al., 2016; Grossheinrich et al., 2010; 2019). The fourth study examined differences in brain structure between late talkers and children with dyslexia (Dynak et al., 2021). Although findings from these studies broadly support the notion that there are observable differences in brain activity (Chen et al., 2016; Grossheinrich et al., 2010) and structure (Dynak et al., 2021) between late and typical talkers, current brain studies with late talkers are limited in number and variety. Further, none of these studies examine brain measures concurrent with the age at which late talking is determined in early toddlerhood. The earlier that differences in brain development among late talkers can be identified, the more it can provide clarity into the neurobiological basis of late talking and DLD. This is particularly important because the onset of differences in spoken language are seen much earlier than preschool- and school-age, i.e., when brain measures were collected in the late talker literature to date, but rather are more prominent in toddlerhood.

The limited research at present on brain-behavior associations in late talkers is not

surprising given the challenges in collecting brain data from toddlers (Bell & Cuevas, 2012). Neural measures such as EEG and magnetic resonance imaging (MRI) typically require a high level of child compliance, including engagement with a task or set of instructions, as well as minimizing movement to limit motion artifacts in the data. Late talkers demonstrate more frequent and intense irritability and temper tantrums compared to peers with typical language skills (Manning et al., 2019; Roberts et al., 2018), making it harder to keep them engaged and cooperative with high-demand tasks. Toddlers, especially late talkers, may not understand instructions or redirection during tasks. Thus, collecting EEG during a baseline state (i.e., a minimal task, such as watching a neutral animation, video, or an experimenter blowing bubbles) alleviates some of these challenges, increasing data retention compared to task-based experiments (van der Velde & Junge, 2020) while still capturing meaningful information about the brain. An examination of the brain in a baseline state can support the understanding of how underlying, intrinsic neural circuitry relates to individual differences, which, in turn, can inform differential manifestations of certain behaviors and skills (Fox et al., 2005; Raichle, 2010).

Baseline EEG can be used to measure brain oscillations that are typically categorized into frequency bands, each with different theorized underlying neurobiological mechanisms (Buzsaki, 2006). Brain oscillations play a functional role in the development of cortical networks (Uhlhaas et al., 2010) and are widely studied. Gamma-band oscillations (30-50 Hz frequency range in children) are among the fastest-oscillating frequency bands widely studied in children (Perone et al., 2018). Reduced gamma power has been noted in neurodevelopmental disorders in childhood including reading disability (Nagarajan et al., 1999), autism spectrum disorder (Tierney et al., 2012), and language disorders (Heim et al., 2011).

1.3.1. Gamma Oscillatory Power as a Correlate of Development and Language Ability

Gamma power, reflecting synchronous cortical activity in the gamma band, is hypothesized to play a role in brain maturation starting from childhood (Uhlhaas et al., 2009). Developmental changes in gamma power are most prominent in frontal regions when children are 3-4 years old, peaking at 5 years old (Takano & Ogawa, 1998). Notably, frontal lobe development supports the rapid increase in language comprehension abilities that occur in early toddlerhood (Redcay et al., 2008). Gamma oscillations are also thought to play a role in sensory perception and processing (Engel et al., 2001; Uhlhaas et al., 2010), especially with regard to linguistic information (Giraud et al., 2007; Pulvermüller, 1999). Neural oscillations in the gamma band entrain to gamma-frequency information in the speech signal (Giraud & Poeppel, 2012), which may be one potential mechanism through which the brain forms representations of sounds to set the foundation for language development (Pulvermüller, 1999). Longitudinal associations between early gamma activity and later language skills have been observed, such that greater gamma power during infancy and early toddlerhood predicted better language outcomes at 2 years of age (Cantiani et al., 2019; Wilkinson et al., 2020) and 4 years of age (Gou et al., 2011).

Gamma power may provide insight into the substantial heterogeneity in expressive language abilities characteristic to toddlerhood, and several studies have investigated the relation between EEG gamma power and expressive language concurrently (Benasich et al., 2008; Romeo et al., 2021; Tarullo et al., 2017; Wilkinson et al., 2019; Wilkinson & Nelson, 2021). However, there is variability across studies in the directionality and strength of the association between frontal gamma power and language. **Table 1** summarizes results from main studies that investigate frontal gamma power in relation to direct child assessments of concurrent expressive language. Of note, simple correlations between frontal gamma power and concurrent expressive

language ranged from strong, positive associations (e.g., $r = 0.75$; Benasich et al., 2008) to negative associations with large and moderate effect sizes (e.g., $r = -0.79$; Romeo et al., 2021).

While we did not discern a clear pattern that explained the heterogeneous results across studies, it is possible that differences in how studies characterized participants may account for some of the different findings reported in the literature. Although no significant relation was found between frontal EEG gamma power and concurrent expressive language ability in a group of 43 typically developing 2-year-olds with low familial likelihood for developing autism (Wilkinson et al., 2019), a significant *negative* association was observed among siblings of children with autism, i.e., considered to have a higher likelihood for developing autism themselves (Romeo et al., 2021; Wilkinson et al., 2019). In one study, this pattern persisted when subgrouping these children on whether they did indeed go on to receive an autism diagnosis (Romeo et al., 2021) whereas in another study it did not (Wilkinson et al., 2019). Though, the former study that found a significant association was a smaller subset of the sample examined in the latter study. Thus, it is unclear whether frontal gamma power functions or presents differently among subgroups of children with neurobiological heterogeneity and how such subgroups would be best defined. Late talking status is a core early indicator of DLD but has not been examined as a potential moderating factor to explain different patterns of frontal gamma power early in development.

1.3. Potential Moderators of Language-Brain Relationships

To explore frontal gamma power as a potential neural correlate of language in toddlerhood, it is important to consider how gamma activity might differ between subgroups of toddlers with varying levels of risk for developing DLD. Factors such as male biological sex and lower socioeconomic status (SES) relate to early vocabulary size as well as persistent language

delays (Sansavini et al., 2021). Sex and SES have also been associated with unique patterns of frontal gamma power. One study found that the presence of a significant, positive association between frontal gamma power and language ability was only present in female children (p = 0.03, partial η^2 = 0.05) in a sample of 4-year-old children living in rural Pakistan (Tarullo et al., 2017). Across studies, children with lower-SES backgrounds had less gamma activity, on average, than their higher SES peers (Cantiani et al., 2019; Tomalski et al., 2013; Troller-Renfree et al., 2022). Further, gamma power at 6 months mediated the positive association between SES and later language performance at 2 years of age which suggests potentially meaningful developmental linkages among gamma power, SES, and language in the first few years of life (Cantiani et al., 2019). Receptive language is likely another potential subgrouping factor to consider, as late talking toddlers with lower receptive language ability are considered at even greater risk for DLD (Sansavini et al., 2021). A meta-analysis conducted among studies with late talking toddlers found that receptive language explained about 12% of variance in language outcome among late talkers in toddlerhood (Fisher et al., 2017). Thus, it is possible that patterns of frontal gamma power among late talkers may depend on multiple factors including biological sex, SES, and toddlers' receptive language ability.

1.4. Present Study

To understand whether frontal EEG gamma power may be a correlate of DLD risk and language development more broadly, it is important to explore how frontal gamma power may differ among populations of children with heterogeneous language and brain development, and whether frontal gamma power contributes to variability in concurrent language skill during early toddlerhood. Thus, the current study was designed to examine frontal gamma power as a potential neural correlate of toddlerhood language ability in two aims. The first aim was to

investigate whether differences in frontal EEG gamma power significantly contributed to late talking status in early toddlerhood using a dichotomous analysis (late talker vs. typical talker). To our knowledge, no study thus far has examined differences in gamma power between late and typical talkers, even given that this distinction is extensively used in clinical settings and behavioral research (Sansavini et al., 2021). We also explored potential subgroup patterns between children with increased likelihood for later DLD. Specifically, we examined whether biological sex, SES, and concurrent receptive language ability moderated the association between frontal EEG gamma power and children's late talker status. In the second aim, we investigated the association between frontal EEG gamma power and overall *continuous* expressive language ability, which varied widely across the full sample. Expressive language was examined as a continuous marker of language ability separate from late talking because although late talkers are typically defined by delayed expressive (or spoken) *vocabulary* (i.e., spoken vocabulary size), late talkers have heterogeneous abilities in expressive *language* as a group (i.e., the functional use of communicative language that includes spoken language and use of gesture). The overarching goal of this work is to advance what is currently known about neural correlates of language ability in toddlerhood, particularly in a large group of children over-sampled for late talking status and therefore at greater risk of DLD.

2. Method

2.1. Participants

All participants were recruited from the greater Chicago, IL area via pediatric clinics, community childcare centers and organizations, and print and social media advertisements for the When to Worry (W2W) Study, which includes two integrated cohorts followed longitudinally (n = 410). The When to Worry about Irritability (W2W-I; Wakschlag, 2016,

R01MH107652) study explores infant irritability as an early indicator of risk for developmental psychopathology and enrolled a cohort of children at age 1 who are oversampled for high irritability. The When to Worry about Language (W2W-L; Norton & Wakschlag, 2018; R01DC016273) extended the W2W-I study to explore various predictors of preschool language impairment and enrolled a cohort of late-talking toddlers at age 2. As such, the percentage of late talking toddlers in the W2W study overall exceeded the up to 20% estimate of prevalence in the general population (Reilly et al., 2007; Rescorla, 1989; Zubrick et al., 2007). The W2W Study was approved by Northwestern University (NU)'s Institutional Review Board. Parents provided informed consent for participation and families received compensation for their time and travel.

Participants were eligible for the W2W-L study if English was the primary language used at home (i.e., spoken to the child about 75% of the time or more), and the biological mother was available to participate and complete surveys around the 24-month timepoint. Children were excluded from participation in the W2W study if parents reported premature birth (i.e., < 36 weeks gestation), developmental diagnosis (e.g., autism spectrum disorder, Down syndrome), serious medical illness (e.g., epilepsy), or hearing loss. Participants were excluded if they did not pass the *Modified Checklist for Autism in Toddlers Revised with Follow-Up* (M-CHAT-R/F; Robins et al., 2001), or if parents later reported an autism spectrum disorder diagnosis at any timepoint during the longitudinal study. Children were also excluded if they demonstrated significant cognitive impairments as indicated by a score below the 5th percentile on the Visual Reception subtest of the *Mullen Scales of Early Learning* (MSEL; Mullen, 1995).

313 children that were screened and eligible to enroll in the W2W-L Study. The current analytic sample consisted of 124 toddlers (mean age = 26.7 months, SD = 1.6, Range = 24 to 30). There were 65 typical talkers and 59 late talkers (50 participants met criteria for vocabulary size

percentile; 9 participants met criteria for both vocabulary size and no word combinations by 24 months). **Table 2** provides demographic characteristics for the groups of late and typical talkers in the final analytic sample. **Supplemental Table 1** compares demographics between the analytic sample and the excluded subsample of the 313 children who were eligible to participate in the W2W-L Study (*N*=189).

2.2. Child Language Assessments, Late Talking Status, and Demographic Covariates

The MSEL Expressive and Receptive Language subscales were administered by a trained research assistant to characterize children's language ability (descriptive statistics in **Table 2**). Late talking status was determined for each child based on data collected between 18 and 30 months of age. Children were identified as late talkers if they were below the 16th percentile for Words Produced on the parent-report *Mac-Arthur Bates Communicative Development Inventories* (CDI) *Words & Sentences* form (Fenson, 2007) and/or, for children at or above 24 months of age, not yet combining words per parent report on the CDI. If the participant was missing a completed CDI form, children were identified as late talkers if they were at or below the 16th percentile on the MSEL Expressive Language subtest (*n*=1 in the final sample used for analysis).

Mothers reported maternal education, and this was used as an indicator of SES in analyses (Noble et al., 2015). Maternal education was quantified in terms of years of education as either 12 (high school or GED), 14 (some college or Associate's Degree), 16 (Bachelor's Degree), or 18 years (graduate degree) and examined as a quasi-continuous variable in analysis (Huttenlocher et al., 2010).

2.3. EEG Data Acquisition

EEG data were recorded with a Biosemi ActiveTwo EEG system (Biosemi, Amsterdam,

Netherlands). Electrodes were affixed to a fabric cap that was secured with a chin strap (Electro-Cap Inc., Eaton, OH). EEG was continuously recorded from 32 active Ag/AgCl scalp electrodes (Fp1, Fp2, AF3, AF4, F7, F8, F3, F4, FC1, FC2, FC5, FC6, T7, T8, C3, C4, CP1, CP2, CP5, CP6, P7, P8, P3, P4, Pz, PO3, PO4, O1, O2, Oz) arranged according to the International 10-20 system with offset values kept below 40 microvolts. EEG was recorded at a sampling rate of 512 Hz with 24 bits of resolution, and with a low-pass hardware filter with a half-power cutoff at 104 Hz. Biosemi recordings are made in single-ended mode that amplifies the difference between each electrode site and a common mode sensor (CMS) electrode with referencing offline. EEG was recorded from the parent and child simultaneously using two BioSemi EEG amplifiers that were interconnected via a daisy chain and linked to an acquisition computer (as in Norton et al., 2022). The current study focused on and exclusively analyzed EEG data collected from the child participants.

2.4. EEG Setup and Baseline Task

Children were seated at a table next to their parent and asked to sit still and quietly while watching a video for 4-6 minutes. During EEG recording, the parent remained quietly seated or completed study surveys next to the child. A research assistant was present in the room behind the child to prevent the child from removing the cap and to redirect the child's attention to the video, if needed. Participants were given the option to watch 1) a standard video of nonsocial, child-friendly animations containing no dialogue or 2) their choice of a video from Netflix or YouTube. Offering this choice of video was intended to maximize child compliance during data collection, especially after having completed multiple behavioral study assessments prior to the EEG. The video presented was coded for whether it included language and examined as a confounding variable in analysis.

2.5. EEG Processing

Following data collection, EEG data were processed in EEGLAB v.14.1.1 (Delorme & Makeig, 2004) and the MADE pipeline for data processing and artifact rejection (Debnath et al., 2020). Data were down-sampled to 256 Hz, then high-pass filtered at 0.1 Hz and low-pass filtered at 60 Hz. Bad channels were identified and removed using the EEGLAB plugin FASTER (Nolan et al., 2010), and participants with >20% bad channels were excluded from further analyses (*n*=4 excluded).

Artifactual independent components (ICs), such as eye movements, were examined. First, we made a copy of the original data in preparation for independent component analysis (ICA). Before ICA, the copied dataset was high pass filtered at 1 Hz and then segmented into 1-second non-overlapping epochs. Epochs were removed from the copied dataset if they contained a channel with an amplitude that exceeded a threshold of +/- 1,000 μV or if blink artifacts were present. Additionally, channels were removed before ICA if for more than 20% of the recording, the channel contained artifacts (i.e., exceeded a threshold of +/- 1,000 μV or if EMG present). These same channels were also removed from the original dataset. ICA was then performed on the copied version of the data and the resulting ICA weights were transferred over to the original dataset which was used as the dataset for the remainder of preprocessing. ICs were automatically identified using the adjusted-ADJUST algorithm implemented in MADE (Leach et al., 2020), a version of the ADJUST plug-in (Mognon et al., 2011) tailored for detecting artifactual ICs in pediatric data. The artifactual ICs were removed from the original dataset.

After ICA removal, the original dataset was then segmented into 1-second epochs without overlap. Epochs with residual ocular artifact were identified and removed if frontal channels (Fp1, Fp2, AF3, AF4) exceeded a voltage threshold limit of 150 μV. Additionally,

epochs were marked as artifact if more than 10% of non-ocular channels exceeded 150 μV. Otherwise, channels that exceeded the threshold limit were interpolated on an epoch-by-epoch basis. Lastly, data were re-referenced to the average of all scalp channels. Participants (*n*=12) were excluded from subsequent analyses if they had <40 clean 1-second epochs (similar to thresholds in similar work, Wilkinson et al., 2019). It was confirmed that remaining participants did not have ≥2 of the 3 electrodes of interest interpolated. **Table 2** provides descriptive statistics for the number of epochs included in analysis.

2.6. EEG Power Analysis

Mean power across all usable epochs was calculated for each participant using a Fast Fourier Transform with a 1-second Hanning window with 50% overlap. Analyses focused on a single frequency band of interest, gamma, defined as 30-50 Hz. Although some studies divide the gamma band into low and high gamma, we examined the entire range because we did not have an *a priori* hypothesis about low vs. high gamma in the current study (Cantiani et al., 2019). Gamma power was calculated at frontal electrodes F3, Fz, and F4 as a region of interest given previous work that highlights the role of gamma power from frontal regions in shaping children's development (Benasich et al., 2008; Gou et al., 2011; Tierney et al., 2012; Wilkinson et al., 2019, 2020). Absolute power was calculated as the mean of gamma power values from each EEG segment from the three electrodes of interest; these values were $\log_{10}$-transformed to align with previous studies and to yield a normal distribution (e.g., Wilkinson et al., 2019). Power values ≥2.5 SD from the group mean (*n*=4) were considered outliers and removed from further analysis.

To ensure that potential differences in videos children watched during EEG recording did not meaningfully affect findings, video stimuli were coded as 1) a video of nonsocial animations

containing no dialogue, versus 2) their choice of video from Netflix or YouTube that contained social content/dialogue. We also examined whether frontal EEG gamma power differed between children who watched the nonsocial video (*n*=68), versus those who watched their choice of video that contained dialogue (*n*=56), and whether this depended on participants' late talker status. After checking that normality and homogeneity of variance assumptions were met, a two-way ANOVA was used to test the effect of video stimulus (nonsocial vs. social) x group (late talker vs. typical talker) on frontal gamma power. Gamma power did not significantly differ between video types, and, further, this did not depend on participants' late talker status (*p*s > 0.3, *ns*). To err on the side of caution, video type was still included as a covariate in all analyses.

2.7. Statistical Analyses

Analyses were conducted in the R software environment (Version 4.3.1; R Core Team, 2023). All analyses included demographic covariates of child age (in months), biological sex, and SES, as well as video type during EEG (nonsocial or social). To investigate the first aim, whether differences in frontal EEG gamma power significantly contributed to late talking status in early toddlerhood, a hierarchical logistic regression was performed with late talker status set as the dependent variable and frontal EEG gamma power as the primary independent variable of interest, controlling for covariates. Late talking status was dichotomously coded as 1=late talker and 0=typical talker. Therefore, logistic regressions were used. We also tested whether the association between frontal EEG gamma power and late talker status depended on the presence or degree of other risk factors associated with persistent language delays using a moderated multivariate regression with late talker status as the dependent variable. Listwise deletion was used if participants did not provide data for the moderators of interest. Variance inflation factors (VIFs) were calculated among variables entered as predictors to detect multicollinearity in the

models. All VIFs across the two models were <1.5, indicating low correlation among variables (Hair et al., 2009). In the second aim of the main analysis, a partial Pearson correlation controlling for covariates was calculated between frontal gamma power and expressive language raw scores. This was done after verifying assumptions of normality in primary variables of interest using the Shapiro-Wilks test.

To supplement the second aim, we estimated the Bayes factor (BF_{10}) for the bivariate Pearson correlation between frontal EEG gamma power and expressive language raw scores. Bayesian analysis is a data-driven way to evaluate the probability that the alternative hypothesis is true (H_1: a correlation exists between the two variables) compared to whether the null hypothesis is true (H_0: the two variables are not correlated) (Wagenmakers, 2007). Bayesian hypothesis testing has been recommended as an additional analysis to complement the shortcomings of null-hypothesis significance testing (i.e., overreliance on *p*-values) and is recommended but underutilized in speech, language, and hearing research (Brydges & Gaeta, 2019). Null-hypothesis testing only considers the likelihood of the data under the assumption that the null hypothesis is true (Wagenmakers, 2007), but the magnitude of *p*-values was not intended to be interpreted to imply *more* or *less* likelihood for the null hypothesis to be true. The Bayes factor, on the other hand, estimates the strength of the evidence in favor of null versus alternative hypothesis (Lee & Wagenmakers, 2014; Quintana & Williams, 2018). Larger values of BF_{10} indicate more evidence in support of the alternative hypothesis. For example, a BF_{10} value of 3 suggests that, based on the newly collected data, the H_1 is 3 times more likely to be true compared to H_0 and this corresponds to a moderate level of evidential strength in favor of H_1 (Quintana & Williams, 2018). Conversely, a BF_{10} of 0.33 suggests the opposite, i.e., moderate evidence in favor of H_0. Bayesian analysis was conducted in JASP v.0.17.1 (JASP Team, 2023)

for the simple Pearson correlation between frontal gamma power and expressive language. The alternative hypothesis was set to "correlated" rather than "correlated positively" or "correlated negatively," as there is no clear consensus on the directionality of the association between frontal gamma power and early language abilities in the literature. This calculation did not account for the contributions from covariates as partial correlation analysis is not yet supported in the JASP software. However, as correlations between frontal EEG gamma power and continuous covariates were not significant, this was likely to have only a minor impact on the Bayes factor estimation.

3. Results

Hierarchical logistic regression was used to estimate whether absolute frontal EEG gamma power significantly contributed to late talking status. **Table 3** shows results of the logistic regression. An additional twelve participants were excluded from the logistic regression only due to missing MSEL data, n=112. The first step of the model included age in months and type of video stimulus presented during EEG (social vs. nonsocial). This initial step of the model was not associated with a significant increase in proportion of model fit relative to the intercept-only model (McFadden's Pseudo R^2 = 0.03, p = 0.10, *ns*). The second step of the model included potential indicators for persistent language delays: biological sex, receptive language raw score, and maternal education in years. MSEL receptive language raw score was a significant predictor of late talker status, as expected (β = -0.30, $p \leq 0.001$). Inclusion of step two predictors improved model fit (ΔR^2=0.15), and the overall model was significant (McFadden's Pseudo R^2 = 0.18, $p \leq$ 0.001). In step three of the model, frontal gamma power yielded a significant contribution to late talking status when examined as an individual predictor in the model (β = 1.96, p = 0.046), addressing our key research question. The positive estimated coefficient for frontal EEG gamma

power in the logistic regression suggests that higher gamma power was associated with a greater probability of being a late talker. The odds ratio is 7.1 for frontal EEG gamma power controlling for demographic covariates and concurrent respective language and larger odds ratios correspond to larger probabilities. Odds ratios > 5 are considered large effect sizes (Chen, Cohen, & Chen, 2010). The overall model was significant (McFadden's Pseudo R^2 = 0.21, $p < 0.001$). Inclusion of frontal EEG gamma power improved model fit between steps two and three (ΔR^2=0.03). **Figure 1** visually demonstrates the difference in distribution of frontal EEG gamma power between late and typical talkers. In step four, we tested whether factors associated with a higher likelihood of persistent language delays (i.e., male biological sex, lower concurrent receptive language ability, and lower maternal education) moderated the effect of frontal EEG gamma power on late talker status with two-way interactions. There were no significant interaction effects between frontal EEG gamma power and any of the potential indicators of persistent language delays. The overall model was significant (McFadden's Pseudo R^2 = 0.23, $p < 0.001$). Model fit improved minimally between steps three and four with the inclusion of moderators (ΔR^2= 0.02).

Next, to understand whether frontal gamma oscillations were associated with expressive language among toddlers along a continuous spectrum of language ability rather than just at the cutoff for late talking, we conducted a partial Pearson correlation between frontal gamma power and concurrent MSEL expressive language raw scores controlling for demographic factors (biological sex, age, and maternal education) and video type during EEG (N=124). We found no significant association between frontal EEG gamma power and concurrent expressive language scores, Pearson r = -0.07, p = 0.4, *ns* (bivariate Pearson r = -0.04, p = 0.6, *ns*). **Figure 2** shows the scatterplot of the relation between gamma power and expressive language to visually

demonstrate no clear association between the two variables. The resulting Bayes factor (BF_{10}) on the bivariate Pearson correlation between frontal EEG gamma power and concurrent expressive language raw scores was 0.11. Thus, the ratio between the likelihood of the data fitting under the alternative hypothesis with the likelihood of fitting under the null hypothesis is 0.11, making it 0.11 times more likely that the data occur given that the alternative hypothesis is true, or rather, 11 times more likely that the data occur given that the *null* hypothesis is true (Jarosz & Wiley, 2014). This indicates "Strong Evidence" in favor of the null hypothesis (i.e., the absence of a correlation between frontal EEG gamma power and concurrent expressive language scores) (Lee & Wagenmakers, 2014).

4. Discussion

The purpose of this study was to investigate whether frontal EEG gamma power was a neural correlate of late talking and expressive language ability in early toddlerhood. We found that frontal EEG gamma power at 2 years of age significantly differentiated a group of late talkers from their typical talking peers when controlling for demographic factors and concurrent receptive language ability. Toddlers were more likely to be late talkers if they had higher frontal gamma power. The presence or degree of other risk factors associated with persistent language delays (e.g., male biological sex, lower receptive language ability, and lower maternal education) did not significantly moderate the association between frontal EEG gamma power and late talking status. In continuous analysis, there was no significant correlation between frontal EEG gamma power and concurrent expressive language skill in the overall sample of 2-year-olds when examined regardless of late talking status (N = 124). Using Bayes factor hypothesis testing, we found "Strong Evidence" in favor of the null hypothesis, that is, the absence of a correlation between frontal EEG gamma power and expressive language ability across the full sample.

Overall, these findings provide evidence to suggest that frontal EEG gamma power may meaningfully differentiate late talking toddlers from their typical talking peers controlling for demographic factors and concurrent receptive language, but not for expressive language measured continuously along a spectrum of ability.

This study is the first to find that frontal gamma power is a potential marker of late talking in toddlers and is the largest study in this area to date. That and our finding of the lack of a significant association (with strong evidence in favor of the null hypothesis) between frontal gamma power and broader expressive language ability in the full sample suggests that frontal gamma activity may be more robust in discriminating between groups of children that differ in neurobiological risk for persistent language difficulties, rather than being sensitive to individual differences in expressive language abilities more broadly. This is contrary to previous work that found a strong, positive correlation between frontal EEG gamma power and concurrent expressive language in a sample of 23 2-year-olds collapsed across family history of language impairment (Benasich et al., 2008) but does reflect other findings that report differential patterns of associations that depend on underlying neurobiology related to risk for developmental disorder (Wilkinson et al., 2019). Still, the directionality of our findings in the dichotomous analysis examining late talker status as the dependent variable differs from previous research that found reduced gamma power in neurodevelopmental disorders such as reading disability, autism spectrum disorder, and language disorders (Heim et al., 2011; Nagarajan et al., 1999; Tierney et al., 2012). Together with other reports of positive longitudinal associations between early gamma power in infancy and later language skills at 2 years old (Cantiani et al., 2019; Wilkinson et al., 2020), it might be expected for late talkers to have lower gamma power. We instead found that late talkers were more likely to have *greater* gamma power, and this finding demonstrated a

strong effect size in our logistic regression model. Though our study differed in that gamma power was considered *a priori* in frontal regions only and during a baseline-state, as opposed to wider variability in brain regions and task-related EEG collected across various ages in previous work (Heim et al., 2011; Nagarajan et al., 1999; Tierney et al., 2012).

Although late talking is considered a risk factor for DLD, still about 60% of late talkers develop typical language by preschool thus representing wide heterogeneity of language outcomes within late talkers as a group (Sansavini et al., 2021). This heterogeneity in developmental outcome among late talkers strongly suggests that late talking toddlers who indeed go on to receive a DLD diagnosis may be developmentally different from those who simply start talking later than other children, as "late blooming" may not necessarily be driven by the same clinical, neurobiological factors that underlie DLD. This within-group heterogeneity is plausible for the makeup of the current sample as the effect of frontal gamma power on late talking status was marginally significant (p=0.046) and the ability to group the presence of neurobiological substrates underlying DLD risk may influence the effect that can be detected. Thus, we tested whether factors associated with a higher likelihood of persistent language delays among late talkers (i.e., male biological sex, lower maternal education, and lower concurrent receptive language ability) moderated the effect of frontal EEG gamma power on late talker status and would thus illuminate subgroups of late talkers for which the effect of frontal gamma power on late talker status was stronger. We found no evidence to suggest these subgroup patterns in our sample. This may also be expected as subtle differences in genotypic risk for language disorders have not yet been robustly identified in the literature and it is not understood how to best characterize the subgroup of late talkers who will have persistent language difficulties (Desmarais et al., 2008). For example, maternal education, one facet of SES, has been

examined as an indicator of socioeconomic advantage and disadvantage contributing to enrichment in children's language environments (Fernald et al., 2013; Hart & Risley, 1995; Noble et al., 2015). Yet, maternal education is a very indirect measurement for the more complex intra/interpersonal and societal factors at play in the socioeconomic influences of children's opportunities to learn, use, and be exposed to language. Additionally, it is important to note that the present sample had a narrow distribution of maternal education, with 87% of participants having a mother with a Bachelor's degree or higher and thus may have lacked a wide enough distribution needed to adequately examine maternal education level as a contributing factor.

There were several advantages to the analytical approach adopted in the current study that may elucidate variability in findings across the literature. Sample size and composition may be a source of variability, particularly in studies that examine brain-behavior associations (Marek et al., 2020). This study addressed limitations related to homogeneous sample composition discussed in similar work (Wilkinson et al., 2019) by investigating across a wide distribution of expressive language skill that is more characteristic of patterns of language development at this age. This permitted us to explore two complementary characterizations of language in toddlerhood using categorical late talker status stratified by expressive vocabulary and continuous expressive language along a continuum from weaker to stronger abilities. Investigation of both is particularly important for keeping in line with clinically relevant definitions of at-risk populations, while also capturing heterogeneity between children's individual abilities (Hearnshaw et al., 2023).

The current study's final sample included 124 toddlers which is considerably larger relative to most previous work and, to our knowledge, is the largest study to-date to examine the association between baseline frontal EEG gamma power and language abilities in children.

Novel to previous work in this area, we used Bayes factor hypothesis testing to supplement results from standard *p*-value null-hypothesis significance testing. We found "Strong Evidence" in favor of the null hypothesis that there is no correlation between frontal EEG gamma power and concurrent expressive language ability. A potential limitation of using Bayes factors, however, is that in a Bayesian framework researchers must specify the shape of the prior distribution of the parameter of the model. It is important to be transparent about this step when interpreting Bayes factors as it introduces a level of researcher subjectivity (Keil et al., 2022). For neuroscience research, it is recommended to follow default prior assumptions to increase the objectivity of analysis (Keysers, Gazzola, & Wagenmakers, 2020) which is the protocol followed in the current study. Also, calculation of the Bayes factor in a logistic regression is not yet supported in statistical software and thus was not examined here. We also parsimoniously selected variables of interest based on support from the literature, i.e., a specific frequency band explored in a predefined brain region relative to explicitly defined domains of language. This reduced the Type I error rate by limiting multiple comparisons that would otherwise be a consequence from exploring several frequency ranges across multiple regions of the brain in relation to widespread domains of language.

Overall, the study expands on what is currently known about the relation between frontal gamma power and language by being the first study to investigate this in the context of late talking in toddlerhood. To better understand how to subgroup later talkers with different neurobiological trajectories of brain development, longitudinal assessments of this sample are currently underway and provide an important focus for follow-up analyses. Findings from the current study suggest promise for future work investigating frontal EEG gamma power particularly as a prognostic marker to predict the likelihood of DLD. To this end, it would be

invaluable for research in this area to continue efforts to better characterize DLD risk using neural correlates and the many other interacting factors that influence whether a child develops a language disorder.

Table 1. Summary of four illustrative studies on the association between frontal EEG gamma power and concurrent expressive language in children.

Authors	*Age Cohort*	*Sample Description*	*n*	*Simple Pearson Correlation, r*	*Interpretation of Effect Size*[a]
Benasich et al., 2008[b]	16 months	Full sample with and without family history of language impairment	22	0.60***	Moderate
	24 months		23	0.75***	Large
	36 months		18	0.40, NS	Small
Romeo et al., 2021[c,d]	24 months	LL for ASD	24	0.51*	Moderate
	24 months	HL for ASD	22	-0.61**	Moderate
	24 months	Subgroup of HL for ASD group without ASD	12	-0.15, NS	Small
	24 months	Subgroup of HL for ASD with ASD diagnosis	10	-0.79**	Large
Wilkinson et al., 2019	24 months	LL for ASD	43	0.01, NS	Small
	24 months	HL for ASD	58	-0.24**	Small
	24 months	Subgroup of HL for ASD group without ASD diagnosis	42	-0.31, NS	Small
	24 months	Subgroup of HL for ASD with ASD diagnosis	16	-0.21, NS	Small
Wilkinson & Nelson, 2021	30 months	Typically developing[e]	12	-0.24, NS	Small
	54 months	Males with Fragile X Syndrome (FXS)	11	0.75**	Large

Notes. Reported are the simple Pearson correlations, if provided, for each study. *p<0.05, **p<0.01, ***p<0.001. Child language was measured via Mullen Scales of Early Learning (MSEL) Expressive Language subtest or Preschool Language Scales (PLS; either 3rd or 5th edition) Expressive Communication subtest. LL = low likelihood; HL = high likelihood; ASD = autism spectrum disorder.

[a] = Interpretation of effect size is based on Cohen (1988).

[b] = The exact correlation for this comparison is not reported in the paper but is instead illustrated in a bar graph in the original paper (Benasich et al., 2008). Thus, the correlation is visually approximated here.

[c] = Explored brain regions in addition to frontal region in correlational analyses that are not reported here.

[d] = Sample in the study conducted by Romeo et al. (2021) was drawn from a larger sample reported on by Wilkinson et al. (2019).

[e] = Participants are age-matched to FXS group in the same study (Wilkinson & Nelson, 2021).

Table 2. Sample characteristics. MSEL Receptive Language scores were not available for 12 participants (l0 late talkers, 2 typical talkers). Some percentages do not add to 100 due to rounding.

	Late talkers	*Typical talkers*
Number of participants	59	65
Age, *mean ± SD*	27.1 ± 1.8 months	26.4 ± 1.4 months
Sex, *% female*	58%	55%
Maternal education, *n*		
High school (12 years)	2	0
Some college/Associate's Degree (14 years)	6	8
Bachelor's degree (16 years)	30	28
Advanced (18 years)	21	29
Race, *n (%)*		
Asian	0 (0%)	1 (2%)
Black/African American	9 (15%)	6 (9%)
White/Caucasian	44 (75%)	51 (79%)
More than one race	4 (7%)	6 (9%)
Unknown/not reported	2 (3%)	3 (2%)
Ethnicity, *n (%)*		
Hispanic/Latino	4 (7%)	5 (8%)
Not Hispanic/Latino	55 (93%)	60 (92%)
Number of EEG epochs included in analysis, *mean ± SD (range)*	245 ± 96 (45-366)	253 ± 113 (44-426)
MSEL Expressive Language raw score, *mean ± SD (range)*	21.8 ± 3.7 (14-28)	27.0 ± 3.8 (17-38)
MSEL Expressive Language T-score, *mean ± SD (range)*	42.3 ± 8.2 (23-56)	57.1 ± 9.6 (36-79)
MSEL Receptive Language raw score, *mean ± SD (range)*	24.8 ± 4.1 (14-33)	28.0 ± 3.5 (15-37)
MSEL Receptive Language T-score, *mean ± SD (range)*	46.2 ± 10.6 (20-64)	56.8 ± 10.0 (24-79)

Table 3. Moderated multivariate regression analysis results for variables predicting late talking status.

Variable	*β*	*Std. Err.*	*McFadden's Pseudo R^2*	*p*
Step 1			0.03	0.10
Age	0.20	0.13		0.11
Video stimulus during EEG (social vs. nonsocial)	0.51	0.39		0.19
Step 2			0.18	<0.001***
Sex (1=F, 0=M)	0.24	0.45		0.60
MSEL receptive language raw score	-0.30	0.08		<0.001***
Maternal education (years)	0.09	0.17		0.59
Step 3			0.21	<0.001***
Frontal gamma power	1.96	0.98		0.046*
Step 4			0.23	<0.001***
Frontal gamma power*sex	2.60	2.24		0.25
Frontal gamma power*MSEL receptive language raw score	-2.81	0.33		0.39
Frontal gamma power*maternal education	-0.55	0.75		0.47

Notes. The dependent variable is late talking status (late talkers = 1, typical talkers = 0). N = 112 due to missing MSEL Receptive Language data. *** $p < 0.001$

Figure 1. Violin plot of frontal gamma power for late and typical talker groups.

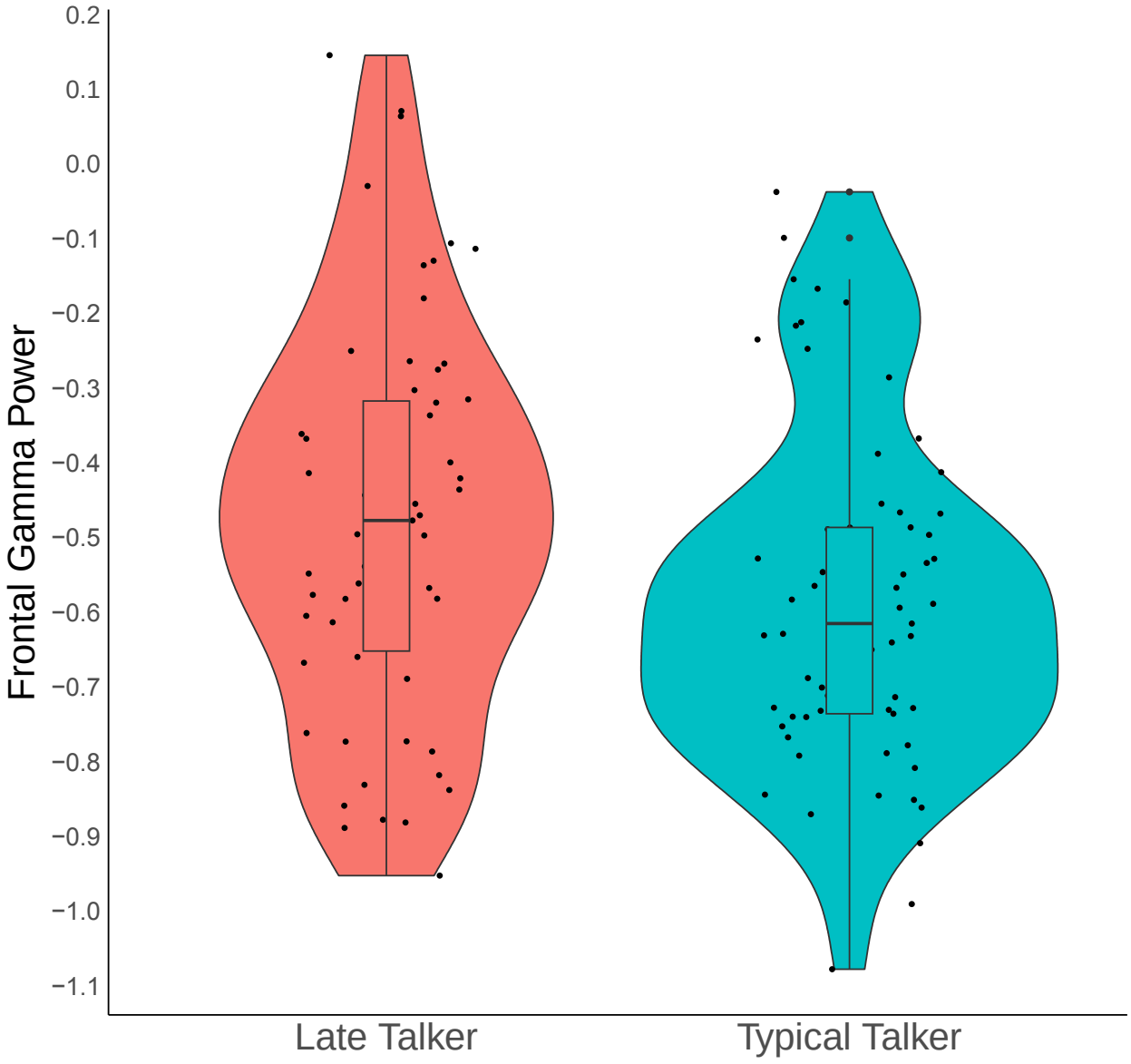
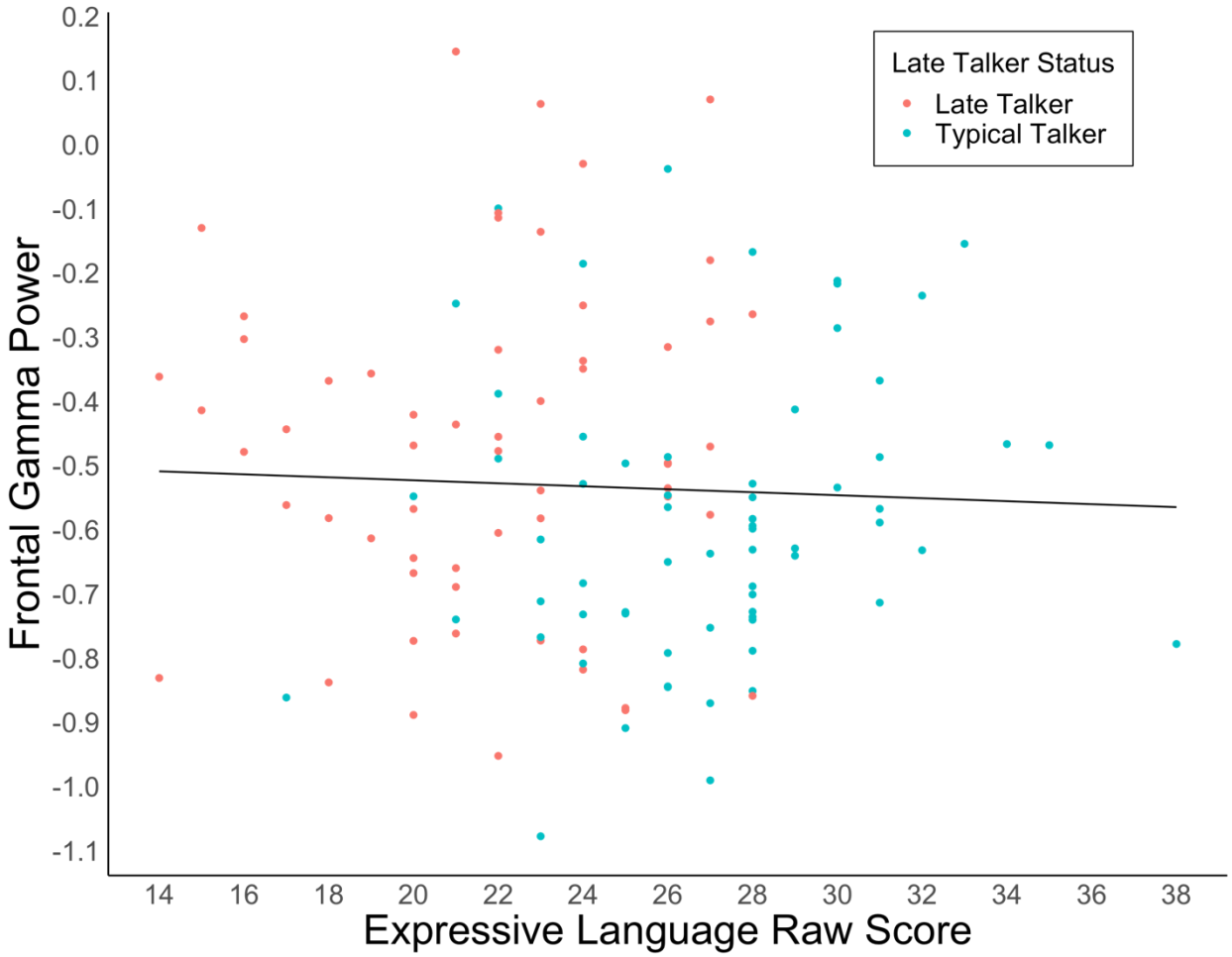


Figure 2. Scatterplot of frontal gamma power and expressive language scores.



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