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Gamma oscillations and auditory perception: A cluster-based statistic investigation in infants at higher likelihood of autism and developmental language disorder

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ABSTRACT

The ability to process auditory information is one of the foundations of the ability to appropriately acquire language. Moreover, early difficulties in basic auditory abilities have cascading effects on the appropriate wiring of brain networks underlying higher-order linguistic processes. Language impairments represent core difficulties in two different but partially overlapping disorders: developmental language disorder (DLD) and autism spectrum disorder (ASD). The aim of this study was to investigate basic auditory processes in 12-month-old infants at high likelihood (HL) of developing either DLD or ASD in response to standard tones embedded in a non-speech multi-feature oddball paradigm to discern early differences in how auditory processing relates to language acquisition. To do so, we focused on gamma-band oscillations due to the role of gamma activity in coordinating activity among neural assemblies and thus enabling both sensory and higher-order processing. Considering reported hemispheric asymmetries in auditory and linguistic processing, we chose to refer to a cluster-based method to investigate the whole scalp activity in the gamma range. Our results show that HL-ASD infants are characterized by

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differences in auditory gamma compared with their typically developing peers. These results may imply an enhanced sensitivity to auditory stimuli in HL-ASD infants that might negatively affect their ability to regulate responses.

Introduction

Acoustically transmitted signals constitute the fundamental building blocks of oral language: being able to extract acoustic components and identify relationships among them are pivotal abilities for the early development of language (Mueller et al., 2012). Substantial empirical evidence points out that basic auditory perception is crucial for acquiring language competences across both typical and atypical development (Corriveau et al., 2007; Edgar et al., 2015; Mueller et al., 2012). Indeed, differences in low-level processing affect the way in which individuals develop higher-order core abilities (D'Souza & Karmiloff-Smith, 2017). As cognitive functions emerge from complex interactions between different brain areas (Johnson et al., 2009), initial impairments related to basic components might have cascading effects on the development of brain structures subtending higher-order cognitive functions (D'Souza & Karmiloff-Smith, 2017; Johnson et al., 2009). Consequently, understanding low-level auditory processes is fundamental to understand linguistic development in both typical and atypical populations and to define and support clinical interventions devoted to improving developmental outcomes (Kujala & Leminen, 2017; Tallal, 2004).

Language development dysfunctions represent the core diagnostic signature of developmental language disorder (DLD; previously known as specific language impairment). DLD is a neurodevelopmental disorder that affects the processing and production of spoken language (Leonard, 2014). Affected children exhibit no clear hearing, neurological, psychiatric, or social impairments, allowing them to respond appropriately to their social environment. However, they still fail to develop age-appropriate language skills. (Cumming et al., 2015). Electrophysiological studies consistently suggest that children with DLD are characterized by a differential processing of non-speech acoustic stimuli (Corriveau et al., 2007), strengthening the argument that lower-level processing difficulties in the auditory domain may subtend dysfunctions in language development (Corriveau et al., 2007; Cumming et al., 2015). Coherently, the etiology of language impairments in DLD has been consistently associated with basic attentional and perceptual processes (Benasich & Tallal, 2002; Tallal, 2004; Weber-Fox et al., 2010). Indeed, the proper functioning of cortical areas specialized in auditory cue perception is critical for language acquisition (Benasich & Tallal, 2002). Thus, despite language deficits being the most obvious characteristic of DLD, subtler deficits in processes essential for language learning may represent the foundations of manifest impairments (Weber-Fox et al., 2010). However, DLD has received considerably less attention compared with other neurodevelopmental disorders (Laasonen et al., 2018). This is surprising given that DLD-associated difficulties are not restricted to the developmental period but rather continue to negatively affect individuals' social, academic, and occupational activities well into adulthood (Laasonen et al., 2018). Thus, although the need to understand etiological and protective factors of the disorder is very high, the mechanisms leading to neurophysiological abnormalities in DLD are not yet clear (Laasonen et al., 2018).

A disorder whose early signs are deficits in language skills in the first year of life is autism spectrum disorder (ASD; Tager-Flusberg, 2016). Interestingly, however, language delays are not included in the *Diagnostic and Statistical Manual of Mental Disorders–Fifth Edition* (DSM–5) as a criterion for a diagnosis of ASD. ASD is a neurodevelopmental disorder characterized by deficits in social communication coupled with restricted, stereotyped, and repetitive patterns of behaviors (American Psychiatric Association [APA], 2013). Among non-social dysfunctions, one of the most observed symptoms in ASD regards aberrant responses to low-level sensory input, especially in the auditory domain (Font-Alaminos et al., 2020; Kuiper et al., 2019; Robertson & Baron-Cohen, 2017; Stroganova et al., 2013).

Auditory processing is well-studied in ASD, with reports of atypical responses at both behavioral and neurophysiological levels across the lifespan (Arnett et al., 2018; Orekhova et al., 2008, 2012). ASD neurophysiological abnormalities throughout various brain and brainstem regions implicated in auditory processing are a consolidated finding (O'Connor, 2012). For example, pre- and post-stimulus abnormalities in processing tones have often been observed in children with ASD (Edgar et al., 2014), and have been found to be associated with atypical hemispheric lateralization (Edgar et al., 2014; Stroganova et al., 2013). Many studies have also identified amplitude and latency differences between individuals with ASD and healthy controls to auditory stimuli across many major event-related potential (ERP) components (O'Connor, 2012). However, the relationship between auditory processing atypicalities and language impairments remains unclear (Arnett et al., 2018; O'Connor, 2012).

To sum up, substantial empirical evidence supports the claim that early auditory abilities affect later outcomes of language development in different neurodevelopmental disorders, including DLD and ASD (Oram Cardy et al., 2008). However, although auditory perceptual processing impairments in both children with ASD and DLD have been identified by separate lines of research (Benasich & Tallal, 2002; Corriveau et al., 2007; Edgar et al., 2014; Kolesnik et al., 2019; Shafer et al., 2005; Stroganova et al., 2013; Weber-Fox et al., 2010), the two disorders have rarely been compared (Oram Cardy et al., 2008).

Given that basic auditory abilities develop very early and represent the foundations of higher-order acquisitions such as language, and failures severely impair subsequent developmental achievements (Benasich & Tallal, 2002), studying infants at higher likelihood of developing either ASD or DLD during the early phases of life is of great importance. Indeed, given that the first months of life are characterized by an impressive amount of neural plasticity compared with later ages, the social and sensory deficits associated with neurodevelopmental disorders might not be fully embedded (Hazlett et al., 2017). Early interventions, tailored on the observed impairments, may prove to be more effective than the ones implemented later in life (Hazlett et al., 2017). Regarding ASD, although behavioral symptoms and phenotypes begin to become apparent already at 18 months of age (Ozonoff et al., 2015), a clinical diagnosis is still often not received until the third birthday (Shumway et al., 2011). To help detect early markers of the disorder, an optimal way is studying infant siblings of children with ASD, referred to as high likelihood of developing ASD (HL-ASD) infants (Ozonoff et al., 2011). Indeed, findings regarding recurrence rates in siblings of children with ASD suggest a rate of 18.7% compared with the general population (Ozonoff et al., 2011). The same can be said for DLD given that studying infants of biological families with a high prevalence of DLD, and thus at higher likelihood of developing DLD (HL-DLD), is fundamental for the identification of early markers of the disorder (Benasich et al., 2006; Cantiani et al., 2016). However, whereas research on familial aggregation of ASD symptoms is thriving, research on the incidence of language difficulties in siblings of DLD children has received less attention. To date, the prevalence of language difficulties in first-degree relatives of individuals with a diagnosis of DLD is estimated at 20% to 80%, depending on the criteria used for DLD (Carroll & Myers, 2010; Flax et al., 2003; Tallal et al., 2001). Previous studies on 6-month-old HL-DLD infants demonstrate that auditory processing of non-speech tones is characterized by impairments in early sensory responses (Benasich et al., 2006; Cantiani et al., 2016; Choudhury et al., 2011). Moreover, in a study comparing 12-month-old HL-DLD and HL-ASD infants, both at risk groups were characterized by slower auditory processing, in the form of delayed auditory ERP responses, for fine-grained acoustic discrimination. Such acoustic discrimination was then found to represent an early predictor of language development at 20 months of age (Riva et al., 2018). Thus, cortical auditory processing dysfunctions may affect language development, representing a possible common mechanism across ASD and DLD (Riva et al., 2018). However, due to the lack of studies comparing DLD and ASD, more evidence is warranted on this topic to disentangle patterns of associations.

Although ERP analyses have proven fruitful, they do not capture certain aspects of the information in the electroencephalography (EEG) signal. A prime example is the patterns of associations with cognitive functions conveyed by frequency bands. Emerging evidence suggests that oscillatory activity in response to auditory stimuli represents a useful marker to assess cortical processing. Among brainwaves, those with the highest frequency, roughly between 30 and 100 Hz, comprise the gamma bandwidth (Herrmann et al., 2010). The mechanisms that originate gamma oscillations are well-known and

are related to the activity of fast-spiking parvalbumin interneurons (Cardin, 2018). It has been proposed that gamma oscillations play a role in organizing information flow and coordination between brain regions (Fries, 2009; Mathalon & Sohal, 2015). Moreover, long-range oscillatory activity within the gamma band correlates with the maturation of perceptual functions during infancy (Csibra et al., 2000; Saby & Marshall, 2012; Uhlhaas et al., 2010). Indeed, gamma oscillations are reportedly implicated in functions ranging from early sensory processing to higher-order cognition across development such as language processing (Asano et al., 2015; Edgar et al., 2015; Jensen et al., 2007; Mathalon & Sohal, 2015). Specifically, gamma-band activity in ASD has been reported to be sensitive to speech sounds and lexical contingencies, as well as being positively related to language performance, with increased performance guided by higher power in the gamma range (Basirat et al., 2008; Edgar et al., 2015; Orekhova et al., 2008; Rojas et al., 2008, 2011; Wilson et al., 2007). However, it remains unclear whether the reported dysfunctions are related to wide ASD characteristics or are specifically related to language impairments per se. In fact, gamma oscillations during auditory discrimination have also been proposed as a central impairment in DLD, with reduced activity in the left hemisphere and increased right lateralized gamma in response to tones in HL-DLD infants (Cantiani et al., 2019) and selective difficulties in the processing of speech temporal modulations in the gamma range (Goswami et al., 2016).

Considering the role that basic auditory processing has in shaping subsequent language acquisitions, the aim of the current study was to investigate early auditory processing in 12-month-old HL-DLD, HL-ASD, and typically developing (TD) infants, focusing on gamma oscillation differences measured through the cluster-based statistic (CBS; Maris & Oostenveld, 2007). By comparing HL-ASD and HL-DLD infants, we aimed to disambiguate impairments related to auditory processing, going beyond the similarities between disorders in an attempt to find specific etiological characteristics. Specifically, we hypothesized that atypical auditory processing in both HL-DLD and HL-ASD would represent stand-alone dysfunctions, thus not a byproduct of language difficulties but rather an independent issue that uniquely contributes to the observed linguistic impairments (Riva et al., 2018). To investigate fundamental auditory processes, we focused on pure tones rather than speech stimuli, ensuring a clearer understanding of the underlying basic dysfunctional mechanisms. We then applied time–frequency decomposition to analyze gamma activity in detail. Furthermore, considering the reported hemispheric asymmetries in both auditory and linguistic impairments in infants at risk for ASD and DLD (Cantiani et al., 2019; Haesen et al., 2011; Oram Cardy et al., 2008; Roberts et al., 2011), as well as the existing gap in the literature regarding oscillatory patterns across these disorders, we chose to investigate topological configurations on the whole sensor space to avoid statistical confounds. We referred to the CBS, an exploratory non-parametric statistic able to control for false positives in sensor, time, and frequency points of interest (Maris & Oostenveld, 2007).

Method

The analyses presented in this study represent a re-analysis of a previously collected dataset, focusing on ERP responses to a rapid auditory processing task. See Cantiani et al. (2016, 2019)) and Riva et al. (2018) for detailed descriptions of previous findings.

Participants

Participants recruitment was part of an ongoing longitudinal project aiming at identifying early risk markers for language learning in ASD, DLD, and the general population, focusing on infants at familiar risk for such disorders (Cantiani et al., 2016, 2021; Riva et al., 2018).

HL-DLD infants were recruited either through advertisement or by physician referral. As described in Cantiani et al. (2016), HL-DLD infants underwent an initial assessment to exclude concomitant disorders such as intellectual disability, ASD, and other sensory or neurological conditions in any first-degree relatives. Then parents were administered a standardized reading performance test (Judica & De Luca, 1993), a single-word test, and a pseudo-word reading test (Sartori et al., 1995). Infants were assigned to the HL-DLD group if at least one first-degree relative had been diagnosed with language

disorders or scored 2 standard deviations below the population mean on at least one of the three reading tests (Cantiani et al., 2016). Recruitment of HL-ASD infants took place through the Italian Network for Early Detection of Autism Spectrum Disorders (NIDA Network). This ongoing multi-site cooperation was developed in order to closely monitor at-risk infants to detect early signs of disorder whenever present. To be recruited into the HL-ASD group, an infant needs to have an older full biological sibling with a clinical diagnosis of ASD, confirmed by the ICD-10 (World Health Organization, 1993), DSM-IV-TR (APA, 2000), or DSM-5 (American Psychiatric Association, 2013) criteria, depending on the time of diagnosis (Riva et al., 2022). TD infants were recruited through advertisements in two hospitals in the North of Italy.

Because the aim of this study was to capture differences in basic auditory perception across 12-month-old HL-DLD, HL-ASD, and TD infants, we restricted our analysis to the standard complex tones presented among the oddball paradigm in Riva et al., 2021 study. Indeed, considering the noisiness of infants' EEG data, this choice enabled us to retain more data in the form of an increased number of participants and data epochs. Descriptions of stimulus characteristics are presented more in depth in the next paragraph.

The final sample consisted of 71 12-month-old infants divided into three groups: TD (n = 25), HL-ASD (n = 25), and HL-DLD (n = 21). All infants passed a hearing screening at birth. Four participants were excluded due to technical errors (TD = 2, HL-ASD = 1, HL-DLD = 1). The groups were matched for sex, age, gestational weeks, and socioeconomic status (SES). Table 1 shows descriptive statistics, in the form of a χ^2 for sex and F tests for the other numerical variables of individual and sociodemographic characteristics of participants.

Inclusion criteria were (a) having native Italian-speaking parents, (b) gestational weeks > 36 weeks, (c) birth weight $\geq$ 2500 g, (d) Bayley cognitive scaled score $\geq$ 7 (Bayley, 2009) or Griffiths developmental quotient $\geq$ 70 (Griffiths, 1984), (e) absence of known medical, genetic, or neurological conditions, and (f) absence of major complications in pregnancy and/or delivery likely to affect brain development. Written informed consent was obtained from all parents prior to testing. The experiment was performed in accordance with relevant guidelines and regulations and was approved by the Eugenio Medea Scientific Institute ethical and scientific committees.

Experimental design: Stimuli and procedure

Our stimuli were complex tone pairs (see Fig. 1). Each tone had a fundamental frequency of 100 Hz with 15 harmonics (6 dB roll-off per octave) and a duration of 70 ms (5 ms rise time and 5 ms fall time). The inter-stimulus interval within the two tones in the pair was set to 70 ms, whereas the time interval between the tone pairs varied randomly between 700 and 900 ms. A temporal jitter of ± 100 ms was added to the inter-trial interval to reduce the distortion that results from overlapping neural activity between previous and subsequent stimuli (Luck, 2014) and to avoid habituation throughout testing. As previously described, the current study represents a re-analysis of a previously collected dataset. Within this context, the complex tone pairs that were the focus of our analyses were presented among deviant stimuli (different for fundamental frequency or duration). Stimuli were presented in the context of a passive oddball paradigm in which 1200 stimuli (80% of which were standard doublets, 10% frequency deviant, and 10% duration deviant) were delivered in a pseudo-random order, so that at least three standard tone pairs were presented before each deviant pair. For our analyses, we considered all tone pairs between two deviants. All stimuli were presented free field at an intensity of 75 dB via speakers located on either side of and equidistant (95 cm) from the participant. A graphic representation of stimuli is presented in Fig. 1.

EEG data acquisition and preprocessing

The experiment took place in a sound-attenuated, electrically shielded room. During EEG recording, each infant sat on the caregiver's lap. The EEG signal was recorded using a 65-electrode HydroCel Geodesic Sensor Net (Electrical Geodesics, Eugene, OR, USA). The vertex was used as online reference, and the signal was sampled at 250 Hz. Impedances were kept below 50 kΩ, as recommended for Electrical Geodesics high input–impedance amplifiers. Data were preprocessed using the EEGLab toolbox Ver-

Table 1
Descriptive statistics of participants' individual and sociodemographic characteristics, including a χ^2 test for sex and *F* tests for the other numerical variables.

	TD (n = 25)	HL-ASD (n = 25)	HL-DLD (n = 21)	Group difference
Boys/Girls	9/16	14/11	11/10	2.24, p = .32
Age (in days)	M = 376.72 SD = 9.64	M = 380.96 SD = 15.97	M = 376.9 SD = 12.94	0.81, p = .44
Socioeconomic status[a]	M = 66.2 SD = 14.73	M = 55.6 SD = 19.38	M = 56.42 SD = 23.45	2.29, p = .10
Expressive language at 20 months of age[b]	M = 41.87 SD = 25.4	M = 38.2 SD = 31.25	M = 25.26 SD = 19.18	2.27, p = .11

[a] Socioeconomic status was scored according to the Hollingshead 9-point scale, whereby a score ranging from 10 to 90 was assigned to each parental job and the higher of two scores was used when both parents were employed (Hollingshead, 1975).
[b] Percentile scores in Language Development Survey (Rescorla & Alley, 2001).

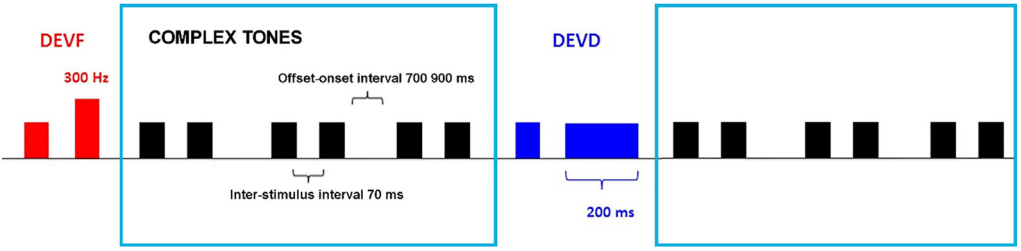

Fig. 1. Graphical representation of the stimuli presented and their characteristics. The focus of our analyses was complex pair tones in between frequency deviant (DEVF) and duration deviant (DEVD) sounds. At least three pairs of tones were presented between deviants.

sion 2020 (Delorme & Makeig, 2004) in MATLAB Version R2020b. First, data were high-pass filtered at 0.1 Hz and low-pass filtered at 100 Hz using the zero-phase Hamming-windowed sinc Finite Impulse Response filter implemented in EEGLab. Cutoff frequencies were 0.05 and 100.05 Hz (−6 dB). The 50-Hz line noise was removed by using the *CleanLineNoise* function incorporated into the PREP pipeline (Bigdely-Shamlo et al., 2015). Data were then cleaned using the Clean Rawdata EEGLab plug-in (Kothe & Makeig, 2013; Plechawska-Wójcik et al., 2019): channels were deemed "bad" and removed if flat for longer than 5 s or through computation of each channel's correlation to its random sample consensus (RANSAC) reconstruction for each window. The correlation threshold for channel removal was set at.80. The maximum number of channels rejected was 13. Then, to all recordings, a principal component analysis (PCA) decomposition was applied to identify artifactual PCs (defined by a comparison against the data's own cleanest parts) in order to reject them and reconstruct activity from the remaining components. Windows' standard deviation was set at 20. The mean number of channels rejected was 7.05 in the HL-DLD group (SD = 2.74), 7.29 in the HL-ASD group (SD = 2.42), and 6.92 in the TD group (SD = 1.73). Through t tests, we controlled for possible differences across groups. The number of channels rejected did not differ significantly across groups (all ps > .54). Then, to reduce the number of artifacts, data were subjected to soft wavelet-thresholding correction. Wavelet-thresholding detects artifacts across time and frequency without distorting spectra (Monachino et al., 2022). This step was performed through an adapted version of the *happe_wavThresh* function comprised in the HAPPE + ER pipeline (Monachino et al., 2022). After these steps, removed channels were spherically interpolated by referencing to the full-rank channel matrix and data were re-referenced to average reference. Data were then divided into 3-s epochs, beginning 500 ms before and ending 2500 ms after the onset of the first tone in the pair, in order to gather enough data to perform time–frequency decompositions and avoid artifacts due to the proximity of the time interval of interest with epoch edges. For the final epoch rejection, a threshold of ± 100 μV was set to remove

abrupt changes in amplitude, and a joint probability-based rejection was used to remove epochs exceeding by 3 standard deviations the mean of activity across channels. Joint probability-based rejection is useful in isolating high-frequency artifacts such as muscle activity (Monachino et al., 2022). The mean number of trials was 986.52 (*SD* = 182.83) for the TD group, 1032.64 (*SD* = 144.56) for the HL-ASD group, and 1079 (*SD* = 112.33) for the HL-DLD group. Given the high number of epochs, we opted for strict parameters for epoch rejection in order to have as clean recordings as possible. The mean number of artifact-free trials was 678 (*SD* = 163.18) for the TD group, 677.88 (*SD* = 165.2) for the HL-ASD group, and 648.56 (*SD* = 146.79) for the HL-DLD group. Consequently, 26.5% of trials were rejected for the TD group, 22.9% for the HL-ASD group, and 24.3% for the HL-DLD group. To check for any disparity between groups, we performed *t* tests; no significant differences were found between groups ($p > .50$).

Time–frequency decomposition

To investigate gamma activity, we executed a time–frequency decomposition focusing on induced power in our EEG data. Time–frequency decomposition was performed using a multi-taper convolution solution to achieve better control over the frequency smoothing, which is advantageous for the analysis of frequencies above 30 Hz. Power was calculated by using the discrete Fourier transform on sliding time windows. Specifically, for each window data were "tapered" and then the power was computed for each tapered data segment and successively combined. The frequencies of interest were set to be from 30 to 80 Hz in 1-Hz steps. The time interval of interest was set to be from 0 to 900 ms after stimulus onset in 10-ms steps. The length of the sliding window was 5 cycles per time window, and the smoothing parameter was set to increase with frequency by 0.4. Activity in response to tone pairs was averaged across trials before statistical analysis. All steps were completed using FieldTrip Version 2020 (Oostenveld et al., 2011) in MATLAB Version R2020b.

CBS computation

As previously noted and due to the exploratory nature of our analysis, to avoid a priori choices of time points, sensors, or frequencies of interest for the analysis of gamma activity across the time and frequency spectra, while controlling for multiple comparisons inherently present in multidimensional EEG data, we applied the CBS (Maris & Oostenveld, 2007). More in depth, for every sample (i.e., triplets composed by channel, frequency, and time points across averaged epochs) experimental group pairs were compared by means of a *t* value to create pairwise group contrast matrices. Selected samples were then clustered in connected sets on the basis of temporal, spatial, and spectral adjacency, and cluster-level statistics were calculated by taking the sum of the *t* values for every cluster. Neighboring sensors were identified through a triangulation algorithm, thus irrespectively of their relative distance, based on their two-dimensional layout. A minimum of two channel clusters were selected to address focal activity without affecting the false alarm rate. The significance probability for the CBS was then calculated via the Monte Carlo method. This is due to the fact that the reference distribution for a permutation test can be closely approximated using the Monte Carlo method with any desired level of accuracy. Thus, we followed the following steps. Because ours was a between-participants design, participants belonging to different groups were randomly sorted in single sets; participants were then randomly drawn from combined samples and switched positions across partitions. This led to the creation of random partitions. The independent samples test statistic (i.e., the maximum of the cluster-level summed *t* values) was then calculated on such random partitions. The computation of the Monte Carlo approximation was set on 1000 random draws; thus, the number of random permutations needed to construct the histogram of the test statistics was set to be 1000, a sufficiently large number to ensure approximation accuracy given our data dimensions. The *p* values were the proportion of random partitions that resulted in a larger statistic than the observed one. A *p* value was extracted for each cluster. The focus of our analysis regarded the average of activity across tone triplets and differences across groups, and as such we applied the between-group version of the CBS. All steps were completed using FieldTrip.

Results

Time–frequency decomposition

The time–frequency decompositions across HL-ASD, HL-DLD, and TD infants are visualized in Fig. 2. Although the HL-ASD group seems to show an overall persistent gamma activation across the whole epoch, the figure well-represents how, across the first 450 ms after tone pairs onset, gamma activity appears to be fairly similar across the three groups, with a substantial decrease becoming apparent after the first 200 ms. Crucially, in the HL-ASD group gamma activity persists after 450 ms, whereas it further decreases in the TD group.

CBS results

Statistical analyses showed a significant difference between HL-ASD and TD infants, t_{sum} = 5283.2, p = .034, 95% confidence interval = .0112; because the analysis was performed comparing the HL-ASD against the TD group through the sum of t values across clusters, this corresponds to a positive cluster in the observed data in which activity in the 38- to 80-Hz gamma range was increased in HL-ASD infants compared with TD infants. This significant cluster emerged 450 ms after stimulus onset and lasted until 580 ms, involving frontal, temporal, and parietal regions. The effect size, calculated using Cohen's *d,* was.255 for the difference between the HL-ASD and TD groups. Fig. 3 depicts the time and spatial extent of the significant positive cluster.

Results did not highlight significant clusters differentiating HL-DLD infants from their TD and HL-ASD peers.

Discussion

Our results show the presence of a significant difference in auditory gamma activity in 12-month-old HL-ASD infants compared with TD infants. This difference corresponded to a cluster in the observed data beginning 450 ms and ending 580 ms after stimulus onset across frontal, temporal, and parietal regions. Although this analysis only allows us to reject the null hypothesis of no observed differences across groups (Sassenhagen & Draschkow, 2019), it is still interesting to note that this observation is consistent with prior evidence of a significant advantage in processing low-level auditory stimuli in ASD, suggesting an overall enhanced sensitivity to simple acoustic features (Riva et al., 2018). Moreover, the observed differences are particularly noteworthy in that they consistently reflect variations in auditory responses, with our samples carefully matched on several factors, including SES. In this context, enhanced auditory processing may arise from a dysregulated sensory profile, characterized by hypersensitivity, related to ASD symptoms (Guiraud et al., 2011; O'Connor, 2012; Riva et al., 2018). Sensory hypersensitivity in ASD may then be explained by a dysfunctional arousal system impairing the ability to regulate responses (Orekhova & Stroganova, 2014; Stroganova et al., 2013). Indeed, atypical arousal is reportedly already present in individuals with ASD in their infancy (Orekhova & Stroganova, 2014), and an atypical balance of excitatory and inhibitory activity within brain circuits is a common feature of ASD (Johnson, 2017).

One of the functions of inhibitory processing is to increase the signal-to-noise ratio by resetting spontaneous neural firing not directly linked to the perceived stimulus (Johnson, 2017). In this context, increases in gamma power may reflect an over-recruitment of local networks oscillating in the gamma range (Ethridge et al., 2016), impairing the binding of salient stimuli properties (Casanova et al., 2020). Finally, gamma oscillation abnormalities in ASD are reportedly more apparent in areas of the brain with complex connectivity topologies (Casanova et al., 2020). Our results may support this hypothesis given that the significant cluster extended to frontal areas, known to be highly interconnected (Alzu'bi et al., 2017). Taken together these results suggest that gamma-band-generating mechanisms may be altered, at least for auditory responses, in ASD. Then, enhanced neural responses to auditory stimuli may result in impairments in synaptic integration in ASD that not only could affect listening but also may affect the downstream integration of auditory information in other areas of cor-

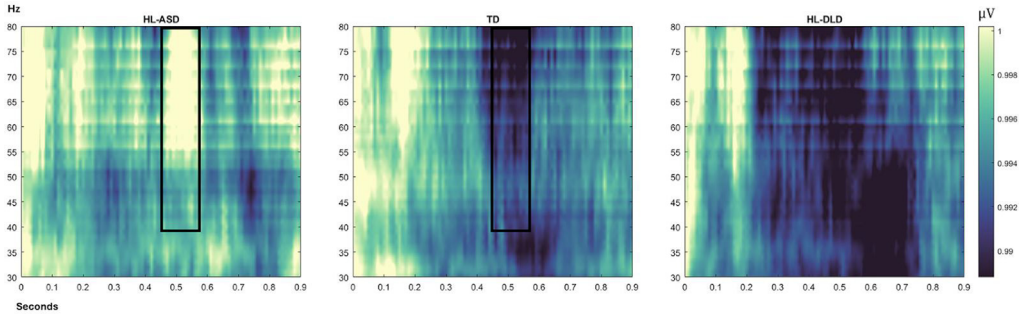

Fig. 2. Time and frequency representation of gamma activity across groups. The x-axis represents time points, and the y-axis represents frequency ranges. The color map represents the power of activity (in μV). Rectangles delimit the extent of the significant cluster across time and frequency ranges. These plots were generated through in-house MATLAB scripts. HL-ASD, high likelihood of developing autism spectrum disorder; TD, typically developing; HL-DLD, high likelihood of developing developmental language disorder. (For interpretation of the reference to color in this figure legend, the reader is referred to the Web version of this article.).

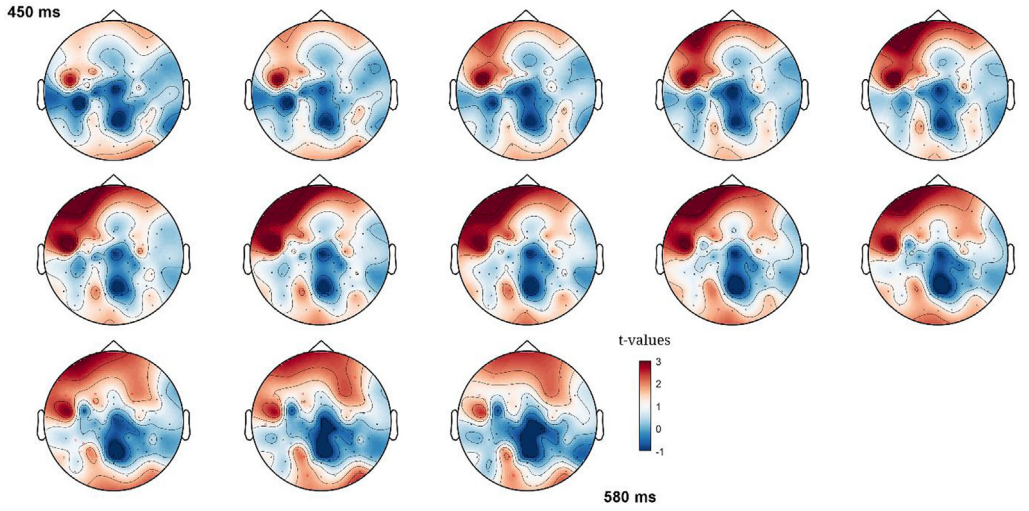

Fig. 3. Time and spatial extents of the significant positive cluster differentiating high likelihood of developing autism spectrum disorder infants from typically developing infants. Red hues represent positive *t* values across clusters, and blue hues represent negative *t* values. Gamma range values were averaged for visualization purposes. Significant channels per time point are signaled in red. These plots were generated through the FieldTrip *ft_topoplotTFR* function. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

tex (Edgar et al., 2015; Riva et al., 2018). Thus, higher-order impairments in domains such as language may become more apparent as stimuli complexity increases (O'Connor, 2012). Moreover, the fact that language is learned predominantly in social context in infancy could further impair the ability to tune to complex linguistic stimuli for infants who later will be diagnosed with ASD (O'Connor, 2012). Given the centrality of both sensory and social dysfunctions in the diagnosis of ASD, further studies aimed at disentangling the role of perceptual complexity from social complexity are extremely needed. Finally, this line of thinking is consistent with the involvement of top-down brain mechanisms across frontal and temporal areas in infants (Polver et al., 2023). Such feedback mechanisms are reportedly the necessary building blocks for higher-order functions such as language processing (Friederici, 2012), and impaired top-down modulation is widely recognized as a core dysfunction in ASD (Pellicano, 2013).

Proceeding further, the lack of significant clusters differentiating HL-DLD infants was an unexpected result given that we anticipated that low-level auditory processing would still be related to the impairments observed in this group (Corriveau et al., 2007; Cumming et al., 2015). Possible explanations are twofold. First, HL-DLD infants may be characterized by preserved auditory abilities and may show impairments only in linguistic tasks, suggesting the presence of a specific and higher-order linguistic impairment. This is partly supported by results from our group suggesting specific impairments in HL-DLD infants in response to complex tones varying in duration or frequency, developed to mimic language characteristics (Cantiani et al., 2016, 2019) and in response to words (Cantiani et al., 2017). In addition, other studies have reported similar impairments in the encoding of temporal modulation in speech in the gamma range (Goswami et al., 2016). Second, given that the CBS is exploratory in nature, the subtly impaired auditory profile of HL-DLD infants could have gone undetected. Studies capitalizing on our results but addressing the question through different and fine-grained analytical techniques could help to answer this question.

In conclusion, our findings indicate that HL-ASD infants are characterized by neurophysiological abnormalities in auditory responses (Riva et al., 2018), suggesting gamma oscillations as a potential early marker of sensory impairments. On the contrary, HL-DLD infants show preserved low-level auditory processing, which may indicate a specific impairment in the processing of linguistic stimuli in this group. A limit of our study regards the limited spatial and temporal inferences we could draw. Future studies could fill this gap by applying source reconstruction techniques on time series to better assess the role played by auditory cortices (Polver et al., 2023). Moreover, interesting insights could derive from comparing HL-ASD and HL-DLD infants who did or did not develop the full-blown disorder. In this sense, differences in the brain activations of these subgroups could shed light on the risk and protective factors. Nonetheless, our results provide a first step in this sense. Indeed, by differentiating brain activities across groups that could develop similar impairments, we can advance in the search of early biomarkers and further inform early interventions.

CRediT authorship contribution statement

Silvia Polver: Writing – original draft, Visualization, Software, Methodology, Formal analysis. **Chiara Cantiani:** Writing – review & editing, Supervision. **Hermann Bulf:** Writing – review & editing, Supervision. **Caterina Piazza:** Writing – review & editing, Validation. **Chiara Turati:** Writing – review & editing, Supervision. **Massimo Molteni:** Resources, Project administration, Funding acquisition. **Valentina Riva:** Writing – review & editing, Supervision, Resources, Project administration, Investigation, Funding acquisition, Data curation, Conceptualization.

Data availability

Data are available on request to the corresponding author due to privacy and ethical restrictions.

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