Auditory Mismatch Responses and Speech Perception Development

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Auditory Mismatch Responses (MMR), as the Mismatch Negativity (MMN) and Late Discriminative Negativity (LDN), change from birth to adulthood, reflecting auditory and brain maturation and language experience. Throughout development, the MMR can present a different polarity, latency, amplitude, and scalp distribution than the adult MMN/LDN. In addition, conventional ERP analysis can be complemented with time–frequency analysis (TF) to preserver non-stimulus locked neural activity, which is abundant in paediatric EEG. Although TF analysis is less popular in the MMN research field, it may help further our understanding of how speech perception develops in early childhood and in clinical populations in particular, as children with Developmental Language Disorder (DLD).

time–frequency analysis | mismatch responses (MMRs) | Late Discriminative Negativity (LDN) | Inter-trial Coherence | Event-related spectral perturbation

1. Introduction

Between the ages of three and six years, children can understand spoken language effortlessly. However, preschoolers do not perceive speech as efficiently as adults do, and there may also be differences between children of the same age who have different language skills, for example, children with typical language development (TLD) versus those with Developmental Language Disorder (DLD). DLD, previously referred to as Specific Language Impairment (SLI), affects around 7% of children and is characterised by a range of language deficits, especially in morphosyntactic and phonological processing, without an identifiable medical cause [1]. The underlying causes of DLD are still largely unknown, but many studies suggest that children with language difficulties process speech differently from their typically developing peers [2][3].

However, the neural mechanisms underlying speech processing in young children are not well understood. Despite plenty of behavioural and brain imaging research, scholars do not know yet what cortical patterns of speech perception are typical at preschool age or whether these patterns vary between children and adults or between children with TLD and DLD [4]. Understanding this is important because early childhood is a period of significant language growth, and spoken language perception is critical for children's later communication and literacy development. It is also during early childhood when many children with language disorders are first diagnosed, suggesting that some language symptoms could become more apparent at this age. Therefore, identifying neural patterns associated with typical and atypical speech processing in children could aid the development of objective

clinical measures, fostering the earlier detection of and intervention in language disorders. Importantly, this would require determining adult reference values first, representing the neurotypical, mature cortical speech-processing patterns against which to compare children's responses.

As for the role of speech perception deficits in DLD, different hypotheses have pointed out that specific problems with processing speech sounds could be an underlying marker of this disorder [5][6]. However, there is no clarity about the nature of these difficulties. Some early theories of DLD state that low-level speech perception deficits are a hallmark of DLD. For example, difficulties in detecting rapid acoustic changes within speech sounds or extracting distinctive acoustic cues from the speech stream could lead to unstable phonological representations, which then manifest as phonological deficits, such as difficulties in repeating non-words (see [7] for a review). Other accounts have proposed that speech perception deficits in DLD could be a consequence and not a cause of language difficulties [8], where less efficient speech processing in DLD derives from limited language skills, and not vice versa. For example, poorer phonological processing skills in DLD than in TLD may affect children's ability to detect subtle acoustic changes in speech sounds but not in other sound contrasts [9]. Notably, several studies have failed to detect any evidence of atypical speech processing in DLD [10], suggesting that either these deficits do not exist or they cannot be detected with the current methods. So far, neurobiological findings are contradictory, making it difficult to identify any neural markers of speech perception deficits in DLD.

An advantageous method to investigate the brain processing of complex, rapidly changing acoustic signals, such as speech, is the electroencephalogram (EEG) because of its high temporal resolution. From the EEG, it is possible to extract event-related potential (ERP) components, such as the Mismatch Negativity (MMN), which has been extensively used across a wide range of ages and populations [11][12], and the less-studied Late Discriminative Negativity (LDN). Unlike early auditory detection responses, such as the P50/P1/N1 components, the MMN and LDN reflect cortical sound discrimination, a process that occurs later in the brain, at fixed latencies (time-locked), after a change in auditory stimulation. In adults, the auditory MMN is a negative deflection of about 0.5–5 μV that occurs 100–250 ms after a discriminable change in any acoustic feature [12]. The LDN appears later (250–400 ms) and seems to be more pronounced in children than in adults [13] and for auditory contrasts involving speech rather than nonspeech sounds [14], for which some studies consider it a signature of sound processing complexity [15]. In infants and young children, the MMN and LDN may present an immature form, so mismatch response (MMR) is a generic term to refer to these responses deviating from the adult-like pattern [16]. Like the adult MMN/LDN, the MMR reflects the brain's sensitivity to physical and abstract changes ("deviants") in a sequence of regular sounds ("standards") in many acoustic contrasts, including speech. The MMR/MMN/LDN can be elicited during unattended listening, making it a valuable measure when behavioural responses are not possible or are less reliable, as in young children or clinical groups [12].

In speech perception research, the MMR/MMN/LDN is used as a pre-attentive discrimination index of general auditory processing elicited by low-level, physical changes (i.e., in pitch, duration, or intensity) but also of speech-specific processing elicited by changes in higher-level, abstract linguistic representations (e.g., phonemic categories, lexical status, or word classes). Thus, the MMR/MMN/LDN not only depends on the bottom-up (afferent) processing of the speech acoustic features but is also influenced in a top-down manner by

psycholinguistic factors, such as the listener's knowledge of phonological categories and structures, as well as the words' grammatical function, distributional frequency, and meaning [17]. Evidence from adults indicates that language-specific top-down mechanisms selectively facilitate the processing of speech [18], developing gradually during childhood from around the age of 7 years [19]. However, there is little evidence about how top-down modulations of speech perception operate at different stages of language development or whether they are impaired in children with language difficulties. Considering several studies indicating reduced or slower MMR/MMN/LDN in DLD (see [20] for a review), it is possible that the MMR/MMN/LDN response could serve as a neural marker of speech-specific difficulties in DLD.

One potential factor contributing to this gap in knowledge about the underlying nature and cause of DLD is the methodological complexity of conducting MMR research in young children (especially in clinical populations) and comparing groups of children and adults [21]. Children are less able to tolerate long, repetitive testing sessions and may become fussy, introducing movement-related artefacts in the EEG. There are also neuroanatomical differences between adults and children (e.g., in head size, skull and cortical thickness, cortical fibre density) that complicate a direct comparison of their ERP responses [22]. In general, adults' ERPs are intrinsically smaller and more consistent in timing than those of children, which are larger and much more variable [23]. Thus, when studying speech perception development across broad age ranges, it would be appropriate to complement conventional time-domain measures (e.g., amplitude and latency) with time–frequency measures (e.g., changes in spectral power or phase coherence over time). This would allow the measurement of important cortical oscillatory activity that is not consistently time-locked to the stimulus and would be otherwise lost in ERP averaging [24].

2. MMR/MMN/LDN in Speech Perception Development

Multiple studies have shown that the MMR changes from birth to adulthood, reflecting auditory and brain maturation [16][25][26]. Throughout development, the MMR can present a different polarity, latency, amplitude, and scalp distribution than the adult MMN/LDN. In infants and young children, the MMR polarity is usually reversed towards positive values [27], with some studies reporting positive MMRs until the age of 6–7 years [28][29]. Others, however, report negative, MMN-like responses much earlier, i.e., in the first six months of life [30]. MMR scalp localisation is more broadly distributed in children than the adult MMN/LDN, which is more focalised and shows the maximum amplitude at the frontocentral electrodes (e.g., Fz, Cz) [31][32].

Regarding temporal patterns, the MMR latency correlates negatively with age during infancy and childhood, with delayed and longer responses in young children than in adults [16][23]. In infants between 7 and 11 months, MMRs have been reported with a latency of 250–500 ms [33]. In three year old children, there is evidence of the MMR peaking between 120 and 400 ms, whereas, for 5–8-year-olds, peak latencies occurred between 190 and 270 ms, a more adult-like range [28]. Shafer et al. [34] observed similar latencies using narrower age bands, with later and longer MMRs in 4–5 than in 6–7-year-old children, whereas Bishop et al. [13] reported similar latencies in children (age: 7–12 years) and adolescents (age: 13–16 years), in both cases slower than in adults. On the contrary, some studies indicate adult-like latencies much earlier, for example, in children between 6 and 13 years [35]. The MMR amplitude, however, seems not to follow a linear trajectory but a U-shaped curve during development [25]. Adult-like

amplitudes are often observed in infants, with significantly smaller responses in early childhood (until around 7–8 years) until late childhood (12–13 years), followed by an increase in amplitudes until late adolescence (16–18 years). Paquette et al. [27] reported smaller MMRs in children (age: 3–13 years) than in adults and in younger (age 3–7 years) than in older children (age 8–13 years).

Another aspect to consider is that, like the adult MMN/LDN, children's MMR patterns reflect the acoustic and linguistic content (e.g., phonological, lexical, semantic information) in the speech input. Thus, there are differences in the MMR elicited by speech versus other nonspeech sounds [13][27][35] and for speech involving different linguistic contrasts. For example, some studies have reported larger MMRs to speech (syllables) than for acoustically matched nonspeech sounds in infants [36] and 6-year-old children [37]. However, in adults, other studies indicate the opposite: a smaller MMN for speech stimuli than for their acoustically matched nonspeech counterparts [38]. For speech stimuli, MMRs show different age-related trajectories for different linguistic features, such as native versus non-native phonemic contrasts [39], word versus non-words, or even distinctions between different word classes [40]. MMRs to non-native phonemic contrasts have been reported at the age of 7 months disappearing by the age of 11 months, whereas the MMR to native phonemes becomes more robust during the same period [33]. For native phonemes, Finnish children at age three years show MMN-like responses in the 300–400 ms range for vowel contrasts [41], whereas French-speaking children of the same age show adult-like MMNs, peaking at 270 ms for syllables with initial consonant contrasts (/bag/versus/da/) [27].

MMRs are also modulated by the linguistic context [40], for example, by the type of syllable or word in which speech sounds are presented. David et al. [14] investigated the discrimination of syllables with different phonological complexities, reporting smaller MMN-like but larger LDN-like responses in children (age: 6–10 years) than in adults for more complex syllables, but no difference between adults and children for less complex ones. Other studies report MMR enhancement when the deviant syllable occurs in a word compared to when it occurs in isolation or in a non-word, which has been linked to top-down lexical or semantic modulations. In 3-year-old children, Strotseva-Feinschmidt et al. [42] reported the effects of the word's lexical frequency on the presence or absence of MMRs to two German function words (articles *der*/*den*). They found that the high-frequency article *der* elicited an MMN-like and an LDN-like response. In contrast, the low-frequency *den* elicited only an LDN, suggesting easier processing of higher-frequency words. In adults, there is evidence of larger MMNs for words than for non-words, indicating an enhancement of the MMN amplitude by the lexical-semantic content of the stimulus [17][43].

In children with DLD, cortical speech processing and its MMR signatures are less well understood, especially when compared with other neurodevelopmental disorders, such as developmental dyslexia or autism [44]. Overall, compared to TD children, children with DLD show poorer and slower cortical discrimination of speech sounds, resulting in smaller MMN amplitudes, delayed latencies, atypical scalp distributions, and less left-hemisphere lateralisation than TLD children (for a review, see [20]).

With regard to the debate concerning the underlying causes of DLD, several studies suggest a connection between the MMR/MMN/LDN and language skills, both in children with TLD and DLD. For example, studies in infants aged 7.5 to 24 months have reported a positive correlation between the MMR amplitude for native phonemic contrasts

and behavioural phoneme discrimination measures [33][45]. Furthermore, Linnavalli et al. [46] demonstrated that children (age 5–6 years) with better phoneme discrimination performance showed larger MMRs than those with poorer behavioural results. Similarly, a study in preschoolers (mean age: 5.6 years) by Norton et al. [47] found significantly larger MMRs in the late time window (300–500 ms) for/ba/-/da/contrasts in children with typical phonological awareness (PA) skills than in those with low PA skills. In 2-month-old infants at risk for DLD, Friederich et al. [48] found delayed MMRs to vowel deviants with different durations. Overall, the findings indicate that the MMR amplitude correlates with language abilities in children with DLD, with weaker or slower MMRs associated with poorer language outcomes (see [20] for a review). More specifically, the evidence indicates a reduced amplitude, particularly over the left scalp areas, and delayed latency in infants and children at familial risk for language deficits or with a DLD diagnosis.

The MMR may also predict children's receptive language skills at later ages [5]. Guttorm et al. [49] found that the MMN measured in infants 1–6 days after birth with and without a risk of developmental dyslexia predicted pre-reading language skills at the age of five years. Specifically, positive MMRs in the right hemisphere were associated with lower phonological, rapid naming, and letter knowledge skills. However, other studies show no relationship between the MMR and behavioural language measures [50], especially at the individual level [10].

In sum, the evidence indicates that MMR patterns in children change as a function of age, which suggests the ongoing maturation of cortical speech processing until later childhood. In addition to this, there is also evidence of different developmental rates for different linguistic features at the cortex. Furthermore, the associations between MMRs to speech sounds and later language skills suggest that the MMR could be a valuable tool for predicting language outcomes in children with DLD.

3. MMR Time–Frequency Analysis and Speech Perception Development

Although time–frequency analysis (TF) is less popular than conventional ERP analysis in the MMN research field, it may help further our understanding of how speech perception develops in early childhood and in clinical populations in particular. TF analysis measures the non-stimulus-locked neural oscillatory activity that is abundant in children and cancelled out by the ERP technique [51]. Moreover, TF analysis increases the ERP signal-to-noise ratio (SNR) [52], which is an advantage when dealing with noisy data (as is often the case in children's EEG), making it more dependable than time-domain measures for MMR/MMN/LDN identification [50].

In adults, studies using TF analysis of the MMN response have found increased neural synchronisation in the theta frequency range (4 to 7–8 Hz) for deviants vs. standard sounds, for example, during the discrimination of sound duration contrasts. Fuentemilla et al. [53] found greater theta inter-trial phase coherence (ITPC) at the temporal and frontal electrode sites and event-related spectral perturbation (ERSP) for deviant (1000 Hz, 25 ms duration) than for standard (1000 Hz, 75 ms) tones at the frontal electrodes. Similarly, two studies in adults by Hsiao and colleagues [54][55] showed larger theta-phase-locking values (PLVs) and spectral power for duration deviants (1000 Hz, 50 ms duration) than for standards (1000 Hz, 100 ms duration). Bishop at al. [13] reported no changes in ERSP

power but a significant increase in theta ITPC during MMN generation, which they considered an index of event-related oscillatory phase resetting. Although these findings indicate a role for increased theta ITPC in auditory deviance detection and the generation of the MMN [50], most of them were elicited by nonspeech stimuli, so it is unclear whether they can be generalised to speech sound processing.

In paediatric research, the few studies using TF analysis of the MMN/MMR suggest a relationship between increased stimulus-induced phase synchronisation and developmental changes in auditory perception. A longitudinal study by Bishop et al. [56] showed that between the ages of 7 and 11 years, there was an increase in theta ITPC for tone deviants in the frontal–central but not in the temporal regions, indicating the greater involvement of areas related to top-down modulations as children grow up. Studies by Müller et al. [57] and Poulsen et al. [58] reported that greater theta phase synchrony for deviants than for standards was present in children, and it increased from childhood to early adolescence, indicating more efficient sound detection. Bishop et al. [13] observed age-related increases in theta phase synchrony for deviant sounds, with the largest ITPC for adults (35–56 years) and larger ITPC for adolescents (13–16 years) than for children (7–12 years). Together, these findings indicate that the maturation of the MMN neural substrates is accompanied by age-related increases in oscillatory synchronisation, mainly in the theta range and frontal cortical regions, suggesting more consistent neural responses and more involvement of areas involved in top-down processing.

Notably, some renowned infant studies using speech stimuli suggest that age-related increases in spectral power and ITPC in the delta, theta, and gamma bands between the ages of 6 and 12 months may reflect selective enhancement and perceptual narrowing for native-language phonemes [59][60]. Moreover, in adults, theta synchronisation is thought to play a critical role in syllabic segmentation [61]. However, there is little research on the developmental trajectories of different theta-band measures. So far, there is little information about how theta brain activity is related to the linguistic content of the speech stimuli or to the language skills in children with TLD and DLD.

Cortical oscillatory dynamics have been far less investigated in DLD than in other neurodevelopmental disorders, such as developmental dyslexia or autism spectrum disorder (for a review, see [51]). However, there is some evidence indicating that atypical oscillatory activity may underlie language disorders [44]. Bishop et al. [62] compared the cortical discrimination of tones and speech sounds in children (7 to 16 years old) with DLD and TLD, measuring low-frequency-band synchronisation in the MMN/LDN intervals. Even though they found no between-group differences in the MMN, the TLD (but not the DLD) group had a significant drop in power in the LDN versus the MMN interval for the low-frequency bands (delta, theta, and alpha). The authors suggested that this lack of event-related desynchronisation in the DLD group after the MMN indicates an inability to disengage neural activity after initially “normal” auditory discrimination responses [62]. Two other studies by Heim et al. [63][64] examined oscillatory dynamics during rapid auditory processing of tone pairs in children between 6 and 9 years with and without language disorders. In the language-impaired group, they found that atypical early processing (45–75 ms) significantly reduced the gamma (29–52 Hz range) amplitude and phase-locking values. The authors interpreted these findings as evidence of altered oscillatory timing in language-impaired children when processing rapid

sequences of tones. Again, they used nonspeech or simple speech stimuli, making it hard to draw conclusions about how the linguistic content of the stimuli might modulate the brain responses.

In conclusion, the MMR is a valuable tool for investigating the neural mechanisms underlying speech processing in children. In typically developing children, the MMR has an identifiable developmental trajectory, and it indicates sensitivity to speech contrasts, positively correlating with later language development. In children with DLD, the MMR has shown reduced amplitudes and delayed latencies, suggesting difficulties in processing the acoustic features of speech sounds. However, only a few studies have exploited the advantages of MMR time–frequency measures to characterise children's responses to speech sounds and conduct comparisons between adults and children.

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