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Maturation of speech-elicited event-related potentials from birth to 28 months: Typical development with minor effects of dyslexia risk

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ABSTRACT

This study investigated in a longitudinal setting the maturation of obligatory auditory event-related potentials (ERPs) during infancy and early childhood, comparing the developmental patterns in infants with or without a familial risk of dyslexia. To that end, we recorded ERPs to repetitive speech sounds at birth, 6 months, and 28 months in a sample of ~200 children over-represented by children at risk for developmental dyslexia. Additionally, we assessed the impact of a music listening intervention on these ERPs. We found that infant P1 and N2 are the most robust ERPs during the first years of life. A distinct, broad infant P1 response was observable already at birth, with a infant P1-N2 pattern emerging by 6 months. Infant P1 amplitude increased from birth to 6 months, whereas its latency decreased from birth to 28 months. Infant N2 latency increased between 6 and 28 months, with no significant changes in amplitude. The control group without familial dyslexia risk exhibited smaller infant N2 amplitudes than the at-risk group at 6 months. No effects of the infant music listening intervention on the ERPs were seen. These results, with a large sample size and longitudinal setting, reflect auditory development, serving as a reference for future studies including clinical groups.

1. Introduction

Measuring auditory event-related potentials (ERPs) is an established method to study the emerging auditory processing and its experience- and maturation-related changes from birth onwards (Alho et al., 1990, or even in fetuses, Huotilainen et al., 2005; for a review, see Kushnerenko et al., 2013). In addition, ERPs have been found to reflect auditory and speech processing deficits in neurodevelopmental conditions such as dyslexia and its risk in infancy (e.g., Thiede et al., 2019; Virtala et al., 2022; for a review, Volkmer & Schulte-Körne, 2018). In the last decades, there has been extensive research on auditory change detection reflected by mismatch responses (Näätänen et al., 2019), but less attention has been directed to sound encoding reflected by the so-called obligatory ERPs to repetitive auditory stimuli. Yet, investigating obligatory ERPs during the first years of life appears promising, as they typically exhibit better data quality and have been linked with language development in infants (Kailaheimo-Lönnqvist et al., 2020; Leppänen et al., 2010; Navarrete-Arroyo et al., 2024a) and older children (Cantiani et al., 2016; Hämäläinen et al., 2018; Lohvansuu et al., 2018). Furthermore, these ERPs have shown potential for reflecting auditory learning resulting from exposure to sounds in both infants (Partanen et al., 2013; Trainor, 2012) and adults (Seppänen et al., 2012; Tremblay et al., 2010). The present study aims to describe the maturation of speech-sound elicited obligatory ERPs in infancy and early childhood in the large longitudinal DyslexiaBaby sample. We also investigate the effects of familial dyslexia risk and a music listening intervention on the responses obtained.

1.1. Obligatory ERPs and their maturation during the first years of life

In adults, obligatory auditory ERPs can be recorded by presenting repetitive sounds, which elicit four peaks termed P1, N1, P2, and N2, observed in fronto-central scalp sites (Ponton et al., 2002; Shafer et al., 2015, for review, see Csibra et al., 2008). Based on cross-sectional

Abbreviations: ERP, event-related potential; EEG, electroencephalogram; ICA, independent component analysis; ROI, region of interest; LGCM, latent growth curve model.

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evidence, in infants and young children the N1 is not yet discernible, and the N2 is considerably larger in amplitude than in teenagers or adults (Ceponiene et al., 2002; Csibra et al., 2008; Ponton et al., 2002; Sussman et al., 2008). Concerning their functional significance, the P1 has been suggested to reflect an early stage of stimulus detection and registration that enables the formation of sound representations in both adults and children (Ceponiene et al., 2002, 2005, 2008). The N2, in turn, is thought to index the building up of neural representations and the processing of complex aspects of acoustic stimuli, such as frequency or intensity (Ceponiene et al., 2002, 2008; Choudhury & Benasich, 2011). In addition, magnetoencephalography (MEG) studies have identified corresponding components, often referred to as P1m and N2m, which share similar functional significance (Gage et al., 2003; Ruhnau et al., 2011). Source localization in these studies has associated the P1m with the activity of the primary auditory cortex, while the N2m has been linked to more distributed sources including the superior temporal gyrus and areas involved in attentional and cognitive control. These source findings support interpretations of the P1 and N2 as reflecting early sensory encoding and higher-order processing, respectively. Notably, the P1–N2 response pattern has been observed in infants and young children in response to both speech (Molfese, 2000; Shafer et al., 2015; van Herten et al., 2008) and non-speech stimuli (Choudhury & Benasich, 2011; Kushnerenko et al., 2002; Ponton et al., 2002; Wunderlich et al., 2006). This suggests that these ERP components reflect fundamental auditory processing of acoustic stimuli, encompassing but not limited to speech.

Studies reflecting the maturation of obligatory ERPs from childhood to adulthood have shown that the P1 and N2 seem to reach their maximum amplitudes in early childhood (6–8 years of age), and then diminish from 9–10 years onwards, with the N2 becoming almost absent in adults (Ponton et al., 2002; Sussman et al., 2008). These responses also decline in peak latencies with increasing age (Ponton et al., 2002; Sussman et al., 2008; Wunderlich & Cone-Wesson, 2006). The N1, in turn, starts to emerge during early childhood, but becomes discernible only between 8 and 12 years of age, gradually increasing in amplitude until becoming the dominant negative component in adulthood (Ruhnau et al., 2011; Wunderlich & Cone-Wesson, 2006).

Focusing specifically on the maturation of these ERPs in infancy and early childhood, there are only three longitudinal studies (Choudhury & Benasich, 2011; Kushnerenko et al., 2002; Shafer et al., 2015), while some others have used cross-sectional designs (Little et al., 1999; Silva et al., 2017; Wunderlich et al., 2006; for reviews, see Wunderlich & Cone-Wesson, 2006). Overall, these studies report that the newborn ERP complex already displays a clearly identifiable infant P1, while infant N2 is not always observed (Kushnerenko et al., 2002; Wunderlich et al., 2006). Both responses were shown to increase in amplitude and decrease in latency during the first year of life, resulting in a clear P1-N2 complex between 6 and 12 months (Choudhury & Benasich, 2011; Kushnerenko et al., 2002; Shafer et al., 2015; Silva et al., 2017). Between 12 and 36 months of age, the infant P1 continues growing in amplitude and decreasing in latency (Choudhury & Benasich, 2011; Shafer et al., 2015; Silva et al., 2017; Wunderlich et al., 2006), whereas the reported findings for the infant N2 are rather inconsistent across studies. Additionally, some studies have reported a P2 component emerging during infancy, peaking earlier with increasing age and decreasing the most during the first months of life (Choudhury & Benasich, 2011; Shafer et al., 2015; Wunderlich & Cone-Wesson, 2006). However, it is important to note that determining whether infantile peaks are directly analogous to adult ERP peaks remains challenging (Kushnerenko et al., 2002).

Whereas the earlier studies provided valuable information on obligatory ERPs in various ages, longitudinal studies using the same paradigm in studying the same children are still scarce. These studies also focus on the development from 3–6 months of age, there being, to our knowledge, only one study reporting newborn data (Kushnerenko et al., 2002). Moreover, the sample sizes were rather low, with more than 19 participants only in Choudhury and Benasich (2011), which followed a sample of 45 infants. Ensuring large sample sizes is especially important in older infants and toddlers, where a lot of movement- and vocalization-related disturbances in the EEG data are expected. Given this, there is a need for longitudinal studies on the morphology and maturation of the obligatory ERPs to speech sounds during the first years of life. This is further highlighted by the importance of an efficient neural speech encoding to support language acquisition (Kuhl, 2004; Kujala et al., 2023), as well as by the reported associations of these ERPs with later language and reading abilities (Cantiani et al., 2016; Choudhury & Benasich, 2011; Guttorm et al., 2010; Kuuluvainen et al., 2016; Leppänen et al., 2010). In the longitudinal DyslexiaBaby sample, larger amplitude of the infant P1 at 6 months was related to better development of pre-linguistic skills during the following 6 months (Kailaheimo-Lönnqvist et al., 2020). In addition, we found that larger amplitudes and shorter latencies of the infant P1, as well as left-lateralized infant P1 and N2, were associated with better pre-reading skills development between 28 months and 4–5 years of age (Navarrete-Arroyo et al., 2024b). In light of these findings, describing the maturation of the obligatory ERPs during infancy and early childhood in the large DyslexiaBaby sample may serve as a reference for future investigations on the effects of interventions and developmental conditions on infant auditory processing and language development.

1.2. Obligatory ERPs and deficient neural speech encoding in dyslexia

Developmental dyslexia, the most prevalent learning disorder, is understood to primarily originate from a deficit in phonological processing in which the ability to acquire and process the native language phonemes is affected (Eden et al., 2016; Peterson & Pennington, 2015; Vellutino et al., 2004). For example, speech-elicited mismatch responses have been found to be diminished (Choudhury & Benasich, 2011; Thiede et al., 2019; van Leeuwen et al., 2006; Virtala et al., 2022) or atypically lateralized (Leppänen et al., 2002; Thiede et al., 2019; van Leeuwen et al., 2006) in children at familial risk for dyslexia (typically due to a dyslexic biological parent), thus reflecting a deficit in auditory discrimination abilities and, specifically, in speech processing. However, studies assessing the impact of dyslexia risk on the maturation of the infant obligatory P1 and N2 components, reflecting the registration and initial processing of speech-sound representations, are scarce.

The infant P1 has been found to be diminished to speech and pure tones both in dyslexia-risk infants (van Herten et al., 2008) and school-aged children with dyslexia diagnosis (Bonte et al., 2007; Stefanics et al., 2011). In addition, the N2 elicited by speech sounds has been reported to be atypically lateralized (e.g. larger amplitudes in the right than the left hemisphere) in at-risk infants (Molfese, 2000; van Herten et al., 2008), but not in dyslexic school-aged children (Bonte et al., 2007; Stefanics et al., 2011). Similar results for both the infant P1 and N2 have been found in infants at risk for other developmental conditions, such as developmental language disorder (Benasich et al., 2006; Choudhury & Benasich, 2011), which is often co-morbid with dyslexia (Ramus et al., 2013). However, previous studies were constrained by small sample sizes, involving a maximum of 17 participants in the risk groups. Additionally, there is a notable absence of longitudinal studies following the maturation of obligatory ERPs across infancy and early childhood in dyslexia-risk children. Studies employing this design would be highly valuable, as they could elucidate differences in neural speech encoding development between dyslexia-risk and typically developing children during the first years of life. This may reveal neural deficits that might be related to future language and reading problems.

1.3. Effects of music on neural speech processing

Given the reported associations between language development and familial dyslexia risk with infant neural speech processing, early

interventions supporting language development could be a promising approach to mitigate the impact of early risk factors on later reading difficulties. In this regard, music interventions are especially promising, as they have been shown to improve literacy skills in both normal readers (Chobert et al., 2014; Moreno et al., 2009) and dyslexic children at school age (Flaugnacco et al., 2015). In addition, even mere exposure to music already during infancy and early childhood (Partanen et al., 2022; Putkinen et al., 2014; Trainor, 2012; Virtala et al., 2023), and even before birth (Partanen et al., 2013), has been shown to shape the auditory system, as reflected by ERPs. In our longitudinal DyslexiaBaby study, a music listening intervention was administered between birth and 6 months of age to infants at familial risk of dyslexia (Virtala & Partanen, 2018), thus becoming the first study investigating the potential benefits of music listening in infants at risk for reading disorders. Passive vocal music listening compared to instrumental music listening and no intervention enhanced neural mismatch responses to phonemic and frequency changes in speech sounds (Virtala et al., 2023). However, the effects of this intervention on neural speech encoding, as reflected by obligatory ERPs, have not yet been addressed. Investigating this would improve our understanding of the neural mechanisms by which music listening can support auditory and phonological development in at-risk children.

1.4. Research questions and hypotheses

The present study aimed to determine the typical morphology and maturation of the obligatory auditory ERPs, here termed infant P1 and N2, during infancy and early childhood. To do that, we measured ERPs elicited by repeating speech sounds at birth, 6, and 28 months in the large, longitudinal DyslexiaBaby sample. Secondly, we aim to compare the elicitation, amplitude, latency, hemispheric distribution, and maturation of these ERP responses in subgroups 1) with high or low familial risk for dyslexia and 2) receiving or not receiving one of two music listening interventions in infancy.

Based on the above-reviewed literature, it was hypothesized that a clear positive response (infant P1) would be observable already at birth, and that a positive-negative pattern (P1-N2) would be evident at 6 and 28 months. We expected the infant P1 to become larger and to decrease in latency with increasing age, and the infant N2 to increase in amplitude from 6 to 28 months. Based on previous reports (Kushnerenko et al., 2002; Shafer et al., 2015), the findings for N2 latencies might be more inconsistent. Additionally, based on previous findings suggesting that language processing may be less lateralized in infants compared to adults (Perani et al., 2011), we did not expect the ERPs to show a strong bias toward either hemisphere. Furthermore, given the lack of studies on the maturation of obligatory ERPs in children at-risk for dyslexia during the first years of life, we were unable to formulate concrete hypotheses on the disparities in the maturation of infant P1-N2 responses between the high and low risk groups. However, based on cross-sectional evidence (Molfese, 2000; van Herten et al., 2008), we expected reduced or atypically right-lateralized responses in the risk group. Finally, based on previous results (Partanen et al., 2013; Seppänen et al., 2012; Virtala et al., 2023) we expected that those infants participating in the music intervention, and particularly those engaged in the vocal compared to instrumental music intervention, may exhibit an enhanced neural speech encoding reflected by a more mature (e.g. larger in amplitude or earlier in latency) infant P1-N2 pattern.

2. Methods

2.1. Participants

A total of 210 infant participants were recruited during pregnancy or around birth via social media, local maternity clinics, and via the website of the DyslexiaBaby study. The recruitment during pregnancy was mainly targeted for parents with dyslexia (target-N = 160), but also infants of non-dyslexic parents (target-N = 50) were recruited with the same strategies. The participant selection process, exclusions, and final sample sizes of the whole sample in different parts of the follow-up are described in Fig. 1 and background information of the final samples in Table 1. To be included in the study, infants were required to be born healthy (5-minute Apgar score of 7–10, or, in two infants with an Apgar score of 6 and two without Apgar scores, no indication of health issues in the newborn measurement) and full-term (gestational age at least 37 weeks and birth weight at least 2500 g). Furthermore, they were required to have normal hearing (confirmed with evoked oto-acoustic emissions (EOAE) test at the hospital after birth, or, in two cases with missing information, with a later screening in maternity clinic) and Finnish had to be (one of) their native language(s).

Within the sample, around ¾ of the infants were at risk for dyslexia, i.e., they had one or two biological parents with dyslexia, confirmed by a recent diagnostic statement from a professional, or if missing, by reading test performance in the DyslexiaBaby study. The criteria for the dyslexia diagnosis were below-norm performance of at least one standard deviation (SD) in reading or writing speed or accuracy across a minimum of two out of four subtests. Nevertheless, in some cases parents did not fully meet these criteria, yet in the diagnostic interview they reported clear reading and writing difficulties during childhood, alongside a familial history of dyslexia. The children of these parents were included in the at-risk group of the study, with the parents classified as compensated dyslexics. In addition, $^{2}/_{3}$ of the at-risk children took part in one of two music listening interventions at 0–6 months (vocal music intervention = VOC, and instrumental music intervention = INS in Table 1), while the rest of them did not (no intervention group = NO-INT in Table 1). Infants who had parents without dyslexia (around ¼ of the sample) comprised the control group, and their parents (or one, if the other parent was not available) had to report neither suspected nor diagnosed dyslexia nor other language- or learning-related disorders.

Infants with a parent diagnosed with attention deficit disorder, or with an individualized curriculum in elementary school suggesting broader cognitive deficits, were not included in the DyslexiaBaby study. If these issues came up when they were already enrolled in the present study, they were excluded from it (Fig. 1 “Parental diagnosis”). Another reason for exclusion was the presence of severe health condition of the children that is known to affect their nervous system and language development (e.g. severe dysmorphias, chromosomal abnormalities, Rolandic epilepsy, brain tumors; Fig. 1 “Child’s later diagnosis”). In addition, children with an unsuccessful implementation of the music intervention were excluded from the present study (Fig. 1 “Failed intervention”, for details see Section 2.2.). Furthermore, in different parts of the longitudinal study, issues related to measurement scheduling (Fig. 1 “Scheduling”), suboptimal data quality (Fig. 1 “Poor data quality”) or families dropping out from the study during the first 28 months (Fig. 1 “Family withdrawn”, “Contact lost”) resulted in excluding the data of certain infants from that specific measurement point. These inclusion criteria were applied to the whole sample, including both risk and control groups. At all three measurement points, complete longitudinal data were available for a total of 75 infants.

The infant P1 and N2 maturation was investigated in the whole sample (Table 1), including all children with usable (without artifacts, no more than five bad channels at birth and 6 months, and six at 28 months, a sufficient number of trials; see section 2.4.1) electroencephalographic (EEG) data from at least one measurement point. In order to study possible effects of the music listening interventions on the infant P1 and N2 and their maturation, the three at-risk groups (VOC, INS, and NO-INT groups in Table 1) and control group were additionally compared. Furthermore, to study possible effects of dyslexia risk on the infant P1 and N2 and their maturation in more detail, an additional high-risk subsample was taken from the whole sample (Table 1). The high-risk group consisted of those infants of the at-risk group who did not participate in the music intervention (no-int group in Table 1; as that may affect their ERPs), excluding those with parents classified as

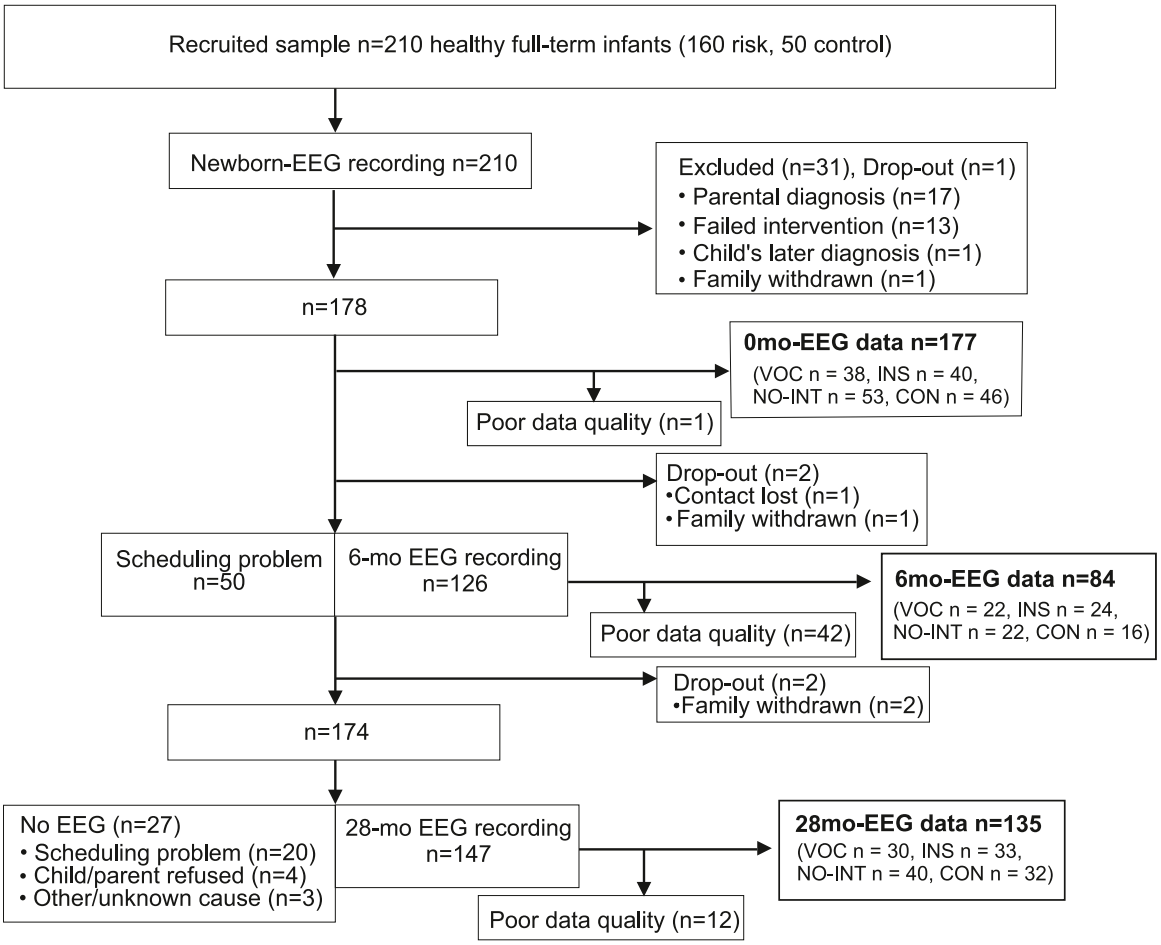

Fig. 1. Description of the participant selection process, drop-outs, and exclusions in the DyslexiaBaby longitudinal electroencephalogram (EEG) data at birth, 6 (6mo), and 28 months (28mo), with final sample sizes of the present study in bold. Adapted from Virtala et al. (2022). VOC = Vocal intervention; INS; Instrumental intervention, NO-INT (no intervention), CON = Control.

Table 1
Background information and sample sizes in the whole sample and in the music intervention (VOC, INS, NO-INT), high-risk (HIGH-RISK) and control (CON) subgroups at birth (Newborn), 6 months (6mo), and 28 months (28mo): amounts of the DyslexiaBaby dyslexia risk and con(trol) participants; gender distributions; Electroencephalogram (EEG) recording age (in days or months), Birth weight (in grams), and Apgar scores at birth. No significant differences were found in background variables.

	Newborn (N = 177)	**VOC (N = 38)**	**INS (N = 40)**	**NO-INT (N = 53)**	**CON (N = 46)**	**HIGH-RISK (N = 38)**
dyslexia risk/ con	131/46					
female/ male	80/97	16/22	21/19	23/30	20/26	14/24
age, days (SD)	9.2 (4.1)	9.5 (3.8)	9.6 (3.5)	9.1 (3.8)	8.9 (5.2)	8.9 (3.7)
Birth weight, g (SD)	3582.5 (452,73)	3619.7 (418.25)	3582.2 (466.96)	3581.3 (397.30)	3553.5 (533.01)	3579.6 (401.76)
Last Apgar (5/10 min)	9.48 (0.66)	9.47 (0.8)	9.40 (0.59)	9.48 (0.70)	9.54 (0.59)	9.42 (0.74)
	6mo (N = 84)	**VOC (N = 22)**	**INS (N = 24)**	**NO-INT (N = 22)**	**CON (N = 16)**	**HIGH-RISK (N = 17)**
dyslexia risk/ con	68/16					
female/ male	36/48	7/15	13/11	9/13	7/9	6/11
age, months (SD)	6.1 (0.3)	6.1 (0.2)	6.1 (0.3)	6.1 (0.2)	6.1 (0.3)	6.1 (0.3)
	28mo (N = 135)	**VOC (N = 30)**	**INS (N = 33)**	**NO-INT (N = 40)**	**CON (N = 32)**	**HIGH-RISK (N = 28)**
dyslexia risk/ con	103/32					
female/ male	60/75	10/20	18/15	19/21	14/18	11/17
age, months (SD)	28.1 (0.4)	28.0 (0.4)	28.1 (0.3)	28.1 (0.4)	28.0 (0.4)	28.3 (0.4)

compensated dyslexics (following the protocol of Virtala et al., 2022). The control infants (con in Table 1) were all included in the control group of the present study.

The Ethics Committee for Gynaecology and Obstetrics, Pediatrics and Psychiatry of the Hospital District of Helsinki and Uusimaa approved the study protocol and the study was performed in compliance

with the Declaration of Helsinki. One or both parents of the newborn participants gave written informed consent to participate in the study prior to the experiment.

2.2. Music listening interventions

In the first EEG recording of the DyslexiaBaby study (at birth), at-risk infants were assigned to vocal music intervention (VOC), instrumental music intervention (INS) and no-intervention control (NO-INT) groups. The group-assignment was pseudo-randomized, meaning that they were essentially randomized, with a few exceptions: the first infants enrolled, as well as infants with an unconfirmed dyslexia status at the time of the EEG recording were assigned to the NO-INT group; if there was a noticeable imbalance in the gender distribution of some groups towards the end of participants enrollment, more boys/girls were assigned to those groups to mitigate the imbalance.

Families assigned to the two intervention groups were instructed to play music (at least one playlist five times/week) to infants in a peaceful environment and not to sing along, starting during the infant's first month of life and lasting until 6 months of age. Families in the NO-INT group did not receive any specific instructions to avoid music or singing, and the three risk groups did not differ in the amounts of musical activities (shared music activities, music listening, musical playschool attendance, Virtala et al., 2023). The online song library provided to the families included a selection of nine 20-min-long playlists of Finnish children's and folk songs either with a vocalist singing (two female, three male singers; vocal intervention) or a musical instrument playing (banjo, mandolin, xylophone, or metallophone; instrumental intervention) the melody, both with the same soft acoustic guitar accompaniment. The intervention was considered successful when all three criteria were met: minimum duration of ~5 months (completed playlists in five 4-week periods), minimum intensity of ~1 h/week (~3 playlists/week) in at least four 4-week periods, and minimum listening amount of 24 h (72 playlists). A total of 13 children with an unsuccessful implementation of the music intervention were excluded from the analyses. Additional information regarding the music intervention can be found in Virtala et al. (2023).

2.3. Experimental stimuli and paradigm

The experimental stimuli and paradigm of this study have been previously described in, e.g. Thiede et al. (2019). The stimuli consisted of a bi-syllabic pseudoword /tata/ (standard stimulus) uttered with stress in the first syllable by a female native Finnish speaker (Pakarinen et al., 2014; Fig. 2), and its three variants (deviant stimuli). Stimulus duration was 300 ms, of which ~250 ms were audible, including a natural ending. The second syllable started at ~ 168 ms, and the second /a/ at ~ 181 ms (Fig. 2). In the deviant stimuli, the original /tata/ sound file was modified (Adobe Audition CS6, 5.0, and Praat 5.4.01 softwares) to change its second syllable's frequency, vowel duration, or vowel identity. The paradigm also contained very rarely-presented novel human (e.g., sigh, cry, laugh) and non-human (e.g., telephone ring, electric drill) sounds. The current study focuses specifically in the obligatory elicited responses (i.e. elicited by the standard stimulus), as the maturation of the speech discrimination responses have already been reported in Virtala et al. (2022) and the effects of the music listening interventions on them in Virtala et al. (2023). Consequently, the data for the deviant and novel stimuli are not included in the analyses of the present study.

The stimuli were presented using a multi-feature paradigm in four blocks of 471 stimuli (each block lasting 7 minutes). On average, 70.1 % of these stimuli were standard, 25.3 % deviant, and the remaining 4.6 % novel sounds. The stimuli were presented in pseudo-random order, with each block starting with four standard stimuli, and with a standard always following a deviant or novel stimulus. The inter-stimulus interval ranged from 850 to 950 ms, alternating in 10 ms steps randomly to minimize predictability effects. The standard stimulus was presented altogether 1340 times during the experiment.

2.4. EEG recordings

EEG data were recorded at birth, 6 months, and 28 months as part of the DyslexiaBaby study. These time points were selected to meet the DyslexiaBaby study's goals of 1) examining neural speech processing in early childhood and its relation to familial dyslexia risk, 2) investigating the links between neural speech processing and behavioral outcomes such as language and pre-reading skills, assessable via standardized tests from around 2–3 years of age, and 3) evaluating the impact of the music listening intervention conducted between birth and 6 months, including pre- and post-intervention measurements. EEG recordings (sampling rate: 500 Hz, low-pass filter: 100 Hz, high-pass filter: 0.5 Hz) were carried out using an electrode cap (ActiCap, Brain Products GmbH,

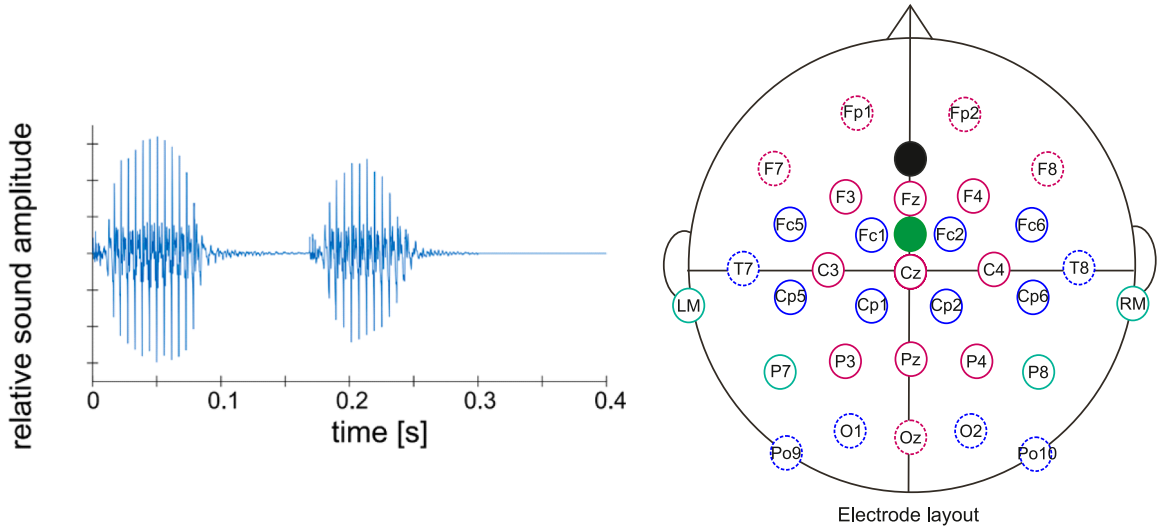

Fig. 2. Stimuli and electrode layout. Left: Waveform of /tata/pseudoword. Right: Electrode layout at 0, 6 (in pink) and 28 months (pink and blue), modified from Virtala et al. (2022). The ground (black) and ref (green, the active online reference) electrodes and the electrodes used as reference electrodes in re-referencing (turquoise, see Section 2.4) were the same at 0 and 28 months. Peripheral electrodes are indicated by a dashed circle and were not included in statistical analyses due to their overall poor signal quality.

Gilching, Germany) with 18 (at birth and 6 months) and 32 (at 28 months) electrodes placed according to the international 10/20 system (Fig. 2). All EEG recordings were conducted with a BrainProducts QuickAmp amplifier (v. 10.08.14; software: BrainVision Recorder 1.20.0801, Brain Products GmbH, Gilching, Germany). Stimuli were presented using Presentation 17.2 software (Neurobehavioral Systems Ltd., USA) through two loudspeakers (Genelec speaker) with an intensity of ≈ 65 dB SPL at the infant's head. During the recordings, the data were referenced to the average of all electrodes. A trained nurse or research assistant performed the recording, which took approximately 1–2 hours, preparations included.

The newborn measurements were conducted at Jorvi Hospital of the Helsinki University Hospital, Espoo, Finland (N = 157/177), or at a sound-proof laboratory of the University of Jyväskylä, Finland (N = 20/177) with identical equipment and protocol. EEG data were recorded while the infants lied on their back in a crib and the stimuli were presented through the speakers at 40 cm from their head. The state of the infant was determined during the recording as "active sleep", "quiet sleep", "awake" or "intermediate sleep stage" (based on Grigg-Damberger et al., 2007) by a trained professional. Data from all different sleep/alertness states were combined for the analyses to maximize the amount of data.

At 6 months, the EEG was collected at the same recording places (Jorvi Hospital N = 66/84, University of Jyväskylä N = 18/84) and with the same settings as in the newborn measurement. Infants were awake and seated on caretaker's lap. During the measurement, the research assistant or nurse engaged with the infants silently by making facial expressions, displaying toys, etc.

At 28 months, EEG was recorded after a 2-hour neuropsychological evaluation, on a different day or, rarely, on the same day after a long break, in a soundproof, electrically shielded laboratory at the University of Helsinki (N = 120/135), or in the same laboratory at the University of Jyväskylä as in earlies phases of the study (N = 15/135), using identical equipment and recording protocol. Children were awake and sitting on parent's lap placed 160 cm from the speakers. During the recording, the child was instructed to not to talk or move, and time stamps indicating any instances of talking or movement were recorded and taken into account during manual rejection of the EEG data (see Section 2.4.1.). In addition, children's attention was directed away from the presented stimuli by displaying muted cartoons.

2.5. Analysis

2.5.1. EEG preprocessing

Before preprocessing, EEG data from those blocks where the infant/child exhibited frequent crying or voicing, or where the 6-month-old or 28-months-old children fell asleep, were omitted from the dataset (Fig. 1 "Poor data quality"). The whole preprocessing pipeline was designed by senior researchers of the team in collaboration with laboratory engineers. Preprocessing was conducted with Matlab 2017a–2020a (The MathWorks, Inc., USA), with Toolboxes EEGLAB 14.0.0b and 2019_0 (Delorme & Makeig, 2004) and ERPLAB 7.0.0 (Lopez-Calderon & Luck, 2014). First, the EEG data was filtered using a 0.025–40 Hz band pass in order to exclude low and high frequency artifacts and to allow for visual inspection. The visual inspection of the data was conducted by researchers and research assistants based on written and illustrated instructions by the research team. During visual inspection, parts with clear muscle-related artifacts were omitted from the data, and eye-movement and heartbeat artifacts were marked for a later removal stage. This procedure was only performed with the 28-month EEG data, as in the newborn and 6-month data the artifacts were not clearly identifiable. Furthermore, during this stage, electrodes with flat or continuously noisy signal were identified as "bad". No more than five electrodes (28 %) in the newborn and 6-month-EEG and six (19 %) in the 28-month-EEG were considered "bad". If the "bad" electrodes were peripheral (newborn and 6 months: Fp1, Fp2, F7, F8, Oz; 28 months: T7, T8, Po9, Po10, O1, O2) they were excluded from the analysis. On the other hand, bad central electrodes (newborn and 6-months: F3, Fz, F4, C3, Cz, C4, P3, Pz, P4; 28 months: FC5, FC1, FC2, FC6, CP5, CP2, CP3, CP6) were marked down to be interpolated (maximum of 2 in the newborn and 6-month data and a 3 in the 28-month data; in case of more bad electrodes, the data of the whole stimulus block were excluded from analysis) in a later stage.

After the visual inspection, EEG data were filtered (0.5–25 Hz band pass) and re-referenced to the average of two mastoid (LM, RM) and two posterior scalp (P7, P8) electrodes. Reference electrodes were considered broken if they were flat or had a signal continuously exceeding ± 250 μV; in this case, the electrode and its contralateral pair were eliminated, and an average of the remaining reference electrodes was used. If both reference electrodes on one side of the head were considered broken, the data of that stimulus block were excluded. Thereafter, bad central electrodes were interpolated using ERPLAB's spherical interpolation algorithm. This process was only performed when necessary (in case of very poor signal quality), to ensure sufficient data quality and consistency. For the 28-month data, following interpolation, eye movement and heartbeat related artifacts were corrected using independent component analysis (ICA) with *fastica* (Hyvarinen, 1999) or *runica* EEGLAB algorithms. Components were not removed if the removal of the component from the data was unsatisfactory based on visual inspection (e.g., artifact was not diminished or algorithm changed other parts of the data). The decision of applying ICA only at 28 months was based on the feasibility of ICA at this age, as the larger head size allowed for a greater number of electrodes, and eye blinks were more clearly identifiable in the continuous EEG. Applying ICA to the 28-month data ensured optimal data quality by removing identifiable eye blink artifacts, thereby reducing noise in the ERP waveforms. In contrast, ICA was not feasible for the 0- and 6-month data due to fewer electrodes and poorly distinguishable eye movements.

Finally, EEG data were segmented into −100–840 ms epochs after stimulus onset separately for each stimulus, electrode, and participant. Baseline correction was applied −100–0 ms prior to stimulus onset. The epochs of those standard stimuli that were immediately following a deviant were excluded from analysis. Furthermore, if an epoch amplitude exceeded ± 120 μV at Fp1 and Fp2 electrodes, it was excluded to avoid eye-movement-related artifacts. We also omitted epochs with a drift of > 100 μV or data points ± 3 SD from the mean amplitudes of all epochs. After that, epochs of the same stimulus type were merged, resulting in one dataset per participant. Data of infants/children with less than 100 accepted epochs for the standard stimulus were excluded (newborn N = 0, 6 months N = 19, 28 months N = 1, Fig. 1).

2.5.2. ERP quantification and statistical analysis

ERP amplitudes were extracted using the toolboxes EEGlab (version 14.0.0; Delorme & Makeig, 2004) and CBRUPlugin (version 2.0b) in MATLAB (Release 2018b; The MathWorks, Inc., Natick, Massachusetts, USA). At birth and 6 months, the individual peak latencies of the infant P1 and N2 were searched from broad time windows in a large region-of-interest (ROI) resulting from averaging the responses from 6 electrodes (F3, Fz, F4, C3, Cz, and C4), chosen based on visual inspection of the average waveforms and in accordance with the fronto-central predominant scalp distribution of the P1 and N2 responses (Liégeois-Chauvel et al., 1994). The selected time windows, reported in milliseconds after stimulus onset, were the following: 150–450 ms (width of 85 ms) for infant P1 at birth, 50–250 ms (width of 40 ms) for infant P1 at 6 and 28 months, and 300–500 ms (width of 80 ms) for infant N2 at 6 and 28 months. For the peak latency search, an additional low-pass filter of 10 Hz was added to the data. The mean amplitudes were quantified after removing the additional 10 Hz filter, by centering a time window at the individual peak (the widths of these time windows are listed above in brackets). Since no negative component was distinguishable in the newborn EEG data, the newborn N2 response could not be analyzed. In the quantification of the 28-month-data, as a larger

amount of electrodes was used in the recording, four additional electrodes (FC1, FC2, FC5, and FC6) were added to the ROI for the mean amplitude quantification, in order to improve the signal-to-noise ratio. Furthermore, in order to study the hemispheric distribution of the responses at all ages, additional left and right ROI mean amplitudes were quantified by averaging the responses from the left and right hemisphere electrodes (all large ROI electrodes except for the midline Fz, Cz).

Maturational changes in mean amplitudes and peak latencies across the large ROI were examined using latent growth curve models (LGCM) in R, implemented via the *lavaan* package. LGCMs use the Structural Equation Modeling framework and allow for the estimation of temporal changes while accounting for multiple time points nested within individuals. Each model included an intercept factor (representing initial levels) and a slope factor (representing change over time), estimated across the defined time points. Analyses were conducted for responses elicited at a minimum of two measurement points within the whole sample. Four separate LGCMs were estimated to examine the infant P1 and N2 components, with mean amplitudes and peak latencies analyzed independently.

For the infant P1 component, the initial LGCM assumed linear change by fixing slope loadings at each time point. However, when non-linear change was suspected, it was tested using a relative change approach. In this approach, the slope was fixed at 0 at 0 months and at 1 at 28 months, with the intermediate loading at 6 months freely estimated. This allowed the slope to represent the total change from the first to the last time point, while the estimated loadings indicated the proportion of change occurring at each intermediate point relative to the total change. Linear and non-linear LGCMs were compared to determine which model better fit the data. This approach was not needed for the infant N2, as it was elicited only at 6 and 28 months. Full information maximum likelihood (FIML) estimation was used to handle missing data, accounting for all available data without imputation and providing unbiased estimates under the missing at random (MAR) assumption.

To assess differences in mean amplitudes, peak latencies, and their maturation between the risk and control groups, dyslexia risk was incorporated as a time-constant predictor in the models. This approach allowed for the estimation of group differences in initial levels (intercept), subsequent time points, and growth trajectories (slope), accounting for dyslexia risk when describing the maturation of obligatory ERPs in the sample. Furthermore, according to previous studies from our group, a supplementary analysis was conducted to compare the elicitation and maturation of obligatory ERPs between the high-risk and control groups. Similarly, differences between intervention groups were analyzed by including intervention as a time-constant predictor. Since the *lavaan* package does not directly handle multi-categorical variables, dummy coding was applied to the three-level categorical variable (VOC, INS, and NO-INT), with the NO-INT group serving as the reference category. Finally, in order to investigate hemispheric differences in mean infant P1 and N2 amplitudes, the intercept and slopes for left and right ROIs were added into the LGCMs, with hemisphere included as a time-constant predictor in the model, and following the same approach as with the rest of predictors.

Furthermore, to minimize overfitting and reduce the risk of bias, a systematic model selection approach was employed. The process began with a null model containing only the intercept and slope. Additional models were then constructed step by step, incrementally incorporating predictors and interaction terms until reaching an omnibus LGCM with all the possible predictors (risk, intervention groups, and hemisphere). Then, model fit was evaluated using the Akaike Information Criterion (AIC), and the best-fitting model was selected for interpretation. Since the models were not nested, the model fit was not addressed through Chi-squared difference tests.

To account for multiple comparisons, a Bonferroni correction was applied. The initial significance threshold of $p < .05$ was adjusted to $p < .0041$ (based on 12 tests in the final LGCMs) to maintain a family-wise error rate of 5 %. All reported p-values are evaluated against this corrected threshold.

3. Results

3.1. Obligatory ERPs and their maturation in the whole sample

At birth, a broad infant P1 response appeared and reached its maximum amplitude around 300 ms after stimulus onset, whereas no negative peaks were elicited. At 6 and 28 months, the waveform consisted of a clear infant P1 followed by an N2. Maturation of the ERPs is illustrated in Fig. 3 and Fig. 4, mean amplitudes and peak latencies are listed in Table 2, and a summary of the statistically significant effects is provided in Table 3. Complete statistics of the final LGCMs, after the model selection approach, are provided in the Supplementary Table S1.

The infant P1 response exhibited a significant increase in amplitude [slope: $\beta = 6.16$, $z = 17.92$, $p < .001$] and a decrease in latency [slope: $\beta = -173.76$, $z = -21.52$, $p < .001$] across the three time points in the whole sample. However, visual inspection of the data (Figs. 3 and 4) suggested non-linear patterns in these changes. Therefore, to test for non-linear change, a relative change approach was adopted by fixing slope loadings at 0 at birth, and at 1 at 28 months, while freely estimating the loading at 6 months. The non-linear latent growth curve models (LGCMs) demonstrated better fit compared to the linear models (P1 amplitude: AIC = 938.68 vs. 1000.74; P1 latency: AIC = 1850.8 vs. 1929.5). Under a linear growth assumption, the expected slope estimate from birth to 6 months would be approximately 21 % ($\beta = 0.21$). However, for the infant P1 amplitude, the non-linear LGCM indicated that 100 % of the change occurred between birth and 6 months [slope: $\beta = 1.15$, $z = 15.38$, $p < .001$], with no change observed between 6 and 28 months. For infant P1 latency, 77 % of the total decrease occurred between 0 and 6 months [slope: $\beta = 0.77$, $z = 58.84$, $p < .001$], while the remaining 23 % occurred between 6 and 28 months. Although the parameter for the 6- to 28-month interval was fixed (precluding direct computation of a *p*-value), the smaller relative change suggests a more gradual decrease in latency during this period.

For the infant N2 response, the LGCMs revealed a significant increase in latency with age between 6 and 28 months [slope: $\beta = 39.45$, $z = 3.09$, $p = .002$]. However, the slope estimate for N2 amplitude was not significant [$\beta = -2.29$, $z = -1.74$, $p = .081$], suggesting that there was no significant change in amplitude over time.

When hemispheric lateralization (left vs. right ROI) was incorporated into the LGCMs, no significant main or interaction effects of hemisphere were detected, and AIC values did not decrease. Following the model selection approach, hemisphere was not included in final models.

3.2. Effects of dyslexia risk and music intervention on obligatory ERPs and their maturation

At birth, the infant P1, and at 6 and 28 months, the infant P1 and N2, were observable in the control and risk groups and in the three intervention groups VOC, INS, and NO-INT. Thus, both responses were included in the group comparison LGCMs. Maturation of the ERPs in these groups is illustrated in Fig. 5, and a summary of the statistically significant effects is provided in Table 4. Complete statistics of the final LGCMs, following the model selection approach, are provided in the Supplementary Table S1.

When familial dyslexia risk was incorporated as a time-constant predictor, a significant regression was observed between the intercept of N2 amplitudes and risk [$\beta = -3.22$, $z = -2.88$, $p = .004$], indicating that the high-risk group exhibited larger N2 amplitudes at 6 months compared to the control group. No significant effects of risk were observed for the N2 amplitudes at 28 months, or for the remaining LGCMs. Moreover, no significant effects of the music intervention were observed, suggesting that the intervention groups did not differ in their obligatory ERPs. Following the model selection procedure, dyslexia risk

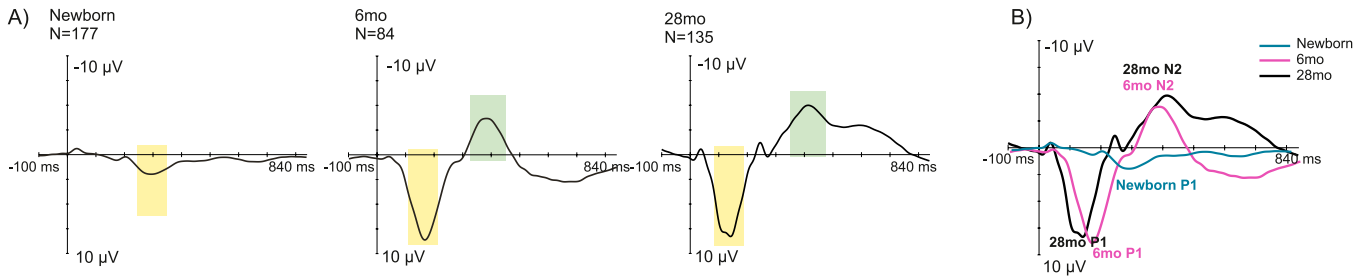

Fig. 3. Maturation of the obligatory auditory event-related potentials, infant P1 and N2. A. ERP waveforms in the whole sample at birth (left), 6 months (6mo, middle) and 28 months (28mo, right), averaged over the large regions-of-interest (ROIs) with baseline at stimulus onset (latency in ms, amplitude in μV). Colorful bars illustrate the time windows used for searching the individual peak latencies (yellow for infant P1, green for infant N2). B. ERP waveforms illustrating infant ERP maturation at birth (blue line), 6 months (pink line) and 28 months (black line), at the large ROIs, with baseline at stimulus onset.

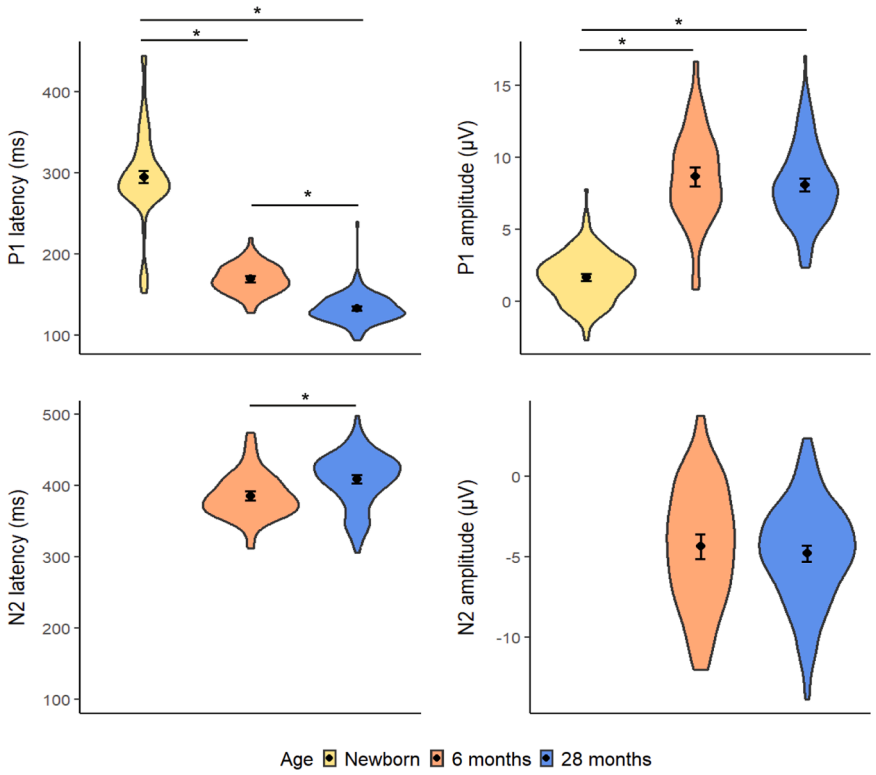

Fig. 4. Mean amplitudes and peak latencies of infant P1 and N2 at ages 0 (yellow), 6 months (orange), and 28 months (blue). In the latent growth curve models, the infant P1 component was larger and earlier at 6 and 28 months than at birth. The infant N2 latency was later at 28 than at 6 months. The error bars represent 95 % confidence intervals. Statistically significant post hoc comparisons are marked with asterisks.

was excluded from all final models except the one for N2 amplitude, and the effect of intervention group were removed from all final models. Additionally, when hemispheric lateralization (left vs. right ROI) was added to the group comparison LGCMs, no significant main or interaction effects of hemisphere with risk or intervention were found.

To further explore the effect of dyslexia risk, a supplemental analysis was conducted to compare the elicitation and maturation of obligatory ERPs between the high-risk and control groups. This analysis confirmed the earlier findings: a significant regression was observed between the intercept and high-risk status [$\beta = -3.19$, $z = -2.70$, $p = .004$], indicating larger N2 amplitudes in the high-risk group compared to controls, with no significant differences in the remaining LGCMs.

4. Discussion

Despite the proposed importance of efficient neural speech encoding abilities for supporting early language development, there is a lack of longitudinal studies with large sample sizes on the typical/atypical maturation of obligatory speech-sound-elicited ERPs during the first years of life. The present study aimed to examine the elicitation of these obligatory auditory ERPs at birth, 6 months, and 28 months, as well as their maturation across these ages, in a large longitudinal sample overrepresented by children at risk for developmental dyslexia. Additionally, the study assessed the impact of dyslexia risk and a music listening intervention on the elicitation and maturation of these responses. At birth, only the infant P1 response was elicited. This response grew in amplitude until 6 months and decreased gradually in latency until 28 months. No significant changes were observed for infant P1 amplitude from 6 to 28 months. The infant N2 component emerged by 6 months and increased in latency until 28 months, with no significant change in its amplitude. Compared to high dyslexia risk infants, somewhat unexpectedly, the control group without familial dyslexia risk exhibited smaller N2 amplitudes than the at-risk group at 6 months. No effects of the infant music listening intervention on the obligatory ERPs

Table 2
Mean amplitudes (in μV, with standard deviation, SD, in parentheses) and peak latencies of the obligatory elicited ERPs in the whole sample at birth (Newborn), 6 months (6mo), and 28 months (28mo) at the large, left, and right regions-of-interest (ROIs).

		Mean amplitude, μV (SD)			Peak latency, ms (SD)
Age	ERP	Large ROI	Left ROI	Right ROI	
Newborn	P1	1.65 (1.73)	1.62 (2.07)	1.78 (1.90)	293.26 (56.9)
6mo	P1	8.75 (2.37)	9.12 (2.54)	8.66 (2.60)	167.75 (13.9)
	N2	−3.36 (3.67)	−3.29 (3.84)	−3.67 (3.61)	383.93 (34.2)
28mo	P1	8.28 (2.41)	8.32 (2.37)	8.25 (2.67)	130.90 (12.5)
	N2	−5.14 (3.33)	−4.94 (3.35)	−5.43 (3.48)	414.35 (33.9)

Note. The electrodes included in the large ROIs at birth and 6 months are F3, Fz, F4, C3, Cz, and C4, and at 28mo, F3, Fz, F4, C3, Cz, C4, Fc1, Fc2, Fc5, and Fc6. The electrodes included in the left and right ROIs at birth and 6 months are F3 and C3 and F4 and C4, respectively, and at 28mo, F3, C3, Fc1, and Fc5 and F4, C4, Fc2, and Fc6, respectively.

Table 3
Summary of the statistically significant ($p < .0045$) main results of the latent growth curve model (LGCM) analyses for mean amplitudes (AMPL) and peak latencies (LAT) of the infant P1 and N2 responses with the effect of age (age in months, mo) as a fixed factor.

	AMPL/ LAT	Estimate (β)	*p*	Description
P1				
Slope (0–28mo)	AMPL	6.16	< .001	Increase in infant P1 amplitude with age.
	LAT	−173.76	< .001	Decrease in infant P1 latencies with age.
Relative Slope (0–6mo)*	AMPL	1.15	< .001	100 % of the increase on infant P1 amplitude between 0 and 6 months.
	LAT	0.77	< .001	77 % of the decrease on infant P1 latency between 0 and 6 months.
N2				
Slope (0–28mo)	LAT	39.45	.002	Earlier infant N2 at 6 than at 28 months.

* *Note*: The LGCMs for infant P1 amplitude and latency employed a non-linear approach to assess relative change. The estimated slope parameter between 0 and 6 months represents the proportion of total change that occurred by the intermediate time point (6 months) relative to the overall change observed at 28 months.

were seen. Overall, this study validates and further extends previous findings on the maturation of obligatory auditory ERPs during infancy and early childhood with a larger sample size (N = 84–177 depending on the age group) compared to a total N = 15–45 in the three previous longitudinal studies by Choudhury and Benasich (2011), Kushnerenko et al. (2002) and Shafer et al. (2015). In addition, it may serve as a foundational reference for future studies investigating the effects of developmental conditions on speech-sound processing and language development.

4.1. Obligatory ERPs and their maturation in the first years of life

We expected that the infant P1 would be observable already in the newborn waveform, with a clear two-peaked pattern (P1-N2) becoming evident at 6 months. Both responses were expected to grow in amplitude and decrease in latency in a somewhat linear trend with increasing age (Choudhury & Benasich, 2011; Kushnerenko et al., 2002; Shafer et al., 2015; Silva et al., 2017; Wunderlich & Cone-Wesson, 2006).

The infant P1 was robustly elicited at birth, grew in amplitude until 6 months, and decreased in latency until 28 months. However, no differences in amplitude were observed between 6 and 28 months of age. These results of growing P1 amplitudes in early infancy support our initial hypothesis and are consistent with the findings of Kushnerenko et al. (2002), who observed that P1 amplitudes to harmonic sounds were significantly larger at 3 months of age than at birth, with no significant growth from 3 months to one year. Additionally, they align with Wunderlich et al. (2006), who cross-sectionally reported larger P1 amplitudes to speech sounds at 1 year than at birth, but did not note any increase from then until 6 years. However, our findings of no change in the infant P1 amplitude between 6 and 28 months partially contrast with previous longitudinal studies describing its gradual increase from 6 to 24 months (Choudhury & Benasich, 2011, using tone stimuli; Shafer et al., 2015, using speech stimuli). One possible explanation for this contrast is the faster presentation rates used in these studies compared to ours (300–600ms vs. 850–950ms), which may have resulted in less salient responses during the early months of life. The results for the infant P1 latency are also consistent with previous studies reporting a significant decrease with maturation, particularly between 3 and 12 months of age (Kushnerenko et al., 2002; Shafer et al., 2015).

The infant N2 was discernible at 6 months and showed a significant increase in latency until 28 months, while its amplitude remained constant. These results align with those of previous studies reporting the presence of this response at around 6 months of age (Benasich et al., 2006; Choudhury & Benasich, 2011; He et al., 2007; He et al., 2009; Little et al., 1999; Shafer et al., 2015). However, these previous studies also report an increase in its amplitude with age, which we did not observe. Furthermore, our findings partially contradict previous studies that have reported the presence of the N2 already at birth (Molfese, 2000; Wunderlich et al., 2006, using speech sounds) or its decrease in latency during the first years of life (Choudhury & Benasich, 2011; Kushnerenko et al., 2002; Shafer et al., 2015; Wunderlich & Cone-Wesson, 2006). Nevertheless, the occurrence of the N2 throughout infancy is inconsistent across studies and often lacks the robustness observed for the P1 (Kushnerenko et al., 2002; Shafer et al., 2015). A possible explanation for this variability is that the emergence of N2 at birth may be highly dependent on stimulus parameters and presentation rate. For example, it has been found that lower presentation rates (above 1 sec) and the use of non-speech sounds as stimuli can facilitate the elicitation of N2 in infants and preschoolers (Kuuluvainen et al., 2016; Ponton et al., 2000; Shafer et al., 2015).

Overall, our results suggest that speech-sound-elicited obligatory ERPs emerge and strengthen rapidly within the first months of life, with an infant P1-N2 complex becoming already robust at 6 months. The presence of a strong infant P1 response already at birth indicates that it is the most prominent ERP during infancy (Shafer et al., 2015; Silva et al., 2017; Wunderlich & Cone-Wesson, 2006). Furthermore, the observed changes in infant P1 latency and amplitude by six months of age coincide with a period of synaptic organization within its neural generators in the primary auditory cortex (Liégeois-Chauvel et al., 1994), suggesting that its maturation reflects an increase in the synaptic density and efficiency of these locations (Sussman et al., 2008; Yvert et al., 2005). Altogether, the findings of the present study support the idea that the first months of life play an important role in shaping the neural mechanisms underlying speech-sound encoding (Puertollano et al., 2024; Kuhl, 2004). Functionally, they suggest that by six months of age, the infant brain registers auditory features of speech sounds, a critical ability to support language acquisition (Best et al., 2016; Cheour et al., 1998; Maye et al., 2002). Additionally, the distinct maturational trajectories of the infant P1 and the N2 suggest that these responses reflect separate neural processes already during the first months of life. This is compatible with the suggestion that while the infant P1 reflects a readiness for speech-sound detection and formation of sound representations, the infant N2 may serve as an index of sensory memory trace

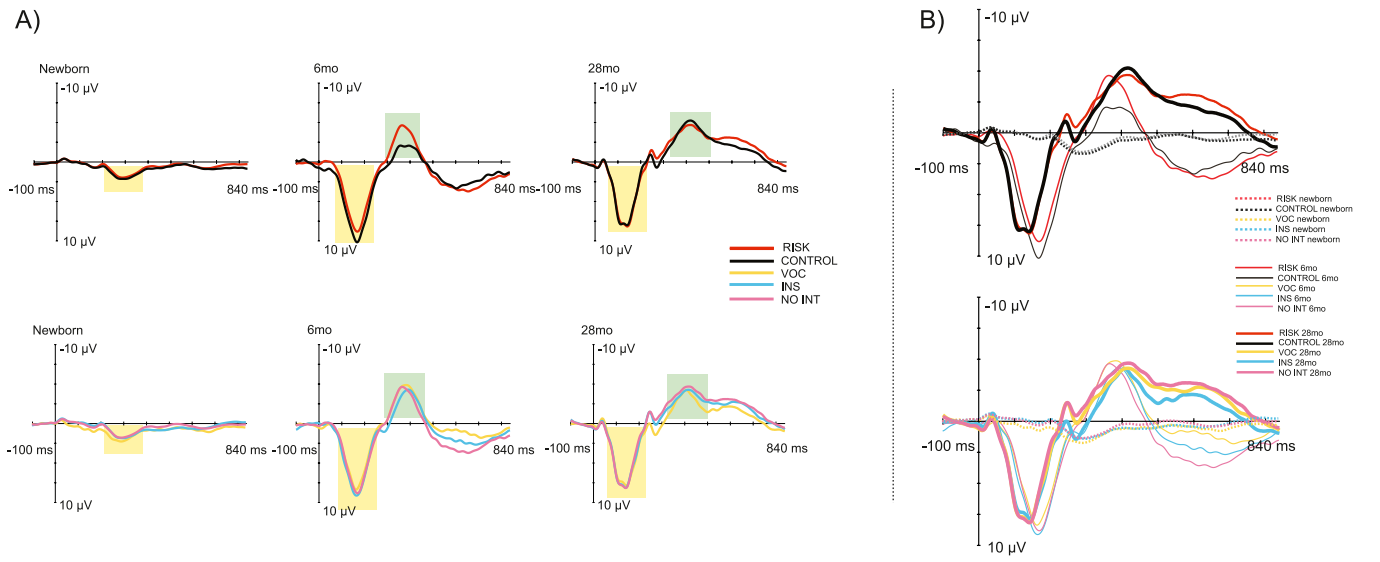

Fig. 5. Maturation of the obligatory auditory ERPs, infant P1 and N2 in the risk and intervention subgroups. A. ERP waveforms in the risk vs. control (upper row) and in the intervention (lower row) subgroups at birth (left), 6 months (6mo, middle) and 28 months (28mo, right), depicted at the large regions-of-interest (ROIs) with baseline at stimulus onset (latency in ms, amplitude in μV). Colorful bars illustrate the time windows used for searching the individual peak latencies (yellow for infant P1, green for infant N2). B. ERP waveforms illustrating infant ERP maturation in the risk vs. control (upper row) and in the intervention (lower row) subgroups at birth (dashed lines), 6 months (thin lines) and 28 months (thick lines), at the large ROIs, with baseline at stimulus onset. VOC = Vocal intervention; INS; Instrumental intervention, NO-INT (no intervention).

Table 4
Summary of the statistically significant ($p < .05$) main results of the latent growth curve model (LMM) analyses for mean amplitudes (AMPL) and peak latencies (LAT) of the infant P1 and N2 responses in the risk vs. control groups, with effects of age (age in months, mo) as fixed factor.

Age	AMPL/ LAT	Effect	Estimate (β)	*p*	Description
N2 Intercept ~ risk	AMPL	risk	−3.22	.004	Smaller infant N2 at 6 months in the control vs the risk group

formation and encoding of more complex acoustic features (Ceponiene et al., 2005, 2008). Nevertheless, it is important to note that in the absence of control non-speech stimuli, these conclusions on the maturation of obligatory ERPs are very preliminary and might rather reflect general than speech-specific early maturation of the auditory system. To better understand whether the neural responses we observed are unique to speech processing or part of a broader auditory maturation process, further research incorporating both speech and non-speech stimuli is needed.

4.2. Impact of dyslexia risk on obligatory ERPs and their maturation

In the present study, the only observed effect of familial dyslexia risk was an enhanced N2 response in the risk group compared to the control group at 6 months of age. No dyslexia risk effects were found on the infant P1 at any age, on the infant N2 at 28 months, or on the maturation of the infant ERPs across the studied age points. These results remained consistent in the additional comparison between the high-risk and control groups. This finding of very little effects of dyslexia risk on the speech-elicited ERPs was not an expected result. In addition, it is in contrast with previous findings of smaller ERPs in infants at risk for dyslexia (Molfese, 2000; van Herten et al., 2008) and developmental language disorder (Choudhury & Benasich, 2011) than in control infants. However, it is important to note that previous literature is limited, often constrained by small sample sizes, and varies significantly in terms of stimulus type and presentation rates. In addition, our results imply a distinct effect of dyslexia risk on neural speech encoding vs. discrimination. Namely, in our DyslexiaBaby sample mismatch responses were shown to differ between risk and no-risk groups consistently across measurement points (Virtala et al., 2022). The effect of dyslexia risk on mismatch responses has been also reported in several other studies (Choudhury & Benasich, 2011; Leppänen et al., 2002; Thiede et al., 2019; van Leeuwen et al., 2006). Therefore, our findings suggest that the auditory deficits associated with dyslexia risk may primarily be related to discriminatory processes, as reflected by mismatch responses (Näätänen et al., 2019), rather than to sound encoding processes represented by the obligatory ERPs.

Finding larger N2 amplitudes at 6 months in the risk group compared to the control group was unexpected and inconsistent with previous literature, which has generally reported diminished amplitudes in risk groups (Benasich et al., 2006; Choudhury & Benasich, 2011; Molfese, 2000; van Herten et al., 2008, however, for increased mismatch responses in dyslexia risk infants, see Virtala et al., 2022). However, two factors should be considered when interpreting this result. First, the control group exhibited greater variability in the individual N2 latencies than the risk group (Supplementary Figure S1). This variability suggests that some children in the control group may have exhibited an emerging N1 component preceding the N2 but within its search window, thus complicating the quantification of the latter. Second, the data quality for the 6-month-olds was notably poorer than for newborns and 28-months-old, as indicated by visual inspection and the high number of excluded infants and rejected epochs (see Section 2.5). Given these considerations, this result should be interpreted with caution. Nevertheless, it cannot be ruled out that it might reflect an effect of familial dyslexia risk on the maturational stage of the neural mechanisms underlying the N1-N2 around 6 months of age, as has been previously suggested in the case of increased positive mismatch responses to salient acoustic changes in dyslexia-risk infants (Leppänen et al., 2010; Virtala et al., 2022).

4.3. Impact of the music listening intervention on obligatory ERPs and their maturation

We observed no significant intervention effects on the mean

amplitudes, peak latencies, or maturation of obligatory ERPs from birth to 28 months of age. This finding contrasts with previous reports on the effects of musical interventions on the early development of auditory ERPs (Partanen et al., 2013, 2022; Trainor, 2012), as well as with the findings of Virtala et al. (2023), who showed that this same intervention enhanced mismatch responses during the first year of life. This discrepancy in the effects of the same intervention on different responses suggests that, similar to how speech-sound-elicited obligatory ERPs seem to be less influenced by developmental conditions, they may also be a less sensitive measure of phonetic learning than the speech-sound discrimination reflected by mismatch responses. However, it is important to note that since children in the NO-INT group were not instructed to avoid music or singing, uncontrolled exposure to these activities outside the intervention could potentially explain the lack of effect. Yet, it should be noted that Virtala et al. (2023), using a sample closely comparable to that of the present study, found no significant differences among the three risk groups in parent-reported music-related variables, including shared music activities, general music exposure, and participation in musical play school.

4.4. Limitations

When interpreting our findings on the typical maturation of the obligatory ERPs, it is important to consider the large proportion (around ¾) of children at risk for dyslexia in our sample. Although we did not find an effect of dyslexia risk on the elicitation or maturation of infant P1 and N2, apart from the enhanced infant N2 at the age of 6 months, other studies have reported alterations in these components in children with dyslexia and other learning disorders (Choudhury & Benasich, 2011; Molfese, 2000; van Herten et al., 2008). It remains possible that in a larger sample of control infants, the peak amplitudes and latencies of the obligatory ERPs might have been, for example, more pronounced across the different measurement points, potentially revealing different maturational trajectories. However, we chose to analyze the entire sample to maintain a larger sample size, which is often lacking in previous longitudinal studies. Supplementary Figure S2 displays only the waveforms of the control group (sample size ranging from 16 to 46 participants) and, except for the N2 amplitude at 6 months, shows a maturation pattern similar to that observed in the entire sample.

Furthermore, the parameters of our experimental paradigm could influence the interpretation and generalizability of our results. For example, our relatively fast rate of auditory stimulus presentation might have reduced ERP amplitudes and may even suppress them (He et al., 2009; Ponton et al., 2000; Shafer et al., 2015). Consequently, studies with slower presentation rates might yield results that differ from those of ours, complicating direct comparisons. In addition, our results are not directly comparable with studies using non-speech stimuli, which elicit enhanced P1 and N2 responses (Kuuluvainen et al., 2016). However, despite these issues, the large longitudinal sample in our study offers a substantial advantage in terms of reliability, significantly contributing to the knowledge on early speech-encoding development.

A further limitation of this study is that EEG recordings at the newborn stage were conducted during varying states of alertness, whereas all recordings at later time points were obtained while the participants were awake. Previous research has demonstrated that differences in alertness, particularly sleep states, can significantly influence neonatal ERPs (Friederici et al., 2002). It is therefore likely that sleep states attenuated the ERP amplitudes observed in newborns in our sample. Despite this limitation, we opted to retain all available data to maximize the sample size and maintain consistency with prior studies. Future research should aim to systematically monitor and control for sleep states during neonatal EEG recordings to better disentangle their effects on ERPs during development and maturation.

Moreover, a potential limitation of the current study is the use of slightly different EEG preprocessing procedures across age groups. Specifically, ICA was only applied at the 28-month time point, as it was not feasible at earlier ages due to fewer electrodes and the difficulty in identifying eye blinks in the continuous EEG. While this decision was based on optimizing data quality at each time point, the variation in preprocessing may introduce some inconsistency across age groups and should be considered when interpreting longitudinal effects.

Finally, in the present dataset, there is a relatively long (22-month) gap between the second and third measurement point. Thus, it may be possible that more measurement points between these ages might have revealed more detailed information on the maturation of these ERPs, for instance, certain developmental phases of faster or slower maturation.

4.5. Conclusions

The present study confirms and extends earlier findings on the maturation of the obligatory speech-sound-elicited ERPs during the first years of life as follows: The infant P1 and N2 components are the most prevalent ERPs in infancy and early childhood. At birth, the infant P1 is the dominant ERP, increasing in amplitude until 6 months and decreasing in latency until 28 months. The infant N2 emerges after P1 by 6 months and slightly increases in latency by 28 months of age. The study found no significant effects of music listening interventions or dyslexia risk on the elicitation and maturation of these responses, except for an enhanced infant N2 amplitude at 6 months in the risk group compared to the control one. Overall, this study offers robust results on the morphology and maturation of the obligatory ERPs during the first years of life, and highlights the importance of the first months of life in shaping neural speech encoding. Furthermore, it creates a solid foundation for reliable investigations into the effects of developmental and acquired conditions on infant speech processing and language development.

CRediT authorship contribution statement

Sergio Navarrete Arroyo: Writing – review & editing, Writing – original draft, Visualization, Methodology, Funding acquisition, Formal analysis. **Paula Virtala:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Conceptualization. **Vesa Putkinen:** Writing – review & editing, Formal analysis. **Teija Kujala:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Data curation.

Declaration of Generative AI and AI-assisted technologies in the writing process

During the preparation of this work, no generative AI or AI-assisted technologies have been implemented.

Declaration of Competing Interest

None of the authors have potential conflicts of interest to be disclosed.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.biopsycho.2025.109050.

Data availability

The data that has been used is confidential.

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