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Development of brain responses to speech sounds and associations with reading and reading related skills in adults with childhood dyslexia

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1. Introduction

Speech perception begins remarkably early in human development with evidence showing that the auditory system is responsive to speech sounds even during the prenatal period (DeCasper and Spence, 1986; Partanen et al., 2013; Kaga, 2022). However, it continues to develop across the lifespan, shaped by both biological maturation as well as experience-driven and environmental factors such as language exposure and learning (McMurray et al., 2018; Colby and McMurray, 2023). During the first year of life, particularly within the sensitive period, speech perception is modified towards specialization to the phonetic elements of the surrounding language (Werker and Tees, 1984; Kuhl et al., 1992; Werker and Curtin, 2005; Kuhl, 2007). This specialization involves not only perceptual tuning, but also the formation of language-specific phonological categories, such as phonemes, reflecting the emergence of more structured representations in the brain (Cheour et al., 1998). Phonological awareness, which is grounded by these phonological representations, has been found to be crucial in learning to read in school-aged children (Melby-Lervåg et al., 2012). Reading, in turn, influences and modifies these phonological abilities (Monzalvo and Dehaene-Lambertz, 2013; Dehaene et al., 2015). It has been found that problems in phonological awareness are not only associated with but also predict future reading difficulties (Scarborough, 1998; Clayton et al., 2020).

Developmental dyslexia manifests as difficulty in acquiring accurate and fluent reading and writing skills, despite adequate sensory acuity, cognitive capacity, or opportunities for learning (Lyon Reid et al., 2003). Dyslexia is regarded as a hereditary learning difficulty with a heritability estimate of 40–60% and is relatively common among school-aged children, with approximately 10% of the population meeting diagnostic criteria (Shaywitz and Shaywitz, 2003). Reading difficulties typically become apparent once children begin learning to read and performing reading-related tasks during the early grades of school (McCardle et al., 2001).

A range of cognitive deficits have been associated with dyslexia. One of the most consistently observed impairments in dyslexia is in the processing of phonological information (Snowling, 1998), particularly phonological awareness, the ability to recognize and manipulate the sound structures of language (McBride-Chang, 1995). Another well-documented difficulty in dyslexia involves rapid automatized naming (RAN), the ability to quickly name familiar objects or symbols, which is often slower in individuals with dyslexia, contributing to challenges in reading fluency (e.g. Neuhaus et al., 2001; Gray and Powell-Smith, 2024). In addition to phonological and naming speed deficits, other cognitive deficits have been identified in dyslexia. These include impairments in verbal short-term memory (Brady et al., 1983; De Jong, 1998; Beneventi et al., 2009; Cowan et al., 2017), sensory processing – particularly auditory temporal processing (Tallal, 1980; Goswami et al., 2011; Vandermosten et al., 2011), and multi-modal associative learning (Windfuhr and Snowling, 2001).

Although developmental dyslexia has been widely studied, the development of speech processing impairments and their impact on reading skills from infancy through childhood and adulthood remain poorly understood. Brain responses to changes in speech sound features in infants with a familial risk of dyslexia have been shown to be associated with later dysfluent reading skills (Van Zuijen et al., 2012; van Zuijen et al., 2013; Schaadt et al., 2015; Lohvansuu et al., 2018), highlighting the critical role of early speech perception in shaping phonological development, and ultimately, reading acquisition. Moreover, developmental studies have shown that individuals with and without dyslexia exhibit distinct brain responses, and these differences are consistently associated with reading skills development (Maurer

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et al., 2009; Hämäläinen et al., 2013; Lohvansuu et al., 2014). Here event-related potentials (ERPs) and their magnetic counterparts (event-related fields, ERFs) to auditorily presented pseudowords were investigated in adults with childhood dyslexia and typically reading adults with and without family risk background for dyslexia aiming to fill this gap in the understanding of the development of the deficiency in speech perception.

1.1. *Deficit in speech perception in dyslexia*

It has been proposed that deficits in speech perception may precede and contribute to impairments in phonological processing, potentially hindering the development of accurate phonological representations (Goswami, 2002). Previous research has shown that individuals with dyslexia often struggle with discrimination and categorization of speech sounds, including consonants, vowels, and phonemic length (Serniclaes and Sprenger-Charolles, 2003; Bogliotti et al., 2008; Pennala et al., 2010). Additionally, differences in speech perception among individuals with dyslexia have been observed in tasks involving the perception of syllable stress (Goswami et al., 2013), lexical tone (Tong et al., 2018), and speech in noise discrimination (Ziegler et al., 2009; Dole et al., 2012).

These results suggest that speech perception difficulties in dyslexia can manifest at different levels, such as words, syllables, phonemes, and affect multiple possible processing stages of speech, including, for example, encoding basic features, forming auditory objects, and linking to phonological representations. Understanding speech perception difficulties is particularly important in the context of dyslexia, reading acquisition, and language development, as speech perception lays the foundation for constructing accurate and stable phonological representations (Schulte-Körne et al., 1999; Goswami, 2002). These representations are essential for mapping spoken language into written symbols, which, in turn, is a foundational process in learning to read (Schulte-Körne et al., 1999; Goswami, 2002).

The behavioral studies reviewed above have extensively documented speech perception deficits as a phenomenon. However, they often leave the underlying processing stages that lead to these deficits unexamined. Electrophysiological evidence, on the other hand, offers insights into atypical speech sound processing in individuals with dyslexia, shedding light on potential deficits at different stages of speech processing (for reviews, see Bishop, 2007; Schulte-Körne and Bruder, 2010; Gu and Bi, 2020), which are reviewed next.

1.2. *Brain responses as a measure of speech perception*

Time-locked EEG activity, such as event-related potentials (ERPs), is a commonly used method to measure how the brain processes sensory stimulation. To assess different stages of auditory processing, certain obligatory and discriminatory ERP components in response to speech can be observed. The obligatory ERP components to auditory stimuli reflect automatic processing stages in the auditory cortex, elicited by any sound. Observed in adults, these early components typically include P1, N1, P2, and N2, reflecting different aspects of encoding of the auditory stimulus (Čeponienė et al., 2002). In small children under 3 years of age, typically P1 and N2 are dominant in the waveform, N1 and P2 emerging later during development, reaching an adult-like appearance during late childhood and early adolescence (Čeponienė et al., 2002).

The discriminatory responses, such as mismatch negativity (MMN or mismatch response, MMR, in newborns), P3a, and late discriminative negativity (LDN), are thought to reflect different types of change detection processes (Cheour et al., 2001; Polich, 2003, 2007; Näätänen et al., 2007). The attention-independent MMN, having its maximum peak amplitude in frontocentral areas, indicates an automatic change detection in response to a rare deviant stimulus in a series of frequent standard stimuli (Näätänen et al., 1978; Näätänen, 1990, 1995; Näätänen et al., 2007). The MMN can be followed by the P3a response, which typically shows a frontocentral scalp distribution and is elicited by a novel or deviant non-target stimuli (Polich, 2007). This component reflects the involuntary orienting of attention, and the engagement of memory-related processes involved distinguishing deviant from standard stimuli (Polich, 2007; Rosburg et al., 2018). The frontocentral LDN, which sometimes emerges in a later time window following the MMN and P3a responses, is thought to reflect higher-order cognitive processing of auditory stimuli and show developmental modulation, with its amplitude typically decreasing with age (Čeponienė et al., 2002).

Earlier ERP studies on speech discrimination in dyslexia have reported differences in both MMN and LDN components between children with and without dyslexia, suggesting atypical auditory processing and delayed higher-order evaluation in individuals with dyslexia (Schulte-Körne and Bruder, 2010). Additionally, the obligatory N1 component has shown differences between adults with and without dyslexia (for reviews, see Schulte-Körne and Bruder, 2010; Gu and Bi, 2020). Furthermore, already before learning to read (or even to speak), individuals with a familial risk of dyslexia show atypical ERPs in response to speech from newborns to 6 years-old children (for review, see Volkmer and Schulte-Körne, 2018). Next, relevant ERP research on speech processing is reviewed with a focus on two phonetic features: (1) phonemic length, which is a semantically distinguishing feature in the Finnish language (Suomi et al., 2008) and has also been linked to processing difficulties in dyslexia (Pennala et al., 2010), and (2) place of articulation, which serves as a contrastive feature across many languages (Blevins, 2004) and has also been associated with atypical speech processing in individuals with dyslexia (Kraus et al., 1996; Schulte-Körne et al., 1998).

1.2.1. *Brain responses to speech in individuals with and without dyslexia*

MMN is alongside LDN a robust indicator of auditory discrimination ability, including speech discrimination, and therefore a clear majority of ERP research on speech perception in dyslexia has used the MMN (and LDN) response to examine sound-discrimination accuracy in individuals with impaired and typical reading skills (for review, see e.g. Bishop, 2007). Where MMN reflects early, automatic detection of changes in speech sounds, the later occurring LDN reflects later, and higher-lever evaluation of differences in speech sounds. Thus, MMN and LDN are frequently studied together to provide complementary insights into auditory processing. Next, ERP findings from binaural studies using a passive oddball design are reviewed, focusing on amplitude changes in response to speech stimuli varying in phonemic length and place of articulation in individuals with and without dyslexia.

In children with dyslexia, studies on speech sounds varying in phonemic length have reported attenuated MMN amplitudes in response to vowel length changes in /ba/ vs. /baː/ contrast, when short /ba/ was played as a standard (Chobert et al., 2012), or as a deviant (Männel et al., 2017). Reduced MMN has also been found in children with dyslexia in response to speech varying in place of articulation in /ba/ vs. /da/ (Schulte-Körne et al., 1998; Lachmann et al., 2005), /da/ vs. /ga/ (Meng et al., 2005), and /ba/ vs. /ga/ contrasts (Hommet et al., 2009), the latter contrast showing also reduced amplitudes in LDN. In addition, /ba/ vs. /da/ contrast has been reported to show attenuation also in N250 amplitude in children with dyslexia (Lachmann et al., 2005). In contrast to the findings of attenuated MMN in reading-disabled children, one study has reported larger amplitudes in MMN and LDN in response to consonant length deviancy (/at:a/ vs. /ata/, short /ata/ as deviant), when comparing school-aged children with dyslexia to typically reading peers with and without familial risk for dyslexia, suggesting compensatory mechanisms in dyslexia (Lohvansuu et al., 2014).

Research findings in adults with dyslexia are partly in line with findings from children, showing diminished MMN responses to speech sounds varying in place of articulation, such as /da/ vs. /ga/ (Schulte-Korne et al., 2001) and /ba/ vs. /ga/ contrasts (Hommet et al., 2009). However, other studies have reported no group effects in change detection response amplitudes to speech sounds varying in vowel length in /ta/ vs. /ta:/ (Thiede et al., 2020) and in /tatata/ vs. /tata:ta:/ contrasts (Kujala et al., 2006) between typically reading adults and adults with dyslexia. Interestingly, N100m amplitude to the vowel /a/ in pseudoword /ata/ has been reported to be enhanced in individuals with dyslexia (Helenius et al., 2002). This finding was supported a couple of years later by a study, which found the N1 amplitude to be stronger to the first segment of pseudoword /tatata/ in adults with dyslexia (Kujala et al., 2006). Overall, research in adults is more fragmented than research done in children, not only in its findings but also in methodologies, such as the criteria for dyslexia, number of participants, age range, and stimuli used, which may affect the inconsistency of the results.

1.2.2. Brain responses to speech in individuals with and without a familial risk of dyslexia

Even before learning to read, a familial risk of dyslexia alone has been shown to be associated with impairments in speech perception (for review, see Volkmer and Schulte-Körne, 2018), which raises the question of mechanisms other than learning to read, that may underlie or contribute to speech perception deficits in dyslexia.

Studies investigating 2 to 6 months-old infants' ability to perceive speech are relatively consistent with later childhood finding, reporting smaller MMR amplitudes to speech deviancy (Leppänen et al., 2002; Van Leeuwen et al., 2006; van Leeuwen et al., 2008), or absent MMR (Leppänen et al., 2002; Schaadt et al., 2015; Van Zuijen et al., 2012) and LDN responses (Van Leeuwen et al., 2006) in responses to speech varying in both length (/ata/ vs. /at:a/) and place of articulation (/da/ vs. /ga/ and /bAk/ vs. /dAk/) in infants at risk for dyslexia when compared to controls. However, MMR responses have also been observed to be larger in infants at risk to deviant speech sounds varying in length (ka:a/ vs. /ka/) (Leppänen et al., 1999). In newborns, and at the age of 1 month, no group effects have been found in response to changes in phonemic length (/ka:/ vs. /ka/) (Pihko et al., 1999) or in responses to speech stimuli varying in place of articulation (/da/ vs. /ga/) (Schaadt et al., 2015).

In preschool-aged children, both diminished and enhanced responses to speech in children with a risk of dyslexia have been reported, as well as group differences not reaching significance (for review, see Volkmer and Schulte-Körne, 2018). Diminished MMR was found in 6-year-old children at risk for dyslexia at the beginning of reading instructions to speech sounds varying in place of articulation in /ga/ vs. /ka/ (Bitz et al., 2007), and /bə/ vs. /də/ contrasts (Noordenbos et al., 2012). Enhanced responses in children at risk for dyslexia have been found in 6-year-old children without any formal reading instructions to speech varying in place of articulation (/ba/ vs. /ta/ and /da/, /ta/ and /da/ as deviants) in an early MMR time window (Maurer et al., 2003), and phonemic length (/ata/ vs. /at:a/, /ata/ as standard), in the latter study group differences occurring only to standard stimulus and in the N250 time window (Hämäläinen et al., 2013). In addition, one study reported no significant group differences in MMR responses to vowel length changes between children at risk and controls (Lovio et al., 2010), indicating that findings in this age range remain somewhat mixed.

1.3. Brain responses to speech linked to reading

In addition to group differences reviewed above, associations between brain responses and reading, or reading-related skills, have been studied (for review, see Volkmer and Schulte-Körne, 2018). Brain responses to speech sounds measured in newborns have been shown to predict later reading skills, with larger MMR amplitudes associated with better pre-reading skills, such as better RAN task scores at 28 months and 4–5 years, and better letter knowledge scores at 4–5 years (Navarrete-Arroyo et al., 2024). In contrast, MMR measured at the age of 1 month and 2 months, did not show any significant correlations with later reading ability (Schaadt et al., 2015; Van Zuijen et al., 2013, respectively). However, the larger MMR measured later in the same infants at the age of five months by Schaadt et al. (2015) was associated with better reading comprehension in grades 5–6, at the mean age of 11.5 years (Schaadt et al., 2015). Further, larger ERP amplitudes to standard stimuli in 6-month-old infants have been associated with faster reading speed at 14 years, better phonological skills, and faster rapid naming speed at preschool age (Lohvansuu et al., 2018).

Studies conducted with preschool and kindergarten-aged children further support the suggestion of a developmental link between speech perception and phonological and reading skills. In the study by Maurer et al. (2009) preschool-aged children with a risk for dyslexia showed stronger left lateralization of the LDN response being associated with better reading fluency in second, third, and fifth grade (at 8, 9, and 11 years respectively) (Maurer et al., 2009). In addition, smaller N250 measured before school entry in response to standard stimuli was shown to correlate with better reading accuracy and reading speed at the end of grade 2, at the average age of 9 years (Hämäläinen et al., 2013).

Overall, the research findings show that larger MMR responses to speech measured in infancy are quite consistently associated with better reading and related abilities later in childhood, which is also supported by studies using non-speech stimuli. Larger ERP amplitudes measured in newborns have been shown to be associated with better reading speed and spelling accuracy at around 9 years of age (Leppänen et al., 2010), as well as larger responses measured at the age of 17 months with better word and pseudoword reading fluency at the age of 9 (Van Zuijen et al., 2012). In addition, the MMN amplitude of 3-year-old children was linked to later reading skills, with larger amplitudes predicting better word reading fluency at the end of grade 2 (Plakas et al., 2013). However, the evidence from studies conducted closer to school entry is somewhat more variable: both larger and smaller amplitudes have been linked to better reading abilities later, as well as stronger left lateralization of the responses, which makes the direction of the association inconsistent.

Some evidence for a continued link between brain responses to speech and reading or reading-related skills also exists in school-age children and adults. In school-aged children, ERPs to phonemic length deviations observed at the age of 9.5 years were found to be positively associated with reading fluency and accuracy, as well as writing accuracy, particularly in the dyslexia group (Lohvansuu et al., 2014). Notably, 82 of the participants in that study also participated in the present study, but the measurements were conducted at an earlier developmental stage. In adults, associations between larger left-hemispheric mismatch field (MMF, magnetic counterpart of MMN) and better working memory skills have been found in adults with dyslexia as well as in controls (Thiede et al., 2020). In addition, larger right-hemispheric MMF in the same study was associated with better reading accuracy and speed within the dyslexia group, suggesting a right-hemispheric language processing compensatory mechanism in dyslexia (Thiede et al., 2020). However, given the limited research on how brain responses to speech relate to reading and reading-related skills in adulthood, this remains an important question for further research.

Based on the evidence reviewed here, it seems that school-aged children with dyslexia, as well as infants at risk for dyslexia, quite consistently show diminished brain responses to speech changing in phonemic length and category when compared to controls. In adults with dyslexia and preschool-aged children with risk for dyslexia, the findings are less consistent, one showing larger or smaller responses, while the others report no differences between individuals with dyslexia and control groups. However, the earlier findings from the Jyväskylä Longitudinal Study of Dyslexia (JLD) sample, the longitudinal sample studied here, showed enhanced brain responses in children with dyslexia both at kindergarten and school age (Hämäläinen et al., 2013; Lohvansuu et al., 2014). Further, brain responses at different ages seem to predict and be associated with reading skills, though the nature of this relationship in adulthood remains not well understood.

1.4. Research questions

Two research questions were examined:

> (RQ1) Group differences in ERPs to speech sounds were observed in childhood in the JLD study at the age of 9.5 years. Here we examine whether the group differences between individuals with and without dyslexia in ERPs to the same speech sounds persist into adulthood?
> (RQ2) Previous JLD and other studies have found associations with concurrently measured reading and related skills and ERP amplitudes. Here we examine these associations in adulthood using combined M/EEG source activity to have the best possible estimates of brain activity related to processing of speech sounds.

To address RQ1, and ensure comparability with the childhood data, participants were divided into three groups based on their childhood reading performance, allowing for the investigation of group differences in ERPs as done in the earlier study (Lohvansuu et al., 2014). For RQ2, no grouping was performed based on the reading skills, instead reading skills were examined as a continuum. Investigating reading skill as a continuum, rather than comparing distinct groups, allows capturing the full range of individual differences in reading ability.

Based on the previous studies on adults and children reviewed above the direction of the effect is difficult to predict as the majority of studies show smaller amplitudes for the change detection responses compared to typically reading controls (e.g. Lovio et al., 2010; Schulte-Körne et al., 1998; for review, see Gu and Bi, 2020), but the earlier studies of the longitudinal sample studied here showed larger amplitudes for the obligatory and change detection responses in individuals who developed dyslexia (Hämäläinen et al., 2013; Lohvansuu et al., 2014). Based on the research evidence from the relationship between brain responses to speech and behaviorally assessed reading skills, associations between brain activity and reading fluency and accuracy, rapid naming as well as working memory were expected (e.g. Hämäläinen et al., 2013; Lohvansuu et al., 2014, 2018; Thiede et al., 2020).

2. Material and methods

2.1. Participants

The participants of this study belong to the JLD, a prospective follow-up study from birth to young adulthood investigating the reading development of participants with and without family risk of dyslexia. At the early adulthood data collection, a total number of 98 participants (60 female) returned for participation at age 26 to 28 ($M = 27.21$, $SD = 0.98$). Seven participants were excluded from the analysis for various reasons: technical issues (3 participants), control participants with poor reading skills (2 participants), hearing problems (1 participant), and potential neurological issues (1 participant). The previous brain data collection phase of JLD took place at the age of 9.5 years ($N = 139$) (Lohvansuu et al., 2014; for reviews, see Lyytinen et al., 2005, 2015; Leppänen et al., 2012; Lohvansuu et al., 2021).

For RQ1 the ERP data of 91 (57 female), and for RQ 2 the combined M/EEG data of 84 adults (52 female) were included in the study. The number of participants differs between RQ1 and RQ2 due to the exclusion of 7 participants with poor MEG data quality. Participants did not report having any neurological disorders; they had a childhood IQ of at least 80, as tested at 8 years with the Wechsler Intelligence Scale for Children – Third Edition (WISC-III: Wechsler, 1991). They were screened for normal hearing (average hearing threshold for both ears at/below 25 dB (BSA)) with an audiometer before the M/EEG recordings. The study was approved by the local ethics committee of the University of Jyväskylä and written informed consent was obtained from all the participants.

For RQ1, participants were divided into groups based on their family risk status for dyslexia (for more information, see Leinonen et al., 2001) and reading skills assessed at the end of Grade 2 as follows (for more information, see Eklund et al., 2013): adults with childhood reading difficulties and family risk (RDFR, $N = 17$), childhood typical readers with family risk (TRFR, $N = 37$), and childhood typical readers from the control group without any family risk for dyslexia (TRC, $N = 37$). Among the control participants, there were two individuals without a family risk for dyslexia who fell below the reading difficulty criteria and were thus excluded from the analysis. To ensure comparability, the grouping criteria were aligned with those used in childhood group comparisons reported in earlier publication (Lohvansuu et al., 2014). For RQ2, no grouping was applied, as reading and related skills were treated as continuous variables. Participants' characteristics can be found in Table 1.

A one-way ANOVA was conducted to examine the effect of the group on reading and related scores in adulthood. The analysis revealed a significant group effect on *reading fluency* ($F(2, 88) = 24.87$, $p < .001$, $\eta^2 = 0.36$), *reading accuracy* ($F(2, 88) = 9.67$, $p < .001$, $\eta^2 = 0.18$), and *rapid naming* ($F(2, 86) = 7.70$, $p < .001$, $\eta^2 = 0.15$). Levene's test for homogeneity of variances indicated that the assumption of equal variances was met for reading fluency ($p = .23$), rapid naming ($p = .31$), therefore Tukey post hoc tests were used for pairwise comparisons. Tukey tests revealed that the dyslexia group (RDFR) scored significantly lower in reading fluency and rapid naming than the two other groups: TRFR (reading fluency: $p < .001$; rapid naming: $p = .012$) and TRC (reading fluency: $p < .001$; rapid naming: $p < .001$). For the reading accuracy comparison, Levene's test indicated unequal variances ($p = .01$), therefore Games-Howell post hoc tests were used. The tests indicated that the dyslexia group scored significantly lower than the two other groups (TRFR: $p = .016$; TRC: $p = .008$). No statistically significant differences were found between the other groups.

The development of reading fluency and accuracy from the age of

Table 1
Description of the participants and means (standard deviations) for behavioral measures.

	RDFR (N = 17)	TRFR (N = 37)	TRC (N = 37)
Gender (female/male)	12/5	23/14	22/15
Age at M/EEG recordings (years)	26.60 (0.94)	27.33 (1.10)	27.37 (0.78)
Reading fluency (composite score)	47.83 (8.52) ˆ	65.66 (12.29)	70.02 (10.30)
Text reading fluency: correctly read words per minute (at 26–28 y)	74.88 (11.12) ˆ	91.85 (16.04)	97.84 (14.62)
Pseudoword text reading fluency: correctly read words per minute (at 26–28 y)	41.11 (8.64) ˆ	59.26 (14.71)	64.72 (14.85)
Word list reading fluency: correctly read words per minute (at 23/26 y)	56.11 (13.02) ˆ	77.66 (16.28)	81.53 (15.24
Pseudoword list reading: correctly read words per minute (at 23/26 y)	21.78 (7.06) ˆ	34.43 (9.47)	36.13 (9.35)
Reading accuracy (composite score)	89.29 (6.44) ˆ	94.28 (3.91)	95.01 (4.26)
Text reading accuracy: percentage of correct words (at 26–28 y)	97.16 (1.92) #	98.38 (1.57)	98.54 (1.20)
Pseudoword text reading accuracy: percentage of correctly read words (at 26–28 y)	93.65 (6.90)	95.47 (4.15)	95.59 (3.66)
Pseudoword list reading accuracy: percentage of correctly read words (at 23/26 y)	77.06 (14.62) ˆ	88.98 (7.84)	89.91 (9.57)
Rapid naming speed (composite score) (at 26–28 y)	30.84 (3.84) ¤	27.00 (5.10)	25.74 (4.10)
Naming of objects: time spent (seconds)	37.42 (4.37) %	33.93 (6.35)	32.42 (4.25)
Working memory (at 26–28 y)	23.59 (3.41)	25.97 (5.72)	27.00 (5.41)

Note: ˆ = RDFR < TRFR and TRC; ¤ = RDFR > TRFR and TRC; # = RDFR < TRC; % = RDFR > TRC.

9.5 years to 23–28 years was further examined. These analyses included 82 participants who had complete data at both timepoints. As shown in Fig. 1, childhood grouping still appears relevant in adulthood. To evaluate developmental change and group differences, a repeated measures ANOVA was conducted with Time (childhood vs adulthood) as a within-subject factor, Group (three levels) as a between subject factor, and reading fluency and reading accuracy as dependent variables. For reading fluency, the analysis revealed no significant effect of time (F(1, 79) = 1.45, $p = .233$, $\eta^2_p = 0.018$), indicating that reading fluency did not significantly change between childhood and adulthood. However, there was a significant main effect of group (F(2, 79) = 29.56, $p < .001$, $\eta^2_p = 0.43$), suggesting that the groups differed in overall reading fluency. For reading accuracy there was a main effect of time (F(1, 79) = 5.57, $p = .021$, $\eta^2_p = 0.06$), indicating improvement in accuracy over time. A significant main effect of group was also found (F(2, 79) = 16.69, $p < .001$, $\eta^2_p = 0.30$), showing that the groups differed in overall accuracy. There was no significant interaction effects between time and group for reading fluency (F(2, 79) = 0.30, $p = .743$, $\eta^2_p = 0.01$) or reading accuracy (F(2, 79) = 1.47, $p = .236$, $\eta^2_p = 0.04$), indicating that the pattern of change over time was similar across groups.

2.2. *Behavioral and cognitive assessments*

In adulthood, reading and related skills were assessed in two waves (at age 23 years and between 26 and 28 years of age). Behavioral assessments at age 23 were conducted in person at the laboratory of the University of Jyväskylä, whereas at ages 26–28 assessments were in majority conducted remotely due to pandemic circumstances. The cognitive data collection at ages 26–28 was conducted in proximity to the MEG/EEG recordings. All assessments were administered by a trained experimenter, and the sessions were recorded for later scoring. Composite scores of *reading fluency*, *reading accuracy*, and *rapid naming of objects and letters* (RAN) were calculated for the correlation analysis.

2.2.1. *Reading skills*

Reading skills were assessed by (1) *oral text reading* (Leinonen et al., 2001), (2) *oral pseudoword text reading* (Eklund et al., 2015*)*, (3) *oral word list reading* (Nevala et al., 2006), *and* (4) *oral pseudoword list reading* (Nevala et al., 2006). Participants were instructed to read each text and word list aloud as accurately and fast as possible.

Oral text reading included 16 sentences in three paragraphs and a total of 207 words. Oral pseudoword text reading consisted of 38 pseudowords presented across eight sentences forming one paragraph. The text followed the structure of the Finnish language but had no meaning. In oral word list reading tasks, participants read a list of 30 words and 30 pseudowords, which were similar in length and structure to the real word items.

From each of the reading tasks, two different measures were obtained: reading fluency (correctly read words per minute) and reading accuracy (percentage of correctly read words). These were transformed into z-scores, and the normal distribution of each variable was checked. The violation of distribution led to the exclusion of the reading accuracy obtained by word list reading due to the ceiling effect. Consequently, two composite scores (average of z-scores) were formed: (1) Reading fluency composite score included the reading fluency measures of text, pseudoword text, word list, and pseudoword list reading (Cronbach's Alpha = 0.90). (2) Reading accuracy composite score included the reading accuracy measures of text, pseudoword text, and pseudoword list reading (Cronbach's Alpha = 0.78).

2.2.2. *Reading related skills*

Measures of reading-related skills for RQ2 were rapid naming (RAN), *working memory* (Digit Span), and *phonological processing* (phoneme deletion). The normal distribution of each variable was checked, and the phoneme deletion score was excluded from the analysis due to the ceiling effect.

To assess the ability to name symbols accurately and rapidly, two subtests of rapid automatized naming test (see Denckla and Rudel, 1976) were carried out at ages 26–28: (1) RAN objects consisted of pictures of 5 different objects, each repeated 10 times, whereas (2) RAN letters included 5 different letters, each repeated 10 times. In both sub tests, the items were arranged in five rows, each containing 10 items, resulting in a total of 50 items per subtest (Ahonen et al., 1999). To ensure the correct understanding of each item in the lists, participants were asked to repeat them aloud before the test started. After that, participants were instructed to name each item on the list aloud as quickly and accurately as possible and in case a mistake occurred to correct it and continue naming. The naming speed of objects and letters showed internal consistency (Cronbach's Alpha = 0.79) and were normally distributed, and therefore a composite score for rapid naming speed was calculated as the average of the time taken in RAN objects and letters.

The working memory of the participants was assessed by Digit Span (WAIS-IV; Wechsler, 2008), which was composed of forward, backward, and sequencing sub-tests. In the task, participants repeat a number sequence stated by the experimenter according to the given instructions, either in the same order, in reverse order, or arranged in numerical order. The working memory score consisted of the total score from all these test components (total raw score range 0–48).

2.3. *M/EEG procedure*

2.3.1. *Recordings*

Brain responses were recorded simultaneously with a 306-sensor (102 magnetometers and 204 planar gradiometers) MEG system

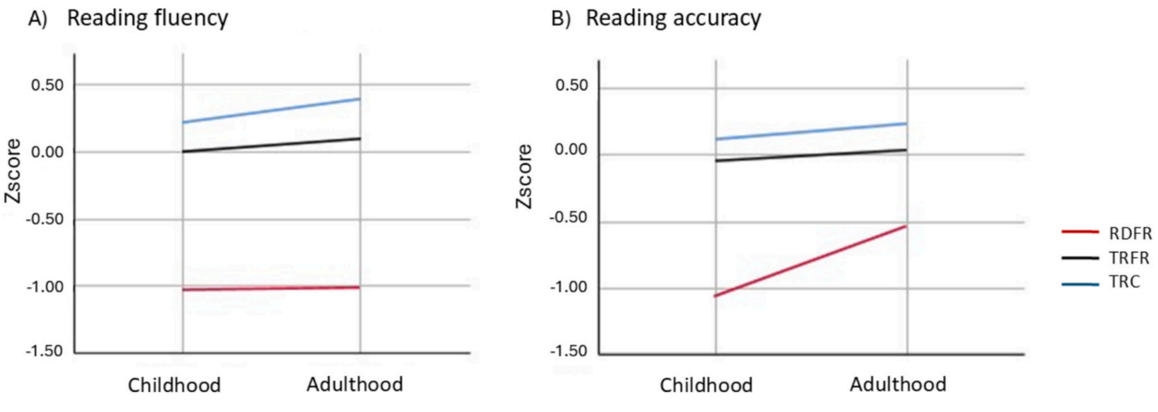

Fig. 1. Development of reading fluency and accuracy from childhood to adulthood by group ($N = 82$). The red line represents the dyslexia (RDFR) group, the black line, the risk (TRFR) group, and the blue line the control group (TRC). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

(Elekta Neuromag®TRIUX, Helsinki, Finland) and a 64-channel MEG-compatible EEG cap (BrainCap, EASYCAP GmbH) using the integrated EEG amplifier of the TRIUX MEG system. EEG was online referred on the right earlobe and impedances were maintained below 20 kΩ, in accordance with manufacturer recommendations. Both data streams (EEG/MEG) were recorded with a sampling rate of 1000 Hz, and an online bandpass filter of 0.01–100 Hz. Prior to MEG/EEG recording, the three anatomical landmarks (nasion, the left preauricular point, and the right preauricular point), electrode positions and positions of the head position indicator (HPI) coils as well as head shape were digitized (Polhemus Fastrak). Continuous HPI was collected during the recording.

2.3.2. *Design*

In the experiment, participants were passively listening to speech sounds presented in an oddball paradigm (Fig. 2) while watching a silent movie. They were instructed not to pay attention to the speech sounds. In the paradigm, the long /at:a/ was presented as a standard (80%, 1000 trials) and the short /ata/ (10%, 125 trials) and long /ap:a/ (10%, 125 trials) as deviants. The total duration of the task was 25 min.

2.3.3. *Stimuli*

The three naturally produced pseudowords used in the experiment consisted each of two syllables, varying in the phonemic length, and articulation place, which are semantically distinguishing features in Finnish language. The standard /at:a/, and the deviant /ap:a/ had a total duration of 450 ms, whereas the short /ata/ as another deviant stimulus, had a total duration of 300 ms (Fig. 3). The duration of the first syllable for each stimulus was 72 ms, including the vowel /a/, and the initial glottal stop. The second syllable, including the explosion of /t/ or /p/ and the vowel /a/, was 133 ms in total duration. Only the length of the silent gap between the vowel and the following stop consonant differed between the short /ata/ (95 ms) and long /at:a/ (255 ms) stimuli. The sound onset of the stimuli was evaluated through microphone recordings of the stimuli presentation, revealing a constant 34 ms delay after the trial onset, caused by the stimulation system. The stimuli were presented with an interstimulus interval (ISI) of 610 ms and delivered through in-ear headphones (Etymotic Research tube system). The intensity of the stimuli was 73.5 ± 0.5 dB(A), which was measured with Brüel & Kjær sound level meter (type 2235).

2.4. *M/EEG analysis*

2.4.1. *Preprocessing*

MEG data were first preprocessed using MaxFilter™ (Version 3.0.17; MEGIN Oy, Finland), applying temporal signal space separation (tSSS) to suppress external interference, interpolate bad MEG channels, and compensate for head movements. The combined M/EEG data were subsequently analyzed using MNE-Python (Version 1.3). Data were resampled to 500 Hz and low-pass filtered at 35 Hz using a zero-phase FIR filter with a Hamming window. Bad EEG channels were identified by visual inspection (mean = 0.18 channels, SD = 0.44, range: 0–2).

Cardiac and ocular artifacts were removed using Independent Component Analysis (FastICA; Hyvarinen, 1999). To optimize ICA decomposition, the analysis was performed on a copy of the data high-pass filtered at 1 Hz, and the resulting unmixing matrix was then applied to the original low-pass filtered data (cf. Winkler et al., 2015). ICA was conducted jointly on MEG (magnetometers and gradiometers) and EEG channels, retaining components that cumulatively explained 98% of the variance. Epochs exceeding 2000 fT/cm for gradiometers, 4 pT for magnetometers, or 250 μV for EEG channels were excluded from ICA computation. Artifact components were identified using automated correlation with EOG (EOG001) and ECG (ECG003) reference channels, supplemented by visual inspection of component topographies and time courses. On average, 2.21 (SD = 0.44) EOG-related and 1.81 (SD = 0.68) ECG-related components were removed per participant.

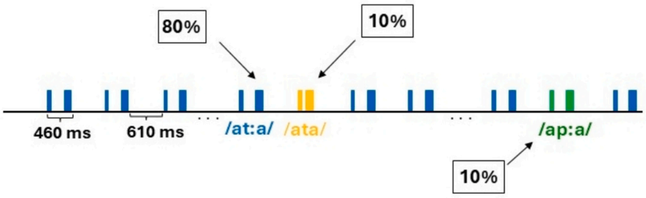

Fig. 2. Illustration of the paradigm (Modified from Lohvansuu et al., 2014).

Following ICA-based artifact correction, continuous EEG data were high-pass filtered offline in MNE-Python using a zero-phase (non-causal) windowed-sinc FIR filter (firwin algorithm, Hamming window). The lower passband edge was set to 0.53 Hz with a transition bandwidth of 0.53 Hz, yielding a half-amplitude (−6 dB) cutoff at 0.27 Hz and a half-power (−3 dB) cutoff at 0.34 Hz. The Hamming window provided a passband ripple of 0.0194 and a stopband attenuation of 53 dB. The filter length was 3115 samples (6.23 s at 500 Hz). Filtering was applied to continuous data using reflection-based padding prior to segmentation into epochs from −300 to 1060 ms relative to stimulus onset. Baseline correction was applied using the −50 to 0 ms pre-stimulus interval. Bad EEG channels were then interpolated using spherical spline interpolation. Epochs with amplitudes exceeding 1000 fT/cm for gradiometers, 4 pT for magnetometers, or 150 μV for EEG channels were rejected. Event-related potentials (ERPs) and event-related fields (ERFs) were computed by averaging the remaining epochs within each condition, after which EEG data were re-referenced to the common average. On average, 914.2 trials (SD = 165.0) were accepted for the standard condition (/at:a/), 113.5 trials (SD = 20.9) for /ata/, and 112.9 trials (SD = 20.2) for /ap:a/ .

For RQ1, the sensor level analysis included only EEG data to ensure comparability to the results obtained at 9.5 years, and for RQ2 the source level analysis of ERPs and ERFs included both data streams (MEG/EEG).

2.4.2. *Source analysis*

For RQ2, combined EEG and MEG source reconstruction was carried out for each condition (/at:a/, /ata/, and /ap:a/). EEG and MEG together provide complementary information improving the source estimation (Ahlfors and Hämäläinen, 2012). The source estimation procedure followed the established M/EEG minimum-norm estimation pipeline implemented in MNE-Python (Gramfort et al., 2013, 2014). The time window of 50–900 ms and all sensors were selected for the analysis to explore the source localization of each component of the waveform elicited by the speech stimuli. First, the source space was created using an anatomical MRI template (fsaverage, FreeSurfer RRID:SCR_001847). Using an automated method detailed by Houck and Claus (2020), the template brain was uniformly scaled and then aligned with each participant's digitized head points (Houck and Claus, 2020). For the forward model, the cortical source space was generated using the “ico4” option, yielding approximately 2562 sources per hemisphere with an average inter-source distance of 6.2 mm. A single-layer boundary-element model was constructed under the assumption of homogeneous conductivity within the intracranial volume. The noise covariance matrix was estimated from the pre-stimulus baseline periods. Finally, the inverse operator (MNE, loose=‘auto’, depth=0.8) was created with whitening transformation based on the noise covariance matrix and applied to the evoked data to compute source estimates.

2.4.3. *Statistical analysis*

For RQ1, group differences in brain responses to speech sounds were assessed using data-driven threshold-free cluster enhancement (TFCE, h = 2.00, e = 0.50, start = 0, step = 0.2; Smith and Nichols, 2009) combined with permutation testing, as implemented for EEG data in MNE Python (Mensen and Khatami, 2013). TFCE finds and enhances clusters of statistically significant effects across time and sensors without

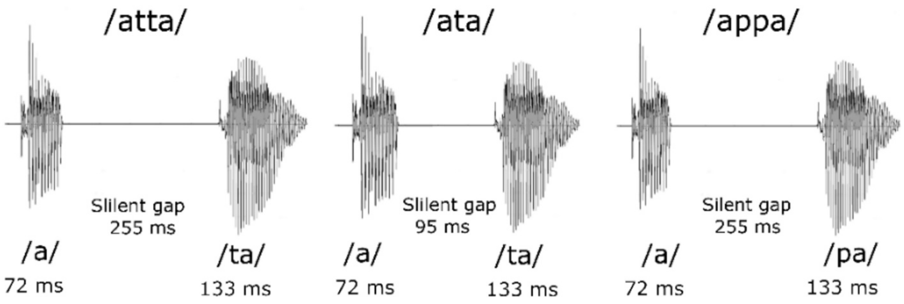

Fig. 3. Sound waveforms of the stimuli.

requiring a predefined threshold, while permutation testing provides robust control for multiple comparisons (Smith and Nichols, 2009;

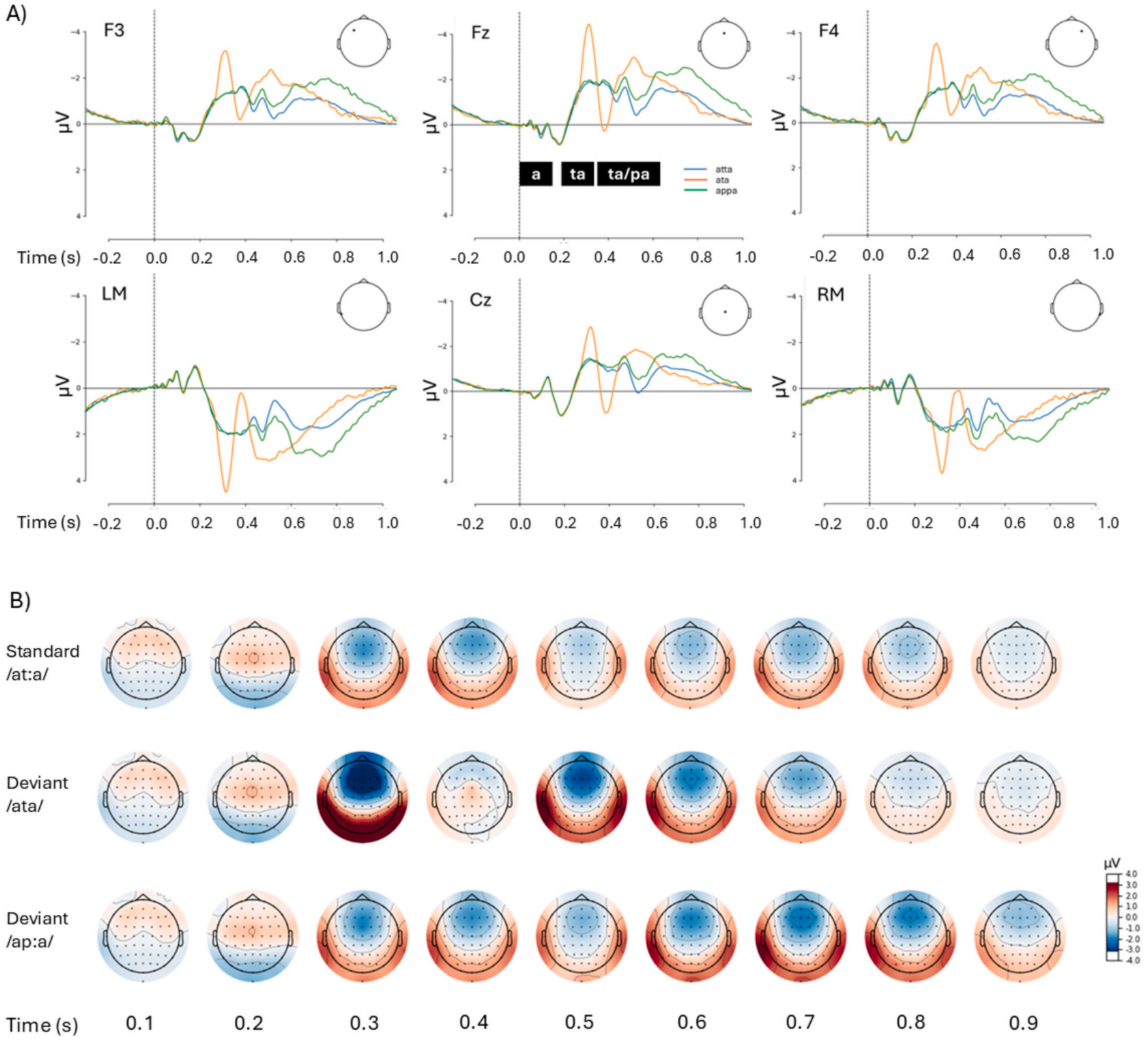

Fig. 4. (A) The grand-average ERP waveforms across all participants (N = 91) for all conditions are shown at channels F3, Fz, F4, left mastoid (LM), Cz, and right mastoid (RM). The blue curve represents the brain response to the standard (/at:a/) sound, the orange curve to the phonemic length-deviant sound (/ata/), and the green curve to the other deviant, changing in place of articulation (/ap:a/). (B) Topography plots across all participants (N = 91) showing responses to all stimuli. The upper row displays responses to the standard stimulus (/at:a/), the middle row to the deviant differing in duration (/ata/), and the bottom row to the deviant differing in place of articulation (/ap:a/). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Mensen and Khatami, 2013). The number of permutations was set to 5000 for each contrast, analysis time window to 50–900 ms, and statistical alpha level to 0.05 (for further information on the permutation testing, see e.g. Maris and Oostenveld, 2007; Smith and Nichols, 2009). In total, nine comparisons were performed (3 groups × 3 stimuli).

For RQ2, correlation analysis using cluster-based permutation statistics was carried out in MNE Python to investigate the associations between the power of source-localized combined ERP/ERF responses to all the speech sounds and reading accuracy, speed and related skills (working memory, RAN) among participants, resulting in 12 correlations (3 stimuli × 4 cognitive measures). The same analysis time window of 50–900 ms as used in RQ1 was used in the correlation analysis.

3. Results

3.1. Group differences in ERPs to speech sounds

3.1.1. Sensor level grand average waveforms

The grand-average ERP waveforms and topographies across all the participants ($N = 91$) for all the conditions followed a typical time course of auditory responses (Fig. 4, panel A; see Fig. 4, panel B for topographies). The latencies measured at channel Fz for obligatory responses to the first syllable were as follows (taking the 34 ms stimulus onset delay into account): the N1 peaked at 93 ms and P2 at 151 ms. Change detection responses for the deviant /ata/ stimulus were the first wave reflecting the MMN peaking at 281 ms, (from deviancy onset 114 ms) the second the P3a (351 ms; from deviancy onset 184 ms), and the last late negativity reflecting either LDN or RON, peaking at 483 ms after stimulus onset (from deviancy onset 316 ms). For deviant /ap:a/ the long-lasting change detection response was observed, where there was

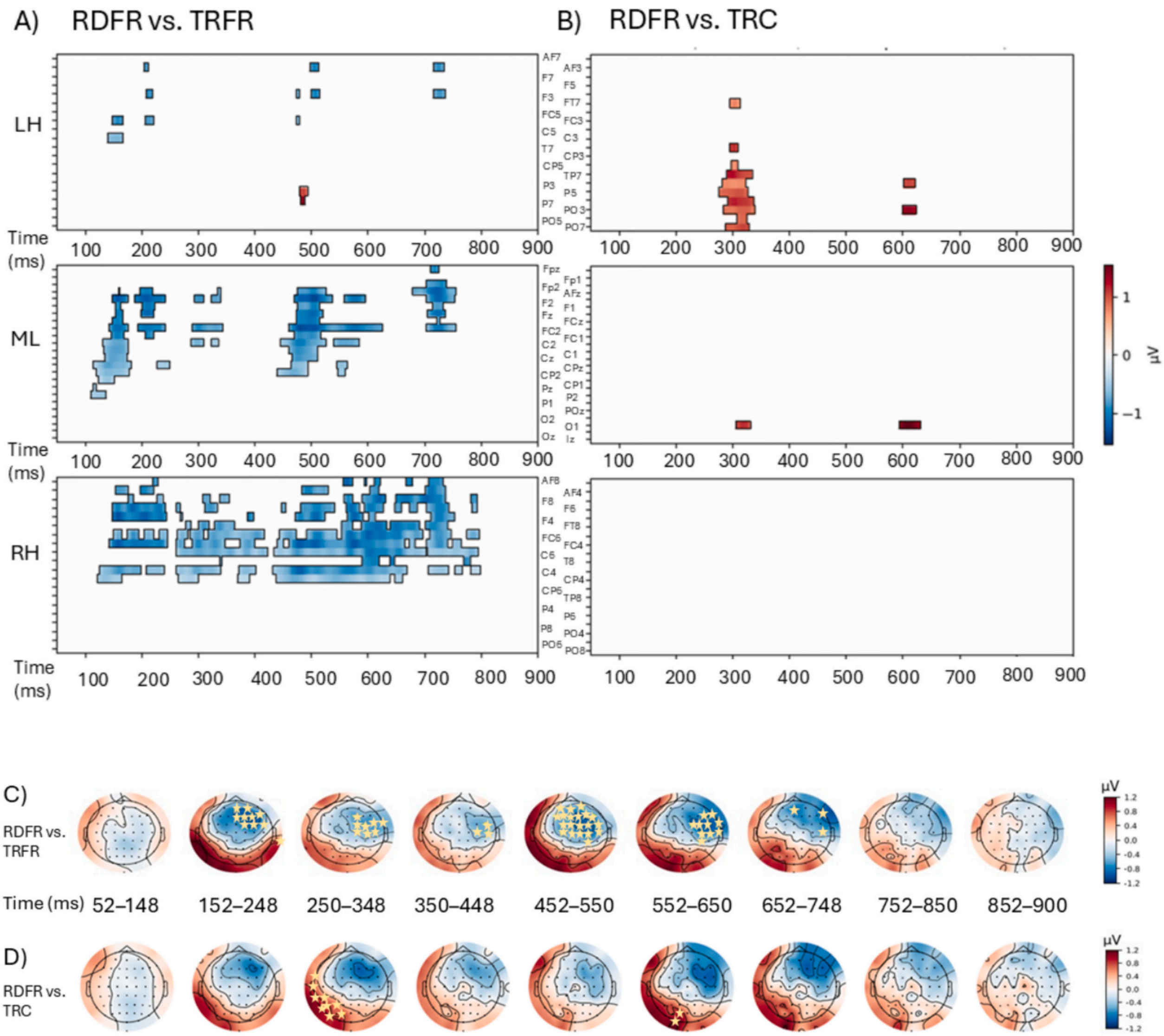

Fig. 5. Statistically significant group differences in EEG responses revealed by cluster permutation tests with TFCE (shown as masked colour boxes) between (A) RDFR vs. TRFR and (B) RDFR vs. TRC, illustrated across channels grouped in the left hemisphere (LH, upper row), midline (ML, middle row), and right hemisphere (RH, bottom row). (C) Topography plot showing the temporal evolution of the significant effects for the RDFR vs. TRFR comparison. Yellow stars indicate the channels that showed statistically significant group differences in response to deviant /ap:a/ sound (corrected $p < .05$; RDFR > TRFR). (D) Topography plot showing the temporal evolution of the significant region for the RDFR vs. TRC comparison. Yellow stars indicate the channels that showed statistically significant group differences in response to deviant /ap:a/ sound (corrected $p < .05$; RDFR > TRC). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

no clear distinction between MMN and LDN (437–593 ms; from deviancy onset 110–266 ms).

3.1.2. *Sensor level group comparisons*

Statistically significant differences were found in responses to deviant speech sound /ap:a/, (i.e. change in articulation place) when RDFR was compared to both TRFR and TRC. Compared to TRFR, the RDFR group exhibited stronger responses recorded by midline (ML) and left (LH) and right (RH) hemisphere channels (see Fig. 5, panel A), as revealed by the TFCE cluster permutation test. These differences emerged starting from 152 ms and persisted until 800 ms (see Fig. 5, panel A & C). When compared to TRC, the RDFR group exhibited stronger responses at channels located in the left hemisphere (see Fig. 5, panel B), particularly around the 300 ms and 600 ms time windows (see Fig. 5, panel B & D). No statistically significant difference was observed between the TRC and TRFR groups for /ap:a/. Further, no statistically significant differences were observed between groups for responses to deviant /ata/ or standard /atta/. Grand average ERP waveforms in the three groups can be seen in Fig. 6. Fig. 7 presents the waveforms categorized by speech sounds, with each group displayed together in one plot across different channels.

3.1.3. *Associations between brain activity and reading skills*

Brain activity was predominantly located in the left and right auditory cortices for all the speech sounds (Fig. 8). No statistically significant correlations were found between brain responses (combined M/EEG source level activation) and behaviorally assessed reading (reading fluency or accuracy) and related skills (RAN or verbal working memory). Histograms of point-wise correlations between neural activity and behavioral measures revealed no evidence of systematic brain-behavior associations (Fig. 9). Across all 12 combinations of behavioral measures and stimulus conditions, correlation distributions were approximately normal and centered near zero (mean r: -0.05 to $+0.09$). The standard deviations were consistent across analyses (SD: 0.086–0.106), and the proportion of correlations exceeding the uncorrected significance threshold ($|r| > 0.215$) ranged from 1.4% to 11.8%—values consistent with or slightly above chance expectations (5%). These results support the cluster-based permutation findings by demonstrating that correlations were weak, unsystematic, and broadly distributed around zero across the source space.

4. Discussion

In this study, two research questions were addressed. First, the persistence of group differences in ERPs to speech sounds (deviants /ata/ and /ap:a/ among standard /at:a/ in a passive oddball paradigm) was investigated from childhood to adulthood among individuals with and without childhood dyslexia and with or without background of familial risk for dyslexia. Group differences were found for deviant /ap:a/ (a change in place of articulation) when childhood dyslexia group with family risk (RDFR) was compared to typically reading peers with family risk (TRFR), and to controls (TRC). No group differences were found between TRFR and TRC groups. For deviant /ata/ (a change in phonemic length), no group differences were observed unlike at school-age when the brain responses differed between children with dyslexia (RDFR) and typically reading groups (TRFR & TRC).

In addition, associations between ERPs/ERFs and reading fluency, reading accuracy, rapid naming, and working memory were assessed. Associations found at school-age between brain responses to the speech sounds and the reading (real word and pseudoword list reading accuracy and fluency) were no longer found in adulthood. Neither were associations found between brain response and reading related skills. Results showed that there are group differences in speech processing, but the difference found in adulthood is not present for the same stimulus as in childhood. Based on the results of the study, group differences seem to change during development. This might be on the one hand due to continued exposure to speech which could influence the ability to perceive speech sounds, and on the other hand due to different linguistic features being more relevant than the others at different stages of development.

Alongside these neural level findings, the behavioral data on reading fluency and accuracy showed stable group differences from childhood to adulthood. This pattern suggests that early differences did not widen over time, which contrasts with the view that early differences typically expand across development (often referred to as the Matthew effect; Stanovich, 1986). Importantly, it should be acknowledged that children in the dyslexia groups have likely received varying levels of educational and intervention support across the follow-up period. In addition, as part of the research program, some participants took part in interventions targeting speech sound perception and temporal processing. Although support may have influenced the developmental trajectories of some participants, group differences in reading outcomes remained evident, suggesting that variation in support or intervention alone cannot account for the patterns observed.

4.1. *Group differences change from childhood to adulthood*

Adults in the JLD sample with childhood dyslexia were found to have

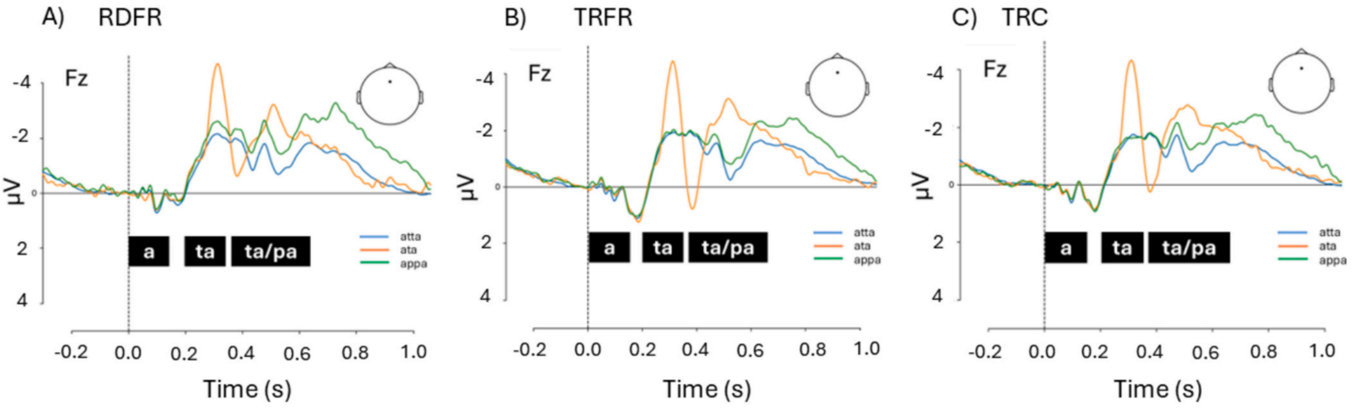

Fig. 6. ERP waveforms at Fz for the responses to all of the stimuli separately for each group are shown: On the left (A) individuals with a familial risk for dyslexia and childhood reading disability (RDFR, $N = 17$), (B) typically reading individuals with a familial risk for dyslexia (TRFR, $N = 37$), and (C) typically reading controls (TRC, $N = 37$). The blue curve represents the brain response to the standard (/at:a/) sound, the orange curve to the phonemic length-deviant sound (/ata/), and the green curve to the other deviant, changing in place of articulation (/ap:a/). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

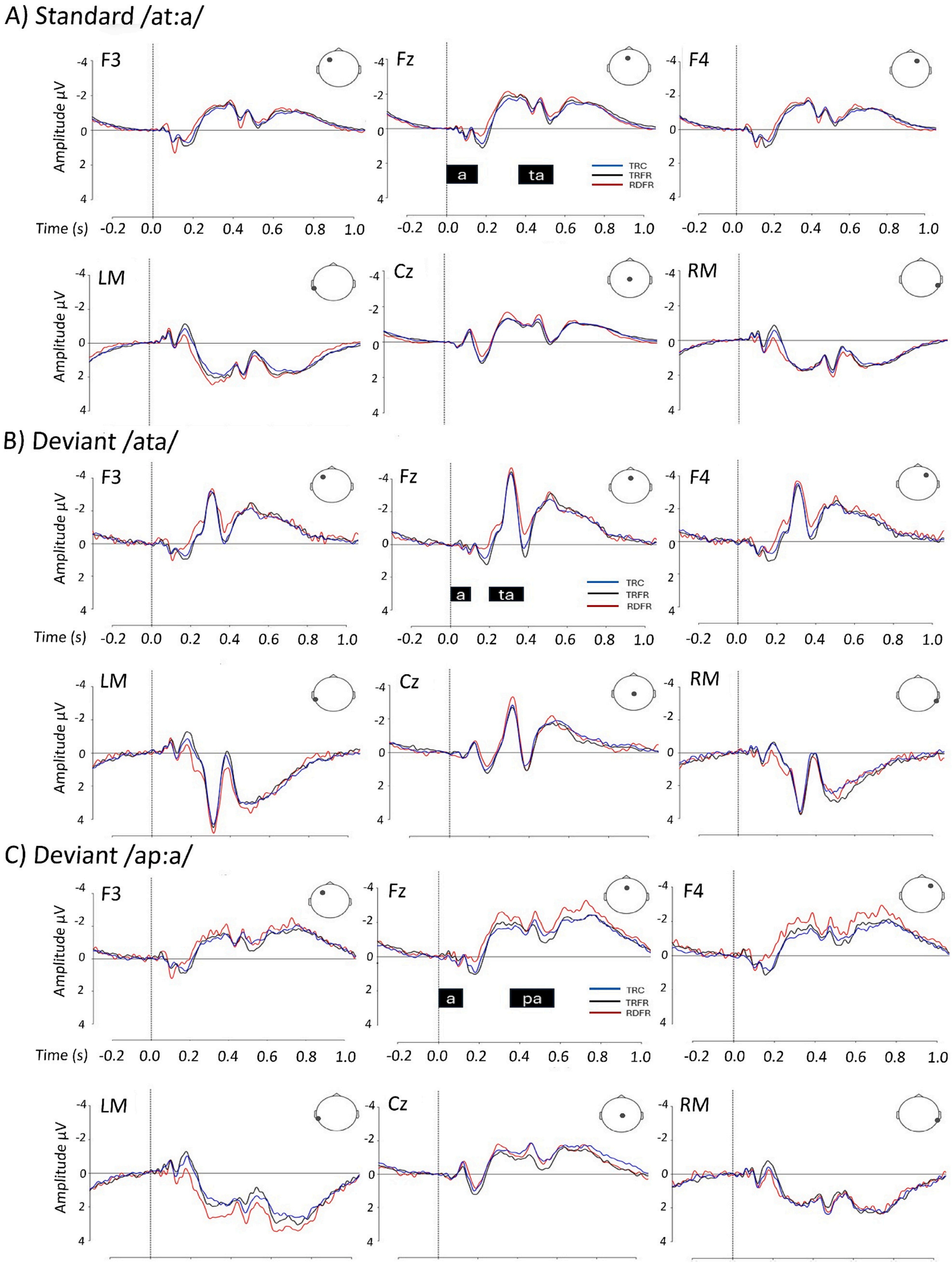

Fig. 7. ERP waveforms categorized by speech sound as follows: At the top (A) the ERP waveforms to standard sound (/at:a/), (B) deviant changing in phonemic length (/ata/), and (C) deviant changing in place of articulation (/ap:a/). The red curve represents the dyslexia group (RDFR), the black the risk group (TRFR), and the blue the controls (TRC). (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

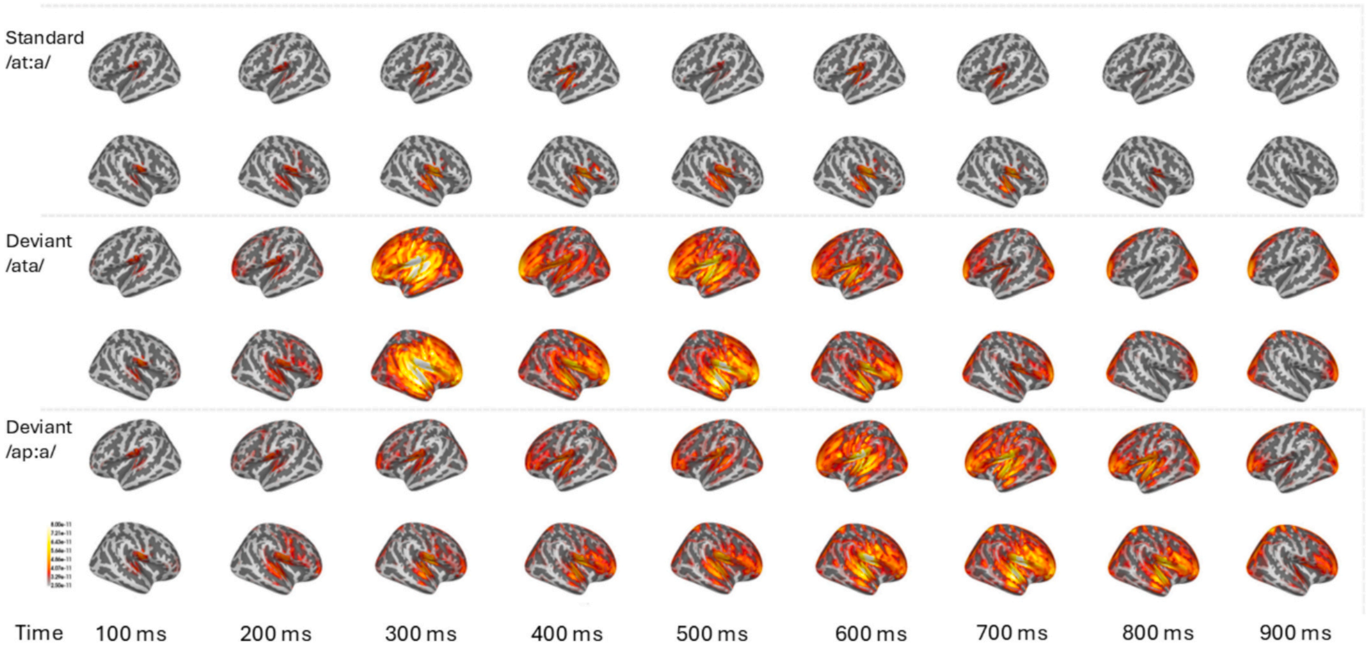

Fig. 8. Combined ERP and ERF source localization for the standard stimulus (/at:a/, upper row), the deviant differing in phonemic length (/ata/, middle row), and the deviant differing in place of articulation (/ap:a/, lower row), showing source-level activity at different time points.

enhanced ERP amplitudes to the deviant stimulus with a change in place of articulation (pseudoword /ap:a/). Whereas at 9.5 years-old children, partly including those tested as adults, the ERP amplitudes to the deviant stimulus with change in the phonemic length (pseudoword /ata/) were enhanced in the dyslexia group (Lohvansuu et al., 2014). In addition, the earlier study with the JLD participants at 6.5 years using the same speech stimuli found greater responses for the speech stimuli /ta/ when presented as the standard stimulus in children with dyslexia compared to the other groups (Hämäläinen et al., 2013). Furthermore, at 6 months of age, the group at familial risk for dyslexia and the control group differed in MMR responses to phonemic length changes, infants at risk for dyslexia showing diminished MMR responses to deviant stimuli (pseudowords long and short /ata/) when compared to controls (Leppänen et al., 2002). Although the direct longitudinal comparison is difficult due to attrition rate and some differences in the stimulation procedure, there seems to be a continuum of differences in individuals with dyslexia (or at risk for dyslexia) compared to the other groups.

In contrast, in other studies, an attenuation of MMN/N250 responses has been reported in children with dyslexia compared to controls regardless of the stimuli used (phonemic length, vowel identity, place of articulation) (Schulte-Körne et al., 1998; Lachmann et al., 2005; Meng et al., 2005; Chobert et al., 2012; Männel et al., 2017). In line with the present findings is the study by Thiede et al. (2020), in which they reported no group differences in MMF amplitudes to pseudowords varying in phonemic length (/ta-ta:/) between adults with and without dyslexia (Thiede et al., 2020). However, in adulthood, the ERPs to speech sounds varying in phonemic length and place of articulation in individuals with dyslexia seem to vary from study to study, some studies showing smaller, the other larger, or no differences when compared to typical readers (Schulte-Korne et al., 2001; Helenius et al., 2002; Kujala et al., 2006; Hommet et al., 2009; Thiede et al., 2020). Nevertheless, a deficit in the processing of speech appears to be present in individuals with dyslexia regardless of age (Volkmer and Schulte-Körne, 2018; Gu and Bi, 2020).

The JLD findings from current and previous studies present enhanced neural responses in individuals with dyslexia (Hämäläinen et al., 2013; Lohvansuu et al., 2014), which demonstrates a clear difference between typical readers and individuals with (childhood) dyslexia (and risk for dyslexia) in auditory processing of speech sounds across time. However, there is an interesting conflict in interpreting the results considering previous findings from other samples: enhanced responses to native speech sound deviancy are seen to reflect better speech processing abilities and better reading and spelling abilities (for review, see Volkmer and Schulte-Körne, 2018). This is partly in contrast to findings from dyslexia groups in the JLD study. Group differences in the partly overlapping sample at the age of 6.5 years were suggested to be maturational in origin, with the enhanced responses in individuals with dyslexia attributed to larger and less specific neuronal populations involved in stimuli encoding (Hämäläinen et al., 2013). Later, at the age of 9.5 years, enhanced responses to speech deviancy were interpreted to reflect a compensatory mechanism in individuals with dyslexia (Lohvansuu et al., 2014; for a review of fMRI results, see Richlan et al., 2011), when a greater number of neurons work together, exerting increased activity and more effort to properly encode the varying stimuli.

In adulthood, enhanced responses were found to deviant speech sounds varying in place of articulation in the dyslexia group, in contrast to childhood, when group differences emerged by the speech sounds varying in phonemic length (consonant duration). However, despite the differences in the size of the responses, it can be concluded, that auditory processing of speech is still affected in adults with childhood dyslexia, as it has also been shown that at 9 years, the JLD children with dyslexia performed worse in phoneme duration discrimination task (Pennala et al., 2010). This interpretation is in line with previous brain activity findings of the relative stability of the impairment of speech processing in individuals with dyslexia at different ages (Gu and Bi, 2020). These differences in responses to different speech sound features between childhood and adulthood could be explained by maturation and continued exposure to speech, which drives the need for further investigation.

4.2. *Mature brain responses to changes in speech not connected to behaviorally assessed reading and related skills*

The participants identified to have dyslexia at the age of 8 years still differed significantly in adulthood from other groups (without reading difficulties) in their reading fluency and rapid naming ability (see Table 1). However, despite the clear differences in reading and related skills, the brain responses no longer correlated with behaviorally

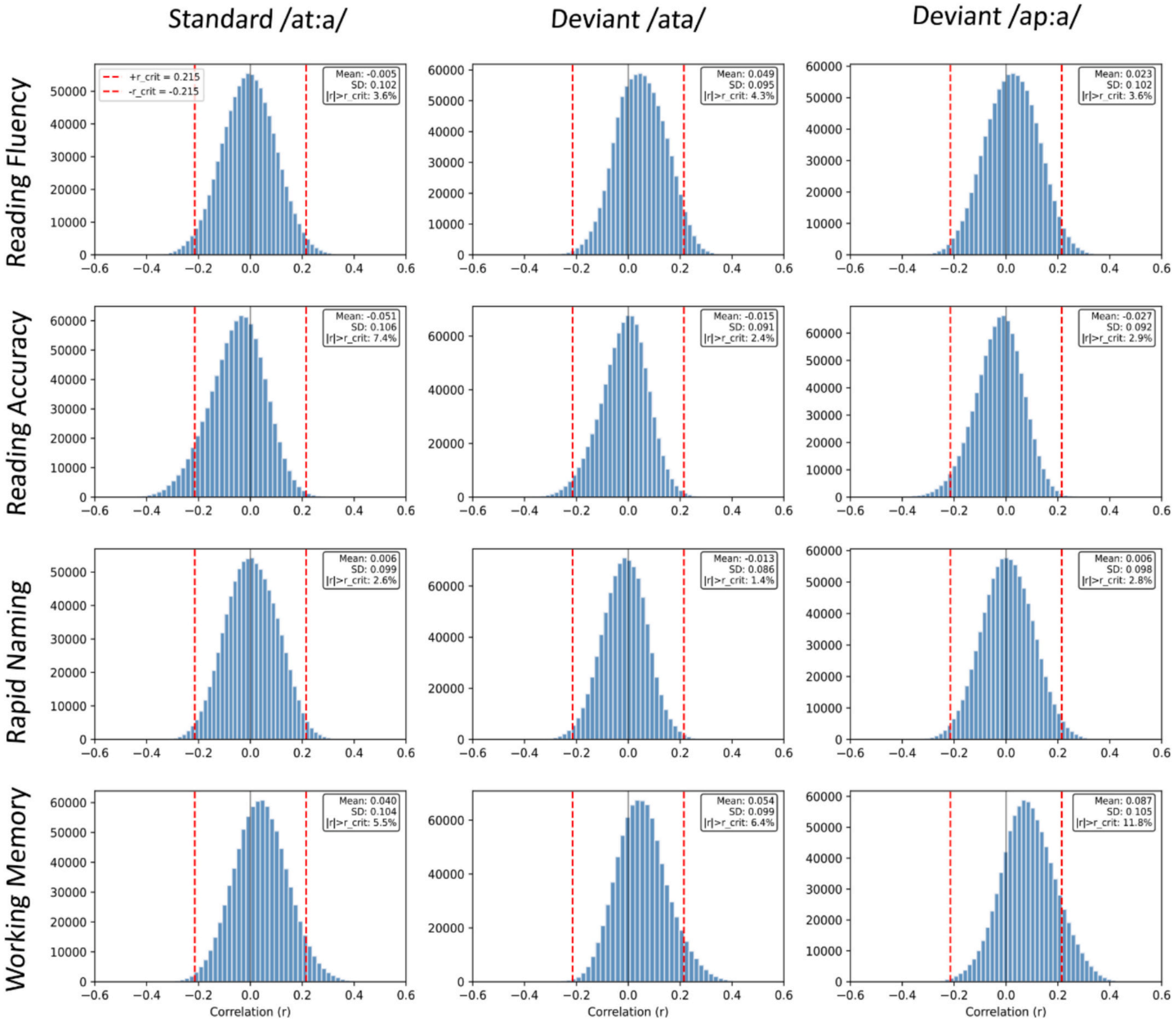

Fig. 9. Histograms of brain-behavior correlations for each data point. Distribution of Pearson correlation coefficients between source-level neural activity (50–900 ms time window) and four behavioral measures (rows: reading fluency, reading accuracy, RAN, working memory) across three stimulus conditions (columns: /at:a/, /ata/, /ap:a/). Red dashed lines indicate the critical r threshold (±0.215) for significance at α = 0.05 (two-tailed). Inset statistics show the mean correlation (Mean), standard deviation (SD), and percentage of correlations exceeding the critical threshold (|r| > r_crit). All distributions are centered near zero, consistent with the absence of significant brain-behavior associations in the cluster-based permutation tests. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

assessed reading skills. This could be because, on one hand, adults' reading skills are likely influenced by many other factors, such as the amount of reading exposure and learning strategies, which can either weaken or strengthen the reading ability and/or potential. Thus, though early differences in speech processing in childhood were linked to reading skills, speech processing may not play a similar role anymore in adulthood. On the other hand, continued exposure to speech could lead to changes in speech processing in adulthood. This possibly distinct development in reading and speech processing could obscure the association which was observed in children. Also, speech perception and reading experience could be more tightly linked at 9.5 years (Lohvansuu et al., 2014) because then the amount of reading exposure has been more limited compared to adults.

4.3. Limitations

This study has certain limitations that should be considered when interpreting the results. There have been minor changes in the experimental setup between different measurement points of the JLD sample. At the ages of 6 months and 6.5 years, the short /ata/ was used as a standard stimulus, and two longer versions of /at:a/ as deviant stimuli (Hämäläinen et al., 2013; Leppänen et al., 2002). At the age of 9.5 years and in adults the same experiment was used where long /at:a/ was the standard and the short /ata/ and long /ap:a/ were used as deviants (Lohvansuu et al., 2014). In addition, the sample across the different measurement points has different sample sizes due to missing data and splitting of the sample to different experiments in infancy (Pihko et al., 1999; Leppänen et al., 1999, 2002, 2010; Guttorm et al., 2001). However, the sample between 9.5 years and adults is largely overlapping (79 were measured with EEG at both ages). Despite the changes in the stimuli used, the responses to common stimuli across the measurement points show developmental changes in the pattern of group differences. Further, it should be noted that 9 group comparisons were conducted which inflates the Type 1 error rate. Determining the best *p*-value correction is not straight forward due to longitudinal priors and possible non-independent effects for all of the speech stimuli.

When compared to previous studies, the current study used different types of stimuli. Many studies in adults have utilized a place of articulation contrast at the beginning of the stimuli, whereas this study focused on changes in phonemic length and place of articulation in the middle of a pseudoword. This choice of stimuli is partly due to the Finnish language, where phonemic quantity is a semantically distinguishing feature. Embedding the deviant feature in the middle of the pseudoword might produce masking effects or make the processing in general more demanding, thereby leading to the difference between the current results and studies of other samples. Further, the use of stimuli with varying phonemic length made the computation of difference waves challenging. This is because obligatory ERP responses depend on the physical timing of the syllables in the stimulus, meaning that the resulting difference waves would have reflected these timing differences rather than pure change detection processes. However, no statistically significant differences were observed between groups for the response to the standard stimulus and therefore it is likely that the observed group differences for the deviant stimulus response were stimulus specific.

4.4. Conclusion

Taken together, the results of the present study, along with those conducted on infants, children, and adults, suggest that the development of the central nervous system influences the manifestation of auditory information and speech processing difficulties in children who later develop dyslexia. These developmental changes, together with increasing exposure to speech, may contribute to the brain responses of individuals with dyslexia gradually becoming more like those of control participants. To gain a deeper understanding of this development, further research is needed.

CRediT authorship contribution statement

Aino Sorsa: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. **Ariane Tretow:** Writing – review & editing, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Weiyong Xu:** Writing – review & editing, Visualization, Supervision, Methodology, Formal analysis, Data curation. **Georgia Gerike:** Writing – review & editing, Investigation, Conceptualization. **Kaisa Lohvansuu:** Writing – review & editing, Visualization, Methodology. **Jarmo A. Hämäläinen:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Project administration, Methodology, Funding acquisition, Formal analysis, Conceptualization. **Paavo H.T. Leppänen:** Writing – review & editing, Visualization, Resources, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization.

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Data availability

Due to the ongoing longitudinal investigation and limitations for data anonymization, the possibility to share data directly is not given. Generated data supporting the findings of this paper are available on request from the authors. Analysis scripts are available at the GitHub repository (https://github.com/weiyongxu/JLD26_ata_atta.git).

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