Neural Correlates of Statistical Learning in Developmental Dyslexia: An Electroencephalography Study

Short title: Statistical Learning in Developmental Dyslexia

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Conflict of Interest

26   The authors declare no competing financial interests.

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34 ## Abstract

35

36 The human brain extracts statistical regularities from the surrounding environment in a

37 process referred to as statistical learning. Recent behavioural evidence suggests that

38 developmental dyslexia affects statistical learning. However, surprisingly few

39 neurophysiological studies have assessed how developmental dyslexia affects the

40 neural processing underlying statistical learning. In this study, we used

41 electroencephalography to explore the neural correlates of an important aspect of

42 statistical learning – sensitivity to transitional probabilities – in individuals with

43 developmental dyslexia. Adults diagnosed with developmental dyslexia (n = 17) and

44 controls (n = 19) were exposed to a continuous stream of sound triplets in which a few

45 triplet endings were location deviants (i.e., were presented from an unexpected speaker

46 direction) or statistical deviants (i.e., had a low transitional probability given the

47 triplet's first two sounds). Location deviants elicited a large location mismatch

48 negativity (MMN), which was larger in controls than dyslexics. Statistical deviants

49 elicited a small, yet significant statistical MMN (sMMN) in controls, whereas the dyslexic

50 individuals did not exhibit a statistical MMN. These results suggest that the neural

51 mechanisms underlying statistical learning are impaired in developmental dyslexia.

52 **Significance statement**

53 We assessed the neural correlates of statistical learning in individuals with

54 developmental dyslexia. Statistical deviants, namely word endings with a low

55 transitional probability (as compared to high probability transitions) elicited a small,

56 yet significant statistical MMN in controls, whereas the dyslexic individuals did not

57 exhibit a statistical MMN. Location deviants elicited a MMN, which was larger in

58 controls than dyslexics. These results suggest that the neural mechanisms underlying

59 statistical learning are impaired in developmental dyslexia.

60   Introduction

61   The brain can identify statistical regularities in sequential information in a

62   process known as statistical learning (Saffran, Aslin, & Newport, 1996) or implicit

63   learning (Perruchet & Pacton, 2006). Statistical learning involves an implicit and innate

64   mechanism by which the brain calculates the transitional probability of sequential

65   information. This learning system is thought to play a crucial role in early language

66   acquisition. For example, 8-month-old infants (Saffran, Aslin, & Newport, 1996) and

67   neonates (Teinonen et al., 2009) can learn the probabilities of syllable transitions, thus

68   enabling them to detect word boundaries and isolate single words in natural speech.

69

70   Developmental dyslexia impairs reading comprehension and spelling in

71   language, and is thought to arise mainly from phonological perceptual difficulties

72   (Snowling, 2000; Ramus et al., 2003; Vellutino et al., 2004). In addition, individuals with

73   developmental dyslexia may further have a wide range of other nonlinguistic as well as

74   linguistic impairments, including weakened auditory statistical learning abilities

75   (Arciuli & Simpson, 2012; Du & Kelly, 2013; Evans, Saffran, & Robe-Torres, 2009;

76   Howard, Howard, Japikse, & Eden, 2006; Menghini, Hagberg, Caltagirone, Petrosini, &

77   Vicari, 2006; Vicari et al., 2005) and non-linguistic perceptual processing (Ahissar,

78   Protopapas, Reid, & Merzenich, 2000; Christmann, Lachmann, & Steinbrink, 2015;

79   Giraud & Ramus, 2013; McAnally & Stein, 1996; Sperling, Lu, Manis, & Seidenberg,

80   2005). Since phonological deficits can also be observed in people without

81   developmental dyslexia (Huettig et al., 2017), and not all children with developmental

82   dyslexia show phonological processing deficits (Lachmann & van Leeuwen, 2008;

83   Morris et al., 1998), phonological deficits may not automatically lead to developmental

84   dyslexia. Thus, at least in some individuals, developmental dyslexia may also have other

85   causes.

86

87   One possible cause is a more domain-general statistical learning deficit, not

88   specific to phonological processing. Particularly, a great deal of evidence has shown that

89   auditory statistical learning is impaired in dyslexic children, adolescents, and adults

90   (Gabay et al., 2015; Dobo et al., 2021; Kahta et al., 2019; Vandermosten et al., 2019),

91   although some other studies suggest that children with and without developmental

92   dyslexia had no difference in statistical learning performance (Witteloostuijn et al.,

93   2019). Despite much behavioral evidence on statistical learning deficits, the underlying

94   neural mechanisms of these deficits remain unclear. This study aimed to investigate the

95   neural correlates of statistical learning in individuals with developmental dyslexia using

96   electroencephalography (EEG), which exhibits a high sensitivity to capture statistical

97   learning even when behavioural measures may not indicate learning effects (Koelsch,

98   Busch, Jentschke, & Rohrmeier, 2016).

99

100   EEG can be used to measure neural activity during statistical learning.

101   Particularly, statistical learning is reflected in the electric brain responses to stimulation

102   (event-related potentials, ERPs). When the brain encodes the statistics of stimulus

103   sequences (e.g. regularities occurring within stimulus sequences), it predicts high-

104   probability stimuli, which is associated with an inhibited ERP response to the predicted

105   stimuli (compared with unpredicted, i.e., irregular or less regular, stimuli). Statistical

106   learning effects can thus manifest as differences in ERP response amplitudes for

107   expected and unexpected stimuli (for review, see Daikoku, 2018). Many studies have

108   suggested that statistical learning is reflected in both early ERP components such as

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109   auditory brainstem response (ABR; Skoe, et al., 2015), P50 (Daikoku et al., 2017;

110   Paraskevopoulos et al., 2012), N100 (Sanders et al., 2002), and mismatch negativity

111   (MMN; Koelsch et al., 2016; Moldwin, Schwartz, & Sussman, 2017) as well as later ERP

112   components such as the P200 (Balaguer et al., 2007; Cunillera et al., 2006) and theN400:

113   (François, et al., 2013). Typically, a mismatch negativity (MMN) is elicited in response to

114   physical deviants in oddball sequences. In such experiments, a series of standard stimuli

115   is interspersed with physical deviants (oddballs; e..g, sound differing in pitch or location

116   deviants; Christmann, Lachmann, & Berti, 2014; Garrido et al., 2008; Rinne, Antila, &

117   Winkler, 2001; Sussman, Winkler, & Schröger, 2003; Winkler & Czigler, 2012). For

118   example, if several sounds are presented from the right side, a sound presented on the

119   left side elicits an MMN, which is generated mainly in the auditory cortex (Garrido et al.,

120   2008).

121

122   Because developmental dyslexia is related to sensory processing dysfunctions,

123   including those of the auditory cortex (Clark et al., 2014; Goswami, 2014; for a review

124   see Gu & Bi, 2020), the MMN has been used to investigate the neural basis of

125   developmental dyslexia (Kujala et al., 2000). A reduced MMN amplitude in children

126   (Lachmann et al., 2005; for an overview see Bishop, 2007) and young adults (Schulte-

127   Körne et al., 2001) with developmental dyslexia reflected impaired performance in

128   syllable and tone discrimination and impaired tuning to native language speech

129   representations (Bruder et al., 2011). The MMN has therefore been suggested as a

130   neurophysiological endophenotype for developmental dyslexia (Neuhoff et al., 2012).

131   However, there is also one study suggesting that only certain aspects of auditory

132   processing may be affected: whereas the pitch MMN was shown to be impaired in that

133   study, was the location MMN enhanced (Kujala et al., 2006). Notably, such MMN effects

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134   to physical changes do not require statistical learning because the perceptual

135   regularities underlying the generation of the classical MMN can be extracted on a

136   moment-to-moment basis, such as a series of stimuli coming from the right side

137   interrupted by a stimulus from the left side. However, statistical learning can also be

138   reflected in the MMN (François, Cunillera, Garcia, Laine, & Rodriguez-Fornells, 2017;

139   Koelsch et al., 2016; Moldwin et al., 2017). Tsogli, Jentschke, Daikoku, and Koelsch

140   (2019) presented sequences of tone triplets that could contain location deviants

141   (comprising stimuli from an irregular location) and statistical deviants (comprising

142   triplet endings with a low probability given the two preceding triplet items; this low

143   transition probability could only be represented based on statistical learning, i.e., after

144   extensive exposure to many triplets). Both statistical and location deviants elicited

145   prominent mismatch ERP responses approximately 150–250 ms after stimulus onset.

146   ERP effects elicited by statistical deviants are termed statistical MMN (sMMN; Koelsch

147   et al., 2016) to distinguish them from the MMN to physical deviance, e.g., elicited by

148   location deviants (Paavilainen, Karlsson, Reinikainen, & Näätänen, 1989; Sams,

149   Paavilainen, Alho, & Näätänen, 1985). In contrast to the MMN eliecited by physical

150   deviance, the elicitation of the sMMN does not occur on a moment-to-moment basis.

151   Instead, it requires a more extended learning period to encode the underlying statistical

152   regularities and to store them in long-term memory. Thus, the sMMN is suitable to

153   investigate acquisition of knowledge regarding the statistical regularity of sound

154   sequences.

155

156   This study investigated how developmental dyslexia affects the classical MMN in

157   response to location deviants and the sMMN in response to statistical deviants in adults.

158   Assuming that dyslexia adversely affects the inferences of sensory statistics in the

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159   auditory cortex (Jaffe-Dax, Kimel, & Ahissar, 2018; Lieder et al., 2019), we hypothesised

160   that statistical and location deviants would elicit weaker MMNs in participants with

161   developmental dyslexia than the controls. Confirmation of this hypothesis would

162   provide evidence that developmental dyslexia is associated with both auditory

163   processing dysfunction and statistical learning of auditory sequences.

164

Materials and Methods

165

Participants

166

167   Twenty-one adults diagnosed with developmental dyslexia (11 females, mean

168   age = 26 years, SD = 5.3) and 20 age- and gender-matched control participants without a

169   diagnosis of dyslexia (14 females, mean age = 26 years, SD = 3.1) were screened for

170   eligibility to participate in this study. We excluded one individual from the

171   developmental dyslexia group and one from the control group because both performed

172   in the nonverbal intelligence test Standard Progressive Matrices (Raven & Court, 1998)

173   with an IQ score < 70 (two SD below the normal range). Three other individuals with

174   developmental dyslexia were excluded because they did not receive a clear diagnosis in

175   childhood. After these exclusions, our study sample included 17 adults with

176   developmental dyslexia and 19 control participants (Table 1), who all met the following

177   inclusion criteria: German as the native language, right-handedness (Edinburgh

178   Inventory; Oldfield, 1971), no history of neurological or audiological disorders, no

179   diagnosis of a general or specific language impairment, no mental retardation, and no

180   formal musical training for more than 5 years (beyond regular school lessons).

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182   The study protocol was approved by the Ethics Committee of the Max-Planck-

183   Institute (approval number: 2018/352). All participants were informed of the purpose

184   of the study and the procedures in place to ensure their safety and the confidentiality of

185   their personal data. They all provided written informed consent to participate in this

186   study.

187

188   *Evaluation of Spelling and Reading Skills*

189   Spelling skills were assessed with the Rechtschreibtest (Ibrahimović & Bulheller,

190   2013). In this test, participants were asked to fill in the missing words of a text, mainly

191   composed of irregular German words, that a skilled and German native speaking

192   experimenter read aloud. Silent text reading speeds and reading comprehension skills

193   were assessed with the Lesegeschwindigkeits-und Verständnistest für die Klassen 5–12

194   (LVGT 5–12; Schneider, Schlagmüller, & Ennemoser, 2017). In this test, participants

195   were asked to read as much of the text as possible within 4 min and to fill in each gap in

196   the text with one of the three possible options.

197

198   *Stimuli*

199   *Sounds*

200   We used the same stimuli and sequences as in a previous study (Tsogli et al.,

201   2019). Each sound consisted of a Shepard tone (Shepard, 1964), combined with the

202   sound produced by one of six different percussion instruments (i.e., a surdo, a

203   tambourine, agogô bells, a hi-hat, castanets, or a woodblock). We obtained the

204   percussive sounds from the Philharmonia Orchestra website

205   (http://www.philharmonia.co.uk/explore/sound_samples). We used six distinct

10

206   Shepard tones based on six frequencies (i.e., F_3 [174.61 Hz], G_3 [196.00 Hz], A_3 [220.00

207   Hz], B_3 [246.94 Hz], $C\#_4$ [277.18 Hz], and $D\#_4$ [311.13 Hz]), each tone resulting from the

208   superposition of nine sinusoidal components spaced an octave apart. The specific

209   combinations of Shepard tones and percussive sounds were counterbalanced across

210   participants. Examples of sounds are provided in Appendix A.

211

212   Another set of six sound combinations was created for a practice phase at the

213   start of each experiment. These sounds were similar to those used in the main

214   experiment but differed in terms of the frequencies providing bases for Shepard tones

215   (i.e., E_3 [164.81 Hz], $F\#_3$ [184.99 Hz], $G\#_3$ [207.65 Hz], $A\#_3$ [233.08 Hz], C_4 [261.62 Hz],

216   and D_4 [293.66 Hz]) and the percussive sounds used (i.e., the sounds of a woodblock, a

217   tambourine, agogô bells, castanets, a hi-hat, and a bass drum). An additional target

218   Shepard tone based on $C\#_5$ (554.37 Hz), that did not have an accompanying percussive

219   sound, was used for a cover task and a passive listening component of the experimental

220   procedure (see Experimental Procedure).

221   All sound stimuli had a tone duration of 220 ms, including rising and falling

222   periods of 10 ms and 20 ms, respectively, a constant loudness, and a sampling frequency

223   of 44,100 Hz with 16 bit resolution.

224

225   *Triplet Sequences*

226   The stimuli descibed above, hereafter referred to as sounds A to F, were

227   combined into sound triplets. Each 220 ms sound was followed by an 80 ms pause, such

228   that the total duration of each triplet was 900 ms. As shown in Figure 1, sounds A and B

229   and sounds C and D were paired to create two distinct two-sound sequences (i.e., AB

230   and CD) that served as the first two sounds of each triplet, which are hereafter referred

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231   to as the triplet roots. Sounds E and F were the sounds that could be used as the last

232   sound of a triplet, which is hereafter referred to as the triplet ending. Combining the

233   two roots and two triplet endings yielded four possible triplets (i.e., ABE, ABF, CDE, and

234   CDF). The assignment of different sounds to roles as triplet roots or endings was

235   counterbalanced across participants as a way of ensuring that any possible acoustical

236   differences between sounds would be cancelled out through particpants and not bias

237   the neural responses of interest.

238

239   **Exposition Sequences.** The sequence of sounds used during an exposition phase

240   comprised 400 sound triplets with a total sequence duration of about 6 min. The triplets

241   were presented in a pseudo-randomised order with no two sequentially adjacent

242   triplets being identical. Each of the two roots had an equal probability of occurring in a

243   given triplet regardless of the ending sound of the previous triplet. Each statistically

244   deviant triplet was followed by at least 3 triplets that were not statistically deviant.

245

246   Each sound stimulus was presented from either a speaker to the participant's

247   right or a speaker to the participant's left. These speakers were positioned at 60° angles

248   in the azimuthal plane. For each participant, one side was pseudo-randomly selected as

249   the standard side for stimuli to be presented from, and the other side was the deviant

250   one. The lateralization of the stimuli was balanced across blocks and counterbalanced

251   between participants, and whether the location was "standard" or "deviant" was

252   considered in the data analyses. For the triplet root sounds (i.e., sounds A, B, C, and D),

253   95% of the stimuli were presented from the standard side and the remaining 5% from

254   the deviant side. For the triplet endings (i.e., sounds E and F), 80% of the stimuli were

255   presented from the standard side and the remaining 20% from the deviant side. The

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256   triplets with endings presented from the deviant side were considered location

257   deviants.

258

259   To generate statistical deviants, we set distinct probabilities for a transition (i.e.,

260   transitional probability) from a given root to a given ending within a triplet. The

261   sensitivity to transitional probability is one of the important aspects of statistical

262   learning mechanisms (Perruchet & Pacton, 2006; Saffran, Aslin, & Newport, 1996). The

263   transitional probability for a given triplet ending was either 90% or 10% depending on

264   root identity (Figure 1a). Triplets containing low-probability root-to-ending transitions

265   were considered statistical deviants. Each statistically deviant triplet was followed by

266   ≥3 triplets that were not statistically deviant. The location and statistical deviance

267   created four triplet categories (Table 2). That is, standard triplets, which accounted for

268   72% of all triplets, were neither location deviant nor statistically deviant. The remaining

269   28% of triplets could be statistically deviant only (8% of all triplets), location deviant

270   only (18% of all triplets), or both statistically deviant and location deviant (2% of all

271   triplets).

272

273   **Behavioural Testing Sequences.** Each behavioural testing phase of the

274   experiment consisted of twelve trials, with each trial involving a pair of triplets

275   separated by a 335-ms pause. The triplets in each pair had the same root but different

276   endings with low vs. high transition probabilities, respectively (i.e., statistically deviant

277   vs. not deviant). The order in which the statistically deviant and standard triplets were

278   played was counterbalanced across trials. Varying the order in which the same-root

279   triplets within a pair were played created four sequentially distinct pairings (i.e., ABE to

280   ABF, ABF to ABE, CDE to CDF, and CDF to CDE). Each sequentially distinct pairing was

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281   played three times during each behavioural testing phase, and consecutively played

282   pairings had alternating triplet roots (i.e., each AB pairing was followed by a CD pairing

283   that was in turn followed by an AB pairing). During behavioural testing phases, all

284   triplets were played from both speakers, i.e., the task focussed on statistical regularity

285   whereas location was not a factor.

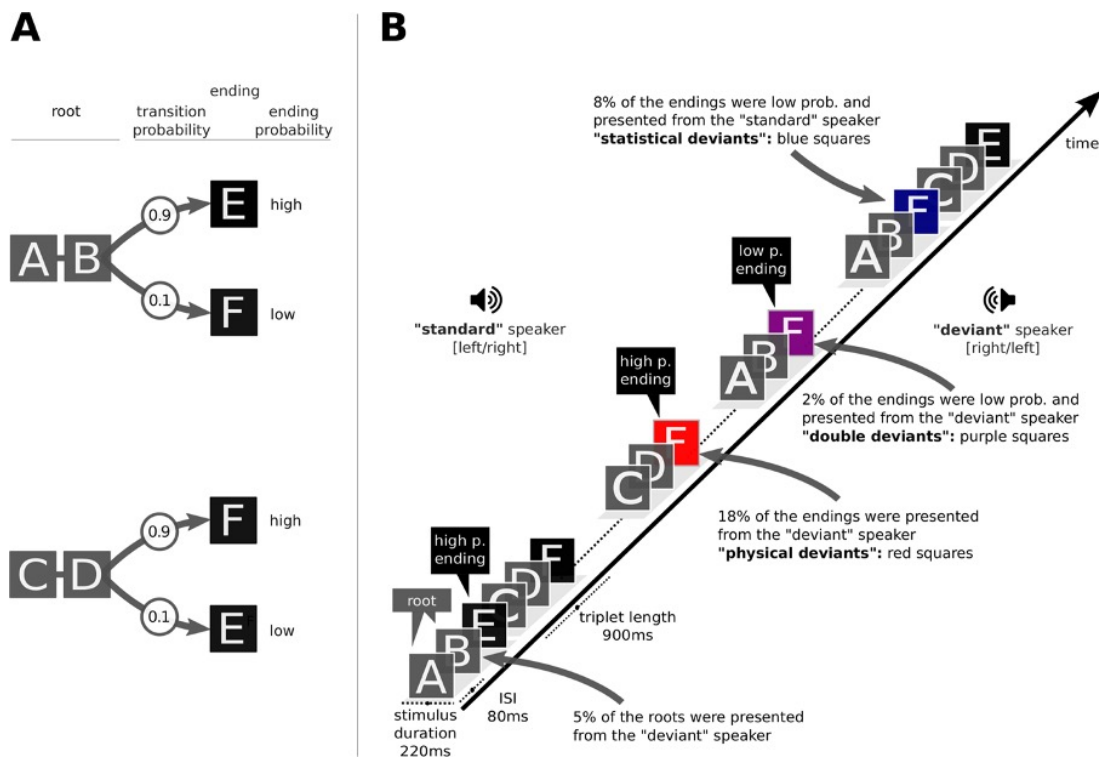**Figure 1**. **Triplets and stream in this study.** (**A**) Four types of triplets generated from six different sounds (designated with letters from A to F) produced by pairing a Shepard tone with various percussive sounds. Each triplet consists of a root containing two conserved sounds (AB or CD) and a triplet ending with a high (90%) or a low (10%) transitional probability given the triplet's root. Each triplet root has a 50% probability of occurring regardless of the ending sound of the previous triplet. (**B**) Example of a possible triplet sequence including standard triplets (triplet endings in black boxes), triplets with a statistically deviant ending (triplet endings in blue boxes) or a location deviant ending (triplet ending in red box), and a triplet with a doubly deviant ending (i.e., statistically deviant and location deviant; triplet ending in purple box). Reprinted, with permission, from Tsogli et al. (2019). Abbreviations: ISI, inter-stimulus interval; p. & prob., probability.

309 ***Experimental Design***

310   The participants completed a multi-stage experiment with six blocks while

311 undergoing EEG monitoring (see 'Collection and Analysis of EEG Data') inside an

312 electromagnetically shielded chamber. Immediately before the experiment, the

313 participants were provided with instructions concerning the procedures for the

314 experiment's different phases (see below). To ensure that only implicit learning could

315 occur, the instructions did not include any description of the possible location or

316 statistical deviance of the triplets. The participants then completed a 1-min practice

317 session in which they were asked to press a key as soon as possible after hearing a

318 target sound (i.e., C♯5 [554.37 Hz]). If necessary, each participant repeated the practice

319 session until they had correctly pressed the key after 80% of the target sound

320 presentations.

321

322   Each of the six blocks included an exposition phase comprising the passive

323 listening of the sequence of 400 triplets and a subsequent behavioural testing phase in

324 which the participants performed actions based on a triplet sequence. During the

325 exposition phase, participants were instructed to react to the high-pitched tones (cover

326 tasks, 0.67% of all tones) while they were exposed to the sequence of 400 triplets

327 described above (see Exposition Sequences). At the same time, they were watching a

328 silent movie (nature or wildlife documentaries) played on a monitor in front of them.

329 While the cover task was not particularly demanding (e.g., in terms of attentional

330 resources) it minimized the possibility of the participants intentionally focusing their

331 attention on statistical properties.

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333   During a behavioural testing phase, the participants listened to paired

334   statistically deviant and standard triplets (see Behavioural Testing Sequences). After

335   each pair of triplets, a participant was asked to choose which triplet in the pair sounded

336   more familiar and to rate their confidence in a given answer on a scale ranging from 1

337   (no certainty) to 5 (certainty).

338

Acquisition and Analysis of EEG Data

340   We obtained 64-channel EEG data (Brain Amp, Brain Products, Munich,

341   Germany) through cap-mounted electrodes placed over the participants' scalps in

342   accordance with the extended international 10-20 system. The left mastoid electrode

343   was used as reference electrode and the neck electrode was used as ground electrode.

344   The electrodes were clustered into six regions of interest: a frontal left region (F7, F5,

345   F3, FT7, FC5, and FC3), a frontal middle region (F1, FZ, F2, FC1, FCZ, and FC2), a frontal

346   right region (F8, F6, F4, FT8, FC6, and FC4), a central left region (T7, C5, C3, TP7, CP5,

347   and CP3), a central middle region (C1, Cz, C2, and CPZ), and a central right region (T8,

348   C6, C4, TP8, CP6, and CP4). Horizontal and vertical electro-oculograms were recorded

349   bipolarly through electrodes placed at the outer canthi of the eyes and above and below

350   the right eye. Electrode impedance was kept < 5 kΩ. Signals were recorded with a 0.25–

351   1,000-Hz bandpass filter and a 500-Hz sampling rate.

352

353   EEG data were analysed in EEGLAB 13 (Delorme & Makeig, 2004) in MATLAB

354   R2018b (The MathWorks, Natick, Massachusetts). Continuous raw data files were re-

355   referenced to the algebraic mean of the left and right mastoid electrodes and filtered

356   with a 0.5-Hz high-pass filter and a 30-Hz low-pass filter implemented with finite

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357   impulse response designs and Blackman windows of 550 points and 2,750 points,

358   respectively. Channels with excessive noise were identified through visual inspection

359   and interpolated when necessary. The mean number of interpolated channels per

360   participant was 0.22. Independent component analysis was used for linear

361   decomposition of continuous data to remove the contributions of artefacts affecting

362   scalp sensors (e.g. slow drifts, eye blinks or movement, and muscle artefacts). Epochs

363   were removed from further analyses if the amplitude changes exceeded ±45 µV in any

364   channels, including the electro-oculograms (less than 10% of the trials). The epochs of

365   target stimuli (cover tasks) were removed in the analysis. In the end, 97.2% (SD±2.6%)

366   and 94.8% (SD±8.0%) was preserved in the dyslexic and control groups, respectively.

367   We also performed student's t-test between groups. There were no significant

368   differences (t(37), 0.69, p=0.50). Selective response averaging was conducted

369   separately for standard triplets, triplets with a location deviant (but not with a

370   statistical deviant), triplets with a statistical deviant (but not a location deviant), and

371   triplets with both a location deviant and a statistical deviant (see 'Exposition

372   Sequences').

373

374   Averages were computed using a 100 ms pre-stimulus baseline. We directly

375   addressed the hypothesis of the present study, focusing on the average MMN

376   amplitudes measured within a 150–250 ms time window. In addition, we analysed an

377   obvious positive component approximately 100–140 ms after the onset of the stimuli,

378   henceforth referred to as P120. That is, we also investigated the average amplitudes

379   within a 100–140 ms time window.

380

18

Statistical Analysis

Between-Group Comparisons of Participant Characteristics

Statistical analyses were conducted using jamovi version 1.2 (The jamovi Project, 2020). We used Bonferroni-corrected *t*-tests (dividing 0.05 by the number of tests) when comparing demographic characteristics and scores on tests of intelligence, spelling skills, and reading abilities between the dyslexia and the control group.

Analyses of Exposition Phase Data

We used separate ANOVA models to analyse the effects of stimulus deviance on P120 and MMN effect amplitudes during the exposition phases. The sMMN response is had a smaller amplitude size compared with the location MMN. Further, the sMMN is a relatively new ERP component, for which the neural mechanism has not yet been elucidated in detail. To determine those neural generators, studies using fMRI or MEG would be most suited, however, such studies still are lacking despite many studies using EEG methodology. Thus, this study directly compared between standard and deviant but did not use the subtraction waveform that is typically used in MMN studies. One ANOVA model featured the within-participant factor of sound location (i.e., location standard triplet endings or location deviant triplet endings), and the other featured the within-participant factor of transitional probability (i.e., statistically standard triplet endings or statistically deviant triplet endings). Both ANOVA models included the between-participants factor of groups (i.e., the developmental dyslexia vs. the control group) and three within-participant factors: the distinction between ERP responses in the frontal vs. central areas of the brain; the distinction between ERP responses in the left, medial, and right areas of the brain (i.e., ERP response lateralisation); and the different experimental blocks. The posterior region was not included because of low

406   amplitude sizes. To boost our signal-to-noise ratio, our ANOVA models included three

407   blocks rather than the actual six by merging the data from pairs of blocks.

408

409   We selected $p < 0.05$ as our threshold for statistical significance and used a false

410   discovery rate method for the post-hoc testing of significant effects. To determine

411   whether the Rechtschreibtest and LVGT 5–12 scores correlated with the P120 and MMN

412   effect amplitudes, we calculated Pearson correlation coefficients.

413

414   *Analyses of Behavioural Testing Phase Data*

415   We used a two-tailed *t*-test to determine whether the frequency of correct answers

416   during behavioural testing exceeded chance levels (i.e., >.5). We also used ANOVA

417   models to compare the dyslexia and control groups in terms of response accuracies and

418   reaction times (RT), and included experimental blocks as a within-subject factor in

419   these analyses. As in our analyses of exposition phase data, we assumed three blocks

420   rather than the actual six in order to boost our signal-to-noise ratios. We selected $p <.05$

421   as our threshold for statistical significance and used Bonferroni correction for the post-

422   hoc testing of significant effects. We conducted Pearson correlation analysis to

423   determine whether response accuracy rates correlated with confidence ratings and

424   dyslexia test scores.

425

426   **Results**

427   ***Participant Characteristics***

428   Relative to the control group, the developmental dyslexia group had lower

429   average scores for spelling abilities, reading comprehension, and reading speed (Table

20

430   1). However, the two groups were comparable in terms of age and intelligence (age:

431    $t(34) = 1.41$, $p = .167$; IQ: $t(34) = .50$, $p = .620$).

432

EEG Results

P120 and MMN Responses to Location Deviance

435   First, we examined whether developmental dyslexia affects ERP responses to location

436   deviants (only standards and deviants presented on high-probability triplet endings

437   were included in this analysis). Location deviants elicited a P120 followed by a location

438   MMN, which was maximal at anterior frontal electrodes (Figure 2a–b). The location

439   MMN appeared in both the dyslexia and control groups, but had a smaller amplitude in

440   the dyslexia group. Compared with the standards, the location deviants elicited larger

441   P120 responses in the control group, whereas the location deviants did not elicit larger

442   P120 responses in the dyslexia group (see Appendix B for mean P120 and location MMN

443   amplitudes in each condition). These observations were reflected in an ANOVA,

444   indicating a significant interaction between sound location (standard, deviant) and

445   group for the P120: $F(1, 34) = 5.52$, $p = .025$, η^2p $= .14$; and the location MMN: $F(1, 34) =$

446   5.16, $p = .03$, η^2p $= .13$; see the Appendix C for complete results. When analysing ERP

447   responses separately in each group, location deviants elicited a significant location

448   MMN in both the dyslexia group and the control group ($p <.001$; Figure 2c). Compared

449   with the standards, location deviants elicited larger P120 responses in the control group

450   ($p = .011$), but there was no significant difference in the dyslexia group ($p = .49$). Higher

451   spelling scores on the Rechtschreibtest correlated with larger location deviance–

452   induced P120 effects ($r = .37$, $p = .03$; see the Appendix C). No other correlations

453   between language aptitude test scores and EEG responses were observed.

21

454

P120 and MMN Responses to Statistical Deviance

456   Next, we examined whether developmental dyslexia affects ERP responses to

457   statistical deviants (only triplet endings without location change were included in this

458   analysis). Statistical deviants elicited a P120, followed by a sMMN, which was maximal

459   over anterior frontal electrodes (Figure 3a–b; see the Appendix B for mean amplitudes

460   of the P120 and sMMN components in each condition). Statistical deviants elicited a

461   P120 in both groups, and an sMMN in the control group but not in the dyslexia group.

462   These observations were reflected in an ANOVA, indicating a significant interaction

463   between transitional probability (high vs. low probability), frontal vs. central, and

464   Group: $F(1, 34) = 5.50$, $p = .025$, $\eta^2p = .14$ (for complete results, see the Appendix D).

465   Post-hoc tests revealed that the sMMN response at frontal electrodes was significant in

466   the control group ($p = .032$) but not in the dyslexia group ($p = .74$; Figure 3c). At the

467   central electrodes, the sMMN was not significant in both groups.

468

469   The effects of statistical learning on P120 amplitudes were prominent at medial

470   electrodes (Figure 3b–c), as reflected by the significant interaction between transitional

471   probability and lateralisation ($F[2, 68] = 4.15$, $p = .020$, $\eta^2p = .11$). Post-hoc tests

472   revealed that the P120 responses to statistical deviants were larger than those to the

473   standards at medial electrodes ($p = .009$); however, no such effects were apparent in the

474   left ($p = .23$) or right region ($p = .53$).

475

476   The effects of statistical learning on P120 amplitudes gradually increased as the

477   experiment progressed to later blocks, as reflected by a significant interaction between

478   transitional probability, lateralisation, and experimental blocks ($F[4, 136] = 2.78$, $p =$

22

479   .029, η^2p = .08). Post-hoc tests revealed that the amplitudes of P120 responses to

480   statistical deviants were larger in the third experimental block than in the first (p =

481   .002) and second blocks (p = .004). Furthermore, in the analysis of ERP responses over

482   the central region, the P120 response amplitudes elicited by statistical deviants were

483   significantly different from those elicited by standards during the third block (p <.001);

484   however, this was neither observed for the first (p = .15) nor the second block (p = .85).

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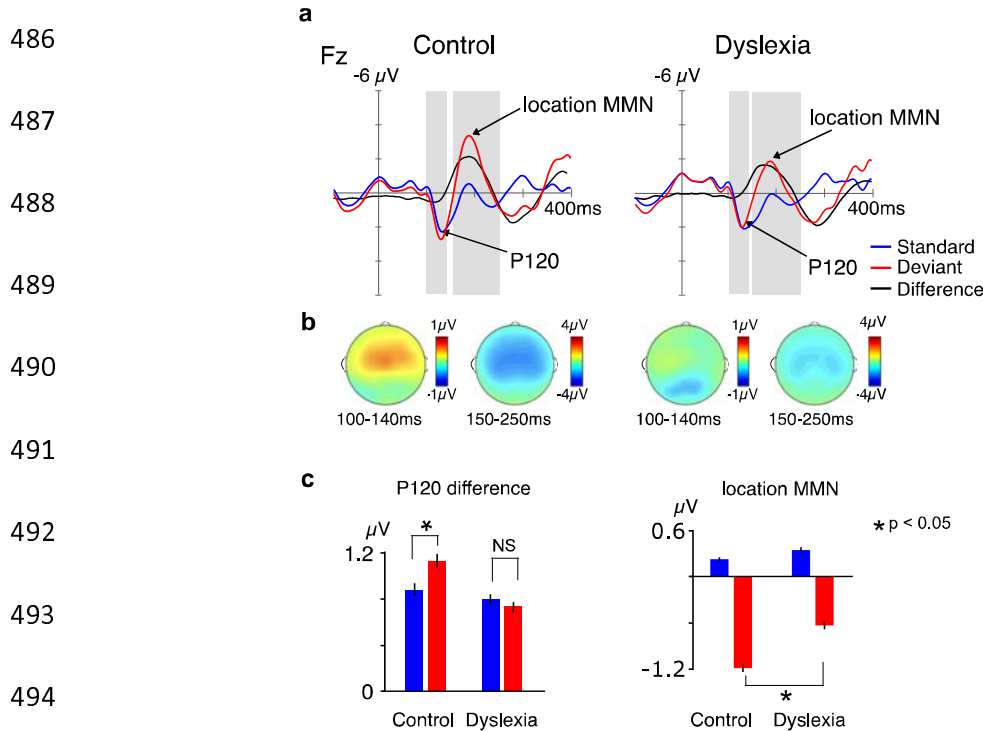
Figure 2. **Location MMN results**. (**a**) Mean ERP responses to triplet endings as recorded from the FZ electrode. Grey areas indicate the time windows used for quantifying the P120 (~100–140 ms) and location MMN (150–250 ms) components. Averaged ERP responses to location standard (blue) and deviant (red) triplet endings, as well as differences between them (black), are shown separately. (**b**) Isopotential maps showing the scalp distributions of differences between the ERPs evoked by location deviant triplet endings and those evoked by location standard triplet endings in the control (left) and dyslexia (right) groups. (**c**) Interactive effects of dyslexia and sound location at an anterior frontal region (average of F7, F5, F3, FT7, FC5, FC3, F1, FZ, F2, FC1, FCZ, FC2, F8, F6, F4, FT8, FC6, and FC4). Location deviant triplet endings elicited larger P120 amplitudes than location standard triplet endings in the control group, but this was not the case in the dyslexia group. Significant location MMN effects were observed in both groups but were larger in the control group than in the dyslexia

24

508   group. Error bars indicate the standard deviation of the mean. Abbreviations: ERP,

509   event-related potential; location MMN, location mismatch negativity.

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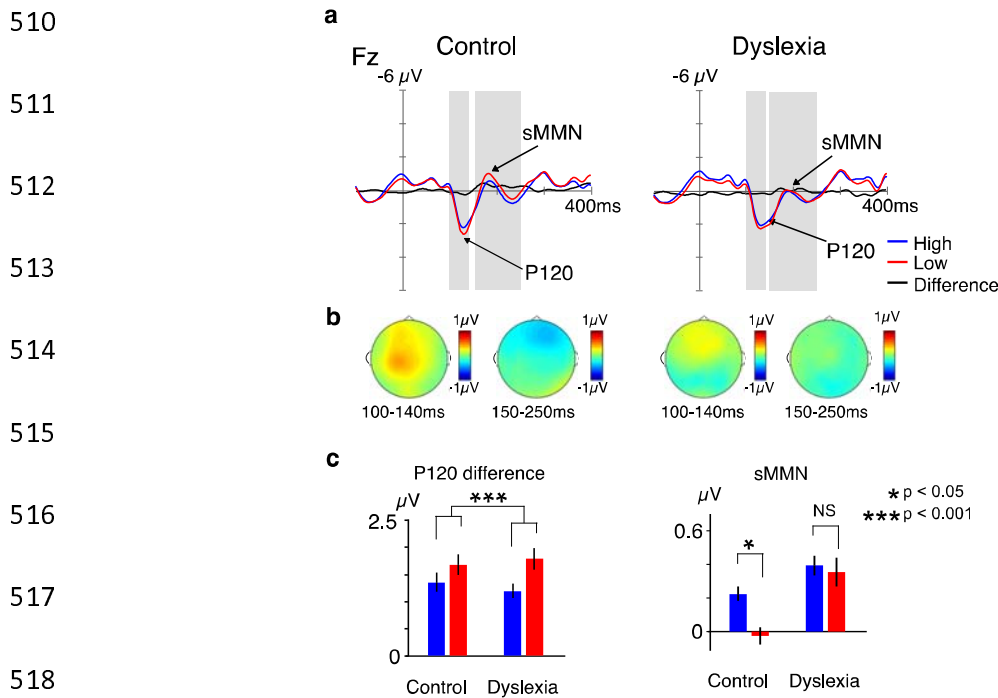
519   **Figure 3. Statistical MMN results.** (**a**) Mean ERP responses to triplet endings as

520   recorded from the Fz electrode. The grey areas indicate the time windows used for

521   quantifying the P120 (~100–140 ms) and sMMN (150–250 ms) component. Averaged

522   ERP responses to statistically standard (blue) and deviant (red) triplet endings, as well

523   as differences between them (black), are shown separately. (**b**) Isopotential maps

524   showing the scalp distributions of differences between ERPs evoked by statistically

525   deviant triplet endings and those evoked by statistically standard triplet endings. (**c**)

526   Interactive effects of dyslexia and transitional probability at an anterior frontal region

527   (average of F7, F5, F3, FT7, FC5, FC3, F1, FZ, F2, FC1, FCZ, FC2, F8, F6, F4, FT8, FC6, and

528   FC4). In the medial electrodes, statistically deviant triplet endings elicited larger P120

529   amplitudes than the statistically standard triplet endings in both groups. Significant

530   sMMN effects were observed in anterior frontal brain areas in the control group, but no

25

531   sMMN effects were observed in the dyslexia group. Error bars indicate standard

532   deviation of the mean. Abbreviations: ERP, event-related potential; sMMN, statistical

533   mismatch negativity.

Behavioural Results

534

535   During the exposition phase, the participants discovered on average 94.3% (*SD* =

536 .02) of the acoustical deviants (i.e., they showed a high performance in the cover task

537 where participants had to detect high pitched tones). This indicates that the

538 participants paid attention to the acoustical stimuli, while the task was relatively simple

539 to carry out.

540

541   At the end of the exposition phase in each block, the participants listened to

542 paired statistically deviant and standard triplets. A participant was asked to choose

543 which triplet in the pair sounded more familiar and to rate their confidence in a given

544 answer on a scale ranging from 1 (no certainty) to 5 (certainty). To evaluate the

545 performance in the behavioural testing phase, we used a two-tailed *t*-test to determine

546 whether the frequency of correct answers during behavioural testing exceeded chance

547 levels (i.e., $p > .05$). Further, we used ANOVAs to compare response accuracies, RT, and

548 confidence ratings between dyslexics and controls. ANOVAs were computed with Group

549 (dyslexia vs. control) as a between-subjects factor and experiment block as a within-

550 subject factor (three blocks; the first, second and third blocks, instead of the actual six

551 blocks of the experiment to obtain a higher signal-to-noise ratio).

552

553   The two-tailed *t*-test revealed the frequency of correct answers was significantly

554 higher than chance levels in the control group ($p = .017$, *Cohen's* d = .325) but not the

555 dyslexic group ($p = .520$). ANOVAs revealed no effects on response accuracies and

556 confidential rating in both the dyslexia and control groups (Figure 4, and see Appendix

557 E). As for the reaction time, a significant main effect of the experimental block was

558 noted ($F[2, 68] = 7.92$, $p < .001$, $\eta^2p = .189$). Post-hoc tests revealed that the reaction

27

559   time in the second and last blocks was significantly faster than that in the first block

560   (2nd: p = .006; 3rd: p <.001). No other effect was found in the ANOVA of behavioural

561   results.

562

563   To determine whether response accuracy rates correlated with confidence

564   ratings, we also conducted a Pearson correlation analysis. Response accuracies did not

565   correlate with confidence ratings in both the control (r = −0.18, p = .19) and dyslexia (r

566   = −0.05, p = .70) groups. Similarly, response accuracies did not correlate with reading

567   and spelling test scores.
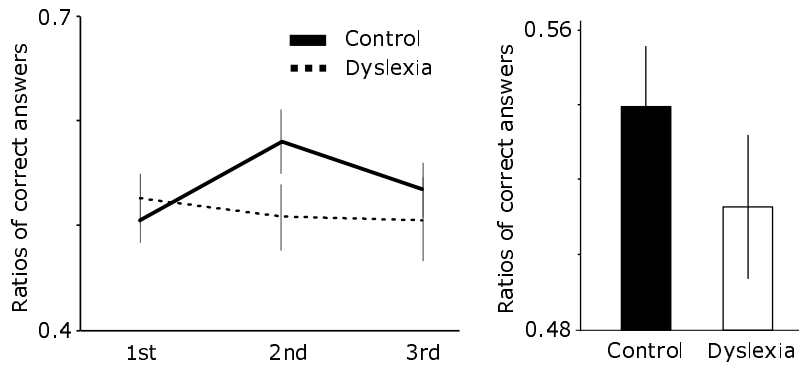
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Correctness ratios

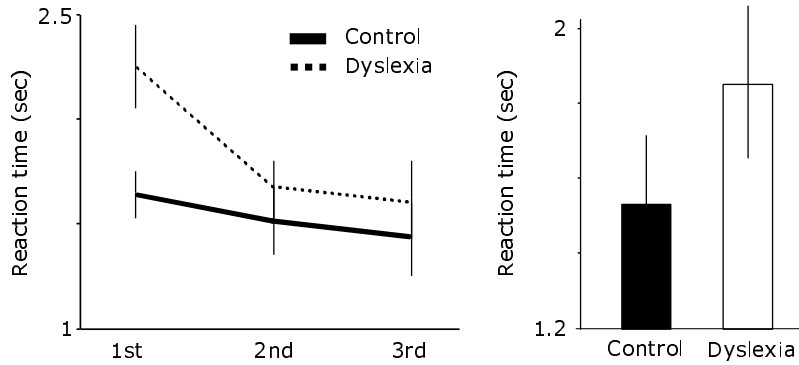## Reaction time

578 **Figure 4. Behavioural testing results across experimental blocks.** Error bars

579 indicate standard deviations. The ANOVA revealed no significance difference in

580 correctness ratios and reaction time between dyslexia and control groups.

581 **Discussion**

582   In this study, we assessed the relationship between developmental dyslexia and

583   ERP responses to a continuous stream of sound triplets in which some triplet endings

584   were either location deviants or statistical deviants. We found that statistical deviants

585   elicited small sMMN and that location deviants elicited a large location MMN. Compared

586   with controls, the participants with developmental dyslexia exhibited a smaller location

587   MMN and no sMMN. Whereas there was a tendency to a higher proportion of of correct

588   responses and decreased reaction times in the control compared to the dyslexia

589   group,there was no significant between-group difference for those two variables,

590   reflecting the ability to learn the statistical characteristics of stimuli. Our findings

591   indicate that the neural functions underlying the sMMN and the location MMN are

592   impaired in individuals with developmental dyslexia. This is in agreement with previous

593   evidence of a smaller MMN in developmental dyslexics (Gu & Bi, 2020; Lachmann et al.,

594   2005; Neuhoff et al., 2012). Our findings also suggest that at the group level, the MMN

595   may be more sensitive to mild difficulties than behavioural measures.

596

597   The location MMN is a response reflecting auditory perceptual memory

598   operations that are instantly updated after new information is obtained (Bendixen,

599   Prinz, Horváth, Trujillo-Barreto, & Schröger, 2008; Sussman & Winkler, 2001). The

600   sMMN, on the other hand, relies on memory representations that are formed from the

601   implicit knowledge of sequential statistical structure (i.e., knowledge of stimulus

602   transitional probabilities; cf., e.g., Koelsch et al., 2016; Tsogli et al., 2019). Notably, the

603   sMMN effects elicited in our experiment were maximal at anterior frontal electrodes

604   (Figure 3b); however, the location MMN effect was more broadly distributed from

605   central to frontal areas (Figure 2b). This suggests the possibility that neural sources of

606   the sMMN are different from those of the location MMN. However, our findings showed

607   that people with dyslexia also exhibited smaller location MMN.

608   A past study found a diminished pitch MMN but an enhanced location MMN in

609   dyslexia (Kujala et al., 2006). We assumed that the combination of location and

610   statistical MMN in our paradigm may lead to different results from Kujala's study.

611   Another possibility is that the acoustic perceptual dysfunction of dyslexic

612   individuals also influenced neural processing of a sMMN. For example, past studies have

613   showed faster decay of dyslexics' perceptual memory trace behaviorally (Jaffe-Dax et al.,

614   2017; Lieder et al., 2019) and in brain activity (Jaffe-Dax 2015, 2017), specifically in the

615   auditory cortex (Gertsovski & Ahissar, 2022 Perrachione et al., 2016). They proposed

616   that it reduces learning of complex rules (e.g. Virtala et al., 2021) and particularly

617   hampers acquisition of categories (Gertsovaki & Ahissar, 2022).

618

619   Both location MMN and sMMN were preceded by P120 responses. Compared

620   with the controls, the participants with developmental dyslexia exhibited no difference

621   in P120 responses between location deviants and standards. Previous studies have

622   reported the atypicalities of the P1 component in sound processing and dyslexia

623   (Stefanics et al., 2011) and implicit and statistical learning (Jaffe-Dax et al., 2017). The

624   P120 found in our study may be a P1-like component in terms of the positive

625   component temporally adjacent to the N1 or MMN effects. Further, given the

626   fundamental differences in the memory system between ERPs elicited by statistically

627   deviant stimuli and location deviants as stated above, the P120 components elicited by

628   statistically deviant stimuli may also be distinguishable from those of the P120

629   components elicited by location deviants. However, considering the difference in the

31

630   time window and that this effect was not hypothesised, it will not be discussed further

631   until future studies replicate this effect. Further studies are therefore necessary to

632   elucidate the neural basis underlying P120 components.

633

634   It has recently been considered that a domain-general statistical learning

635   impairment, rather than a specific impairment in phonological processing (Ramus et al.,

636   2003; Vellutino et al., 2004), may underlie developmental dyslexia. For example,

637   individuals with developmental dyslexia show weaker domain-general statistical

638   learning across sensory domains (Hung, Frost, & Pugh, 2018), such as auditory (Arciuli

639   & Conway, 2018; Dobó et al., 2021; Gabay et al., 2015; Kahta & Schiff, 2019;

640   Vandermosten et al., 2019) and visual stimuli (Sigurdardottir et al., 2017). Furthermore,

641   statistical learning impairment in developmental dyslexia is not limited to speech

642   stimuli but also occurs in non-speech stimuli (Plakas, van Zuijen, van Leeuwen,

643   Thomson, & van der Leij, 2013). Our results support these past findings; our study

644   explored effects of deviation from predictions based on physical and statistical

645   properties using non-speech auditory stimuli, and our findings showed that statistical

646   learning processes and basic auditory processes are affected in individuals with

647   developmental dyslexia.

648

649   Some new theoretical frameworks have been proposed to explain statistical

650   learning impairment in dyslexia. For example, a multicomponent memory network,

651   referred to as the Statistical Learning and Reading (SLR) model (Lee et al., 2022),

652   represents a domain-general system that consists of a short-term memory subsystem,

653   an explicit declarative long-term memory subsystem, and an implicit procedural long-

654   term memory subsystem. The two long-term memory subsystems are distinguishable in

32

655   terms of the attentional demands required for encoding and storage of information,

656   with more controlled attention (i.e., a top-down selective attention to learning stimuli)

657   than automatic attention (i.e., a bottom-up involuntary attention to salient stimuli)

658   needed in the explicit declarative subsystem, and, conversely, more automatic attention

659   than controlled attention needed in the implicit procedural subsystem. The model

660   output is a statistically optimal representation as manifested by the neural and

661   behavioural response of statistical learning and reading activities.

662

663   In this study, participants conducted the behavioural testing at the end of each

664   block. In this experimental paradigm, it would be possible that they tended to pay more

665   attention to the tone sequence in the later blocks since they anticipated they may

666   experience a test later on. However, it is also important to see the time course of

667   statistical learning effects. Indeed, this study detected that participants did not show

668   statistical learning effects in the first block and a difference between groups. However,

669   the difference gradually became larger particularly in the second block whereafter it

670   stayed at about the same level (Figure 4, top). Further, the control group but not the

671   dyslexic group showed an above-chance level in behavioral performance of statistical

672   learning. However, no significant group difference may suggest the possibility that the

673   two groups learned the sequence equally. Further research is necessary to reveal why

674   and how the MMN is more sensitive to group difference than behavioural measures.

675

676   In conclusion, our findings lay forth evidence that location deviants elicit a

677   distinct classical MMN, which was larger in controls than in individuals with

678   developmental dyslexia. Statistical deviants elicited a sMMN in controls, whereas in

679   individuals with developmental dyslexia, sMMN was not recognizable. Our findings add

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680   to and are consistent with the so far scarce evidence showing that statistical learning

681   and the underlying neural correlates may be impaired in individuals with

682   developmental dyslexia. Thus, exploring those neural correlates may contribute to a

683   better understanding of the cognitive processes underlying the acquisition of the rules

684   and regularities that guide the arrangement of elements in ordered sequences such as

685   language and music.

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1005 **Tables**

1006 **Table 1. Demographic and diagnostic data (raw scores) in the control and**

1007 **dyslexia groups.**

	Control	Dyslexia	*t*(34)	*p*	*Cohen's d*
N (females)	19 (13)	17 (12)			
Age	25.6 (±3.3)	23.8 (±4.2)	1.41	.167	.472
Intelligence	28.0 (±3.7)	27.0 (±3.9)	0.79	.436	.263
Spelling	70.8 (±4.1)	46.5 (±9.6)	9.71	<.001	3.373
Text reading					
Reading Comprehension	48.5 (±10.6)	32.5 (±8.9)	4.91	<.001	1.637
Reading Speed (number of read words)	1190.7 (±232.8)	803.94 (±202.4)	5.29	<.001	1.766

1008 Note. Averages are reported as mean ± standard deviation. The intelligence scores and
1009 spelling and reading test scores are raw scores.
1010

1011

49

1012 **Table 2. The 2x2 types of triplet endings based on location and statistical**

1013 **constraints.**

		Transition probability	
		High (90%)	Low (10%)
Sound	Standard (80%)	Standards (72%)	Statistical Deviant (8%)
Location	Deviant (20%)	Location Deviant (18%)	Double Deviant (2%)

1014

1015

1016 **Extended data**

1017

1018 **Appendix B**. **Mean ERP amplitudes under different conditions**. ERP, event-related

1019 potential.

1020

1021 **Appendix C. ANOVA results for the effects of location deviance**. (a) Correlation

1022 analysis and ANOVA results for P120 component amplitudes and (b) ANOVA results for

1023 location MMN effects. Location MMN, location mismatch negativity.

1024

1025 **Appendix D. ANOVA results for the effects of statistical deviance**. (a) ANOVA results

1026 for P120 component amplitudes and (b) sMMN effects. sMMN, statistical mismatch

1027 negativity.

1028

1029 **Appendix E. ANOVA results for data from a behavioural test assessing the**

1030 **statistical learning of stimulus characteristics**.