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Commercial Interest:

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**Visual motion and decision-making in dyslexia: Reduced accumulation of sensory evidence and related neural dynamics**

**Abbreviated title: Visual motion and decision-making in dyslexia**

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**43 Abstract**

44 Children with and without dyslexia differ in their behavioural responses to visual information,  
45 particularly when required to pool dynamic signals over space and time. Importantly, multiple  
46 processes contribute to behavioural responses. Here we investigated which processing  
47 stages are affected in children with dyslexia when performing visual motion processing  
48 tasks, by combining two methods that are sensitive to the dynamic processes leading to  
49 responses. We used a diffusion model which decomposes response time and accuracy into  
50 distinct cognitive constructs, and high-density EEG. 50 children with dyslexia (24 male) and  
51 50 typically developing children (28 male) aged 6 to 14 years judged the direction of motion  
52 as quickly and accurately as possible in two global motion tasks (motion coherence and  
53 direction integration), which varied in their requirements for noise exclusion. Following our  
54 pre-registered analyses, we fitted hierarchical Bayesian diffusion models to the data, blinded  
55 to group membership. Unblinding revealed reduced evidence accumulation in children with  
56 dyslexia compared to typical children for both tasks. Additionally, we identified a response-  
57 locked EEG component which was maximal over centro-parietal electrodes which indicated  
58 a neural correlate of reduced drift-rate in dyslexia in the motion coherence task, thereby  
59 linking brain and behaviour. We suggest that children with dyslexia tend to be slower to  
60 extract sensory evidence from global motion displays, regardless of whether noise exclusion  
61 is required, thus furthering our understanding of atypical perceptual decision-making  
62 processes in dyslexia.

63

**64 Significance statement**

65 Reduced sensitivity to visual information has been reported in dyslexia, with a lively debate  
66 about whether these differences causally contribute to reading difficulties. In this large pre-  
67 registered study with a blind modelling approach, we combine state-of-the art methods in  
68 both computational modelling and EEG analysis to pinpoint the stages of processing that are  
69 atypical in children with dyslexia in two visual motion tasks that vary in their requirement for  
70 noise exclusion. We find reduced evidence accumulation in children with dyslexia across  
71 both tasks, and identify a neural marker, allowing us to link brain and behaviour. We show  
72 that children with dyslexia exhibit general difficulties with extracting sensory evidence from  
73 global motion displays, not just in tasks that require noise exclusion.

74

## 75 **Introduction**

76           It has long been suspected that visual processing relates to the reading difficulties  
77 characterising developmental dyslexia (e.g., Hinshelwood, 1896; Lovegrove et al., 1980).  
78 One visual function that develops atypically in those with dyslexia is visual motion  
79 processing: an important ability contributing to scene segmentation, depth perception and  
80 object recognition (Braddick et al., 2003). Difficulties in global motion tasks requiring  
81 integration over space and time have been widely reported in dyslexia (Benassi et al., 2010).  
82 Typically, participants are required to detect or discriminate coherently moving signal dots  
83 amongst randomly moving noise dots (Newsome & Paré, 1988). In this ‘motion coherence’  
84 task, dyslexic individuals tend to have elevated psychophysical thresholds, requiring higher  
85 proportions of signal dots to perform at the same level of accuracy as those without dyslexia  
86 (Benassi et al. 2010). The nature of the relationship is still being debated, with some  
87 researchers proposing a causal relationship between motion sensitivity and reading ability  
88 (Boets et al., 2011; Gori et al., 2016; but see Goswami, 2015; Joo et al., 2017; Olulade et al.,  
89 2013; Piotrowska & Willis, 2019).

90           Atypical global motion processing in dyslexia may reflect reduced sensitivity to rapid  
91 temporal information originating from deficiencies in the magnocellular system (Livingstone  
92 et al., 1991; Stein, 2001, 2019; Stein & Walsh, 1997) or related dorsal stream (Braddick et  
93 al., 2003; Hansen et al., 2001), which are particularly specialised for motion perception  
94 (Livingstone & Hubel, 1988). Alternative accounts suggest that dyslexic individuals have  
95 difficulty filtering out the randomly moving noise dots in motion coherence tasks (“noise  
96 exclusion”; Conlon et al., 2012; Sperling et al., 2006) or difficulties integrating over space  
97 and time (Benassi et al., 2010; Hill & Raymond, 2002; Raymond & Sorensen, 1998).

98           Despite focusing on the sensory parameters of visual motion stimuli, these accounts  
99 give little consideration to the dynamic processes leading to atypical behavioural responses  
100 in dyslexia, and particularly, whether decision-making processes are affected. Here we  
101 explicitly modelled the decision-making process using a popular cognitive model of accuracy  
102 and response time: the diffusion model (Evans & Wagenmakers, 2020; Ratcliff, 1978; Stone,

103 1960). The decision is modelled as a noisy evidence accumulation process from a starting  
104 point towards one of two decision bounds (Figure 1). This modelling approach will help  
105 identify the locus of atypical processing in dyslexia, with two further advantages. First, the  
106 resulting parameters may be more sensitive to group differences than accuracy or response  
107 time alone (Stafford et al., 2020) and second, the parameters relate well to neural measures  
108 (Kelly & O'Connell, 2013; Manning et al., 2021a; Turner et al., 2015). Accordingly, we  
109 combined the diffusion model with a neural measure sensitive to the dynamic processes  
110 contributing to behavioural responses (EEG), bridging brain and behaviour.

111

112 *[insert Figure 1 about here]*

113

114 The diffusion model was recently applied to motion coherence performance in  
115 children with varying reading abilities (O'Brien and Yeatman, 2020). Poorer reading was  
116 related to lower drift-rates, wider decision bounds, and more intra-individual variability in  
117 starting point and non-decision time. Therefore poor readers accumulated motion evidence  
118 more slowly and responded more cautiously than good readers.

119 Here, we used diffusion models to identify the processing stages affected in children  
120 with dyslexia across two global motion tasks. The first task was a standard motion  
121 coherence task (cf. O'Brien & Yeatman, 2020). The second task was a direction integration  
122 task not used before with dyslexic individuals, whereby dot directions are sampled from a  
123 Gaussian distribution, with difficulty manipulated via the standard deviation of the  
124 distribution. In this task, the optimal strategy is to average over all dots, with no noise  
125 exclusion requirement. The reason for presenting both tasks to children with dyslexia was to  
126 determine whether differences in model parameters are found for both motion tasks,  
127 suggesting a general motion-processing deficit (cf. magnocellular/dorsal deficit; Braddick et  
128 al., 2003; Stein, 2001), or whether differences in model parameters are found particularly for  
129 the motion coherence task, reflecting noise exclusion difficulties (Conlon et al., 2012;  
130 Sperling et al., 2006).

## 131 **Methods**

132

### 133 *Pre-registration*

134 We pre-registered our inclusion criteria and analysis plan before completing data  
135 collection and before commencing analyses (<https://osf.io/enkwm>). When analysing the data  
136 we used a blind modelling approach to ensure that modelling decisions were not biased by  
137 our hypotheses. Our pre-registered primary research questions and hypotheses were:

138 *1. Do children with dyslexia have reduced drift-rates in a motion coherence task*  
139 *compared to typically developing children?* We hypothesised that children with dyslexia  
140 would have reduced drift-rates in the motion coherence task compared to typically  
141 developing children, in line with the results of O'Brien and Yeatman (2020) and reports of  
142 reduced motion coherence sensitivity in dyslexic individuals (Benassi et al., 2010).

143 *2. Do children with dyslexia have reduced drift-rates in a direction integration task*  
144 *compared to typically developing children?* If children with dyslexia show difficulties with all  
145 global motion tasks (in line with impaired magnocellular/dorsal stream functioning; Braddick  
146 et al., 2003; Stein, 2001), then we would expect children with dyslexia to have a reduced  
147 drift-rate in this task as well. Instead, if the performance of children with dyslexia in a motion  
148 coherence task is limited solely by difficulties with noise exclusion (Conlon et al., 2012;  
149 Sperling et al., 2006), we would expect to see no difference between children with and  
150 without dyslexia in this task, as it does not require segregating signal dots from randomly  
151 moving noise dots.

152 *3. Do children with dyslexia show increased boundary separation?* We hypothesised  
153 that children with dyslexia would have wider boundary separation compared to typically  
154 developing children in both tasks, following O'Brien and Yeatman (2020).

155 *4. Do children with dyslexia show increased non-decision time?* We hypothesised no  
156 group differences in overall non-decision time in either task, following O'Brien and Yeatman  
157 (2020).

158

## 159 *Participants*

160           We collected data from 50 children with dyslexia and 60 typically developing children  
161 who met our inclusion criteria. Specifically, participants were required to be aged 6 to 14  
162 years (inclusive), have verbal and/or performance IQ scores above 70 (measured using the  
163 Wechsler Abbreviated Scales of Intelligence, 2<sup>nd</sup> edition [WASI-2]; Wechsler, 2011) and to  
164 have normal or corrected-to-normal acuity, as measured using a Snellen acuity chart (with  
165 binocular acuities of 6/9 or better for children aged 6 to 8 years and 6/6 or better for children  
166 aged 9 to 14 years). Children in the dyslexia group were required to have a dyslexia  
167 diagnosis (or be in the process of obtaining one,  $n = 1$ ), and to have a reading and spelling  
168 composite score of 89 or below, which was computed by averaging the standard scores for  
169 the spelling subtest of the Wechsler Individual Achievement Test (WIAT-III; Wechsler, 2017)  
170 and the Phonological Decoding Efficiency subtest of the Test of Word Reading Efficiency  
171 (TOWRE-2; Torgesen et al., 2012). A cut-off of 89 was chosen to correspond to 1.5 standard  
172 deviations below the mean of typically developing children in a similar study (Snowling et al.,  
173 2019a, 2019b). Children in the typically developing group were required to have composite  
174 scores above 89 and to have no diagnosed developmental conditions. Datasets from an  
175 additional 4 typically developing children were excluded due to poor visual acuity ( $n = 1$ ),  
176 having a composite score of 89 or below ( $n = 2$ ), or failing to pass criterion on the task ( $n =$   
177 1), and datasets from an additional 11 children with dyslexia were excluded due to poor  
178 visual acuity ( $n = 2$ ) or having a composite score above 89 ( $n = 9$ ).

179           We then selected 50 typically developing children to best match the children with  
180 dyslexia in terms of age and performance IQ using the R MatchIt package (Ho et al., 2011),  
181 so that the final dataset included 50 children with dyslexia (24 male) and 50 typically  
182 developing children (28 male). As shown in Table 1, the children with dyslexia had slightly  
183 higher ages and lower IQ values on average than the typically developing children. EEG  
184 data were collected during task performance in 47 typically developing and 44 children with  
185 dyslexia (although EEG data were available only in the motion coherence task for one child  
186 with dyslexia). The EEG data from these participants were included in a paper investigating

187 responses locked to the onset of coherent motion in typically developing children and  
188 children with autism or dyslexia (Toffoli et al., 2021), and the larger group of 60 typically  
189 developing children were used to form the comparison group in an autism study (Manning et  
190 al., 2021b).

191 *[insert Table 1 about here]*

192

### 193 *Apparatus*

194 The tasks were presented on a Dell Precision M3800 laptop (2048 x 1152 pixels, 60  
195 Hz) using the Psychophysics Toolbox for MATLAB (Brainard, 1997; Kleiner, Brainard & Pelli,  
196 2007; Pelli, 1997). EEG signals were collected using 128-channel Hydrocel Geodesic  
197 Sensor Nets connected to Net Amps 300 (Electrical Geodesics Inc., OR, USA) and  
198 NetStation 4.5 software. A photodiode attached to the monitor independently verified  
199 stimulus presentation timing. Participants used a Cedrus RB-540 response box (Cedrus, CA,  
200 USA).

201

202 *[insert Figure 2 about here]*

203

### 204 *Stimuli*

205 Stimuli were 100 white, randomly positioned dots (diameter  $0.19^\circ$ ) moving at  $6^\circ/s$   
206 within a square aperture ( $10^\circ \times 10^\circ$ ) on a black background, with a limited lifetime of 400 ms.  
207 Each trial had a fixation period, a random motion period, a stimulus period, and an offset  
208 period, with a red fixation square ( $0.24^\circ \times 0.24^\circ$ ) presented throughout (see Figure 2). By  
209 presenting random (incoherent) motion before the stimulus period, we could dissociate  
210 evoked responses to directional motion from pattern- and motion-onset evoked potentials.  
211 The start of the stimulus period was highlighted to participants with an auditory tone. In the  
212 motion coherence task, directional motion (leftward or rightward) was introduced in a  
213 proportion of 'signal' dots, while the remainder of the dots continued to move in random  
214 directions. In the direction integration task, the directions of dots in the stimulus phase were

215 distributed according to a Gaussian distribution with a mean leftward or rightward direction.  
216 The fixation period, random motion period and offset period had jittered durations within a  
217 fixed range, while the stimulus period was presented until a response or 2500 ms had  
218 elapsed. The offset period continued the directional motion to temporally separate motion  
219 offset from the response.

220

### 221 *Experimental task procedure*

222 Children completed motion coherence and direction integration tasks within child-  
223 friendly games (based on Manning et al., 2019, 2021a). Using animations, participants were  
224 told that fireflies were escaping from their viewing boxes, and they were asked to tell the  
225 zookeeper which way the fireflies were escaping. There were 10 'levels' of the game. Levels  
226 1-5 corresponded to one task (either motion coherence or direction integration), and Levels  
227 6-10 corresponded to the other task, with the order of tasks being counterbalanced across  
228 participants. Levels 1 and 6 were practice phases, and the remaining 4 levels for each task  
229 were experimental blocks. In the motion coherence task, difficulty was manipulated by  
230 varying the proportion of coherently moving dots, and in the direction integration task,  
231 difficulty was manipulated by varying the standard deviation of the Gaussian distribution from  
232 which the dot directions were sampled.

233 In the practice phases, four demonstration trials were presented with no random  
234 motion phase and an unlimited stimulus phase, so that the experimenter could explain the  
235 task. Participants reported stimulus direction using a response box. The first two  
236 demonstration trials were 'easy' (100% coherence or 1° standard deviation), and the last two  
237 were more difficult (75% and 50% coherence, or 10° and 25° standard deviations). Following  
238 the demonstration trials, there were up to 20 criterion trials with a coherence of 95% or a  
239 standard deviation of 5°. These trials introduced the random motion phase. Participants were  
240 told that the fireflies would be going "all over the place" at first, and that they must wait for an  
241 alarm (auditory beep) before deciding which way the fireflies were escaping. A time limit was  
242 enforced, with visual feedback presented on the screen if participants did not respond within

2500 ms (“Timeout! Try to be quicker next time!”). Feedback on accuracy was given for responses made within the time limit (“That was correct!”, or “It was the other way that time”). When participants met a criterion of four consecutive correct responses, no more criterion trials were presented. Next, there were eight practice trials of increasing difficulty (motion coherence task: 80%, 70%, 60%, 50%, 40%, 30%, 20%, 10%; direction integration task: 5°, 10°, 15°, 20°, 30°, 40°, 50°, 60°) with feedback as before. Level 1 was repeated for one typically developing child and 2 children with dyslexia who did not meet the criterion of four consecutive correct responses on the first attempt, but passed on the second attempt.

Levels 2-5 and 7-10 each contained 38 trials, with 9 repetitions of each of two difficulty levels (motion coherence task: 30%, 75%; direction integration task: 70°, 30° SD), for each motion direction (leftward, rightward), and an additional 2 catch trials presenting 100% coherent (0° SD) motion. The experimental phase for each task therefore consisted of 152 trials. No trial-by-trial feedback was presented during the experimental phase, apart from a ‘timeout’ message if no response was made within 2500ms after stimulus onset. At the end of each level, participants were given points for their speed and accuracy in the preceding block (computed by  $(1 / \text{median response time}) * \text{the number of correct responses} * 2$ , rounded to the nearest integer). If participants obtained a score under 10, a score of 10 points was given to maintain motivation. Trials were presented automatically, although the experimenter could pause and resume trial presentation if necessary. The experimental code can be found here: <https://osf.io/fkjt6/>.

263

#### 264 *General procedure*

The procedure was approved by the Central University Research Ethics Committee at the University of Oxford. Parents provided written informed consent and children gave verbal or written assent. All children took part at the University of Oxford apart from one child with dyslexia who was seen at school without EEG. During the experimental tasks, participants sat 80cm away from the computer screen in a dimly lit room. For children who participated with EEG, we fitted the net prior to the experiment and ensured that electrode

271 impedances were below 50 k $\Omega$ . EEG data were acquired at a sampling rate of 500Hz with a  
272 vertex reference electrode.

273 Children were closely monitored by an experimenter sitting beside them. The  
274 experimenter provided general encouragement and task reminders, pausing before the start  
275 of a trial if needed (e.g., to remind the child to keep still). Children had short breaks at the  
276 end of each 'level' and a longer break at the end of the first task (at the end of 'level 5').  
277 During the longer break, electrode impedances were re-assessed for children wearing EEG  
278 nets. Children marked their progress through the levels using a stamper on a record card.  
279 The children also completed a Snellen acuity test, the WASI-2, the TOWRE-2 and the  
280 spelling subtest of the WIAT-III. The whole session took no longer than 2 hours and children  
281 were given a gift voucher to thank them for their time.

282

### 283 *Diffusion model analysis*

284 Initially, a blinded analysis was conducted to ensure that modelling decisions were  
285 made without being biased by the hypotheses under test. The first author (CM) prepared a  
286 blinded dataset in which group membership was randomly permuted (see also Dutilh et al.,  
287 2017) and one of the authors (NJE) ran diffusion model analysis on this blinded dataset.

288 Prior to modelling, trials with response times under 200 ms were removed  
289 (corresponding to 0.20% of trials in the typical group and 0.24% of trials in the dyslexia  
290 group). Trials without a response (i.e., no response made within the 2500ms deadline) were  
291 modelled as non-terminating accumulation trajectories, with the probability of a non-  
292 response occurring being the survivor function for the model at the time of the 2500 ms  
293 deadline (Evans et al., 2018; Howard et al., 2020; Ulrich & Miller, 1994). These trials  
294 accounted for 1.02% of the data in the typical group and 1.26% of the data in the dyslexia  
295 group. We fit the data from each task with hierarchical, Bayesian diffusion models with 5  
296 parameters: 1) average drift-rate across difficulty levels *v.mean*, 2) boundary separation *a*, 3)  
297 non-decision time *ter*, 4) difference in mean drift-rate between difficulty levels *v.diff*, and 5)  
298 starting point *z*. The stochastic noise within the model (*s*) was fixed at 0.1 to solve a scaling

299 problem within the model, as per convention (Ratcliff, 1978). There were 3 hyperparameters  
 300 for each parameter reflecting the mean ( $\mu$ ) and standard deviation ( $\sigma$ ) across the two groups  
 301 and the difference between groups ( $\delta$ ). Importantly, this parameterization allowed us to  
 302 explicitly set priors on the differences between groups, which was the key effect of interest  
 303 within the current study. More specifically, the priors were:

304 *Data level:*

$$y_{pi} \sim \text{diffusion}(a_p, z_p, Ter_p, v_{pi}, s)$$

305 *Parameters:*

$$a_p \sim N_+(\mu_a \pm \delta_a, \sigma_a)$$

$$z_p/a_p \sim TN_{0,1}(\mu_z \pm \delta_z, \sigma_z)$$

$$Ter_p \sim N_+(\mu_{Ter} \pm \delta_{Ter}, \sigma_{Ter})$$

$$v_{p1} - v_{p2} \sim N(\mu_{v.diff} \pm \delta_{v.diff}, \sigma_{v.diff})$$

$$\frac{v_{p1} + v_{p2}}{2} \sim N(\mu_{v.mean} \pm \delta_{v.mean}, \sigma_{v.mean})$$

$$s = 0.1$$

306 *Hyperparameters:*

$$\mu_a \sim N_+(0.2, 0.2)$$

$$\mu_z \sim TN_{0,1}(0.5, 0.2)$$

$$\mu_{Ter} \sim N_+(0.3, 0.3)$$

$$\mu_{v.diff} \sim N(0, 0.1)$$

$$\mu_{v.mean} \sim N(0.3, 0.3)$$

$$\sigma_a, \sigma_z, \sigma_{Ter}, \sigma_{v.diff}, \sigma_{v.mean} \sim \Gamma(1, 1)$$

$$\delta_a, \delta_z, \delta_{Ter}, \delta_{v.diff}, \delta_{v.mean} \sim N(0, 0.01)$$

307

308 where  $y$  reflects the data, and subscripts  $p$  and  $i$  reflect the participant and difficulty  
 309 level respectively. The priors for the  $\mu$  and  $\sigma$  parameters were based on those used in  
 310 previous studies implementing hierarchical diffusion models (e.g., Evans & Brown, 2017;

311 Evans & Hawkins, 2019; Evans et al., 2019), and the priors for the  $\delta$  parameters were based  
312 on the “moderately informative priors” used for the differences between conditions in Evans  
313 (2019). We used a differential evolution Markov chain Monte Carlo algorithm (DE-MCMC;  
314 Ter Braak, 2006; Turner, Sederberg, Brown, & Steyvers, 2013) to sample from the posterior  
315 with 15 interacting chains, each with 4000 iterations, the first 1500 of which were discarded  
316 as burn-in. We also implemented a migration algorithm (see Turner, Sederberg, Brown, &  
317 Steyvers, 2013), where chains were randomly migrated every 14 iterations between  
318 iterations 500 and 1100. We calculated Bayes factors through the Savage-Dickey ratio.  
319 Where we found evidence of group differences, we established the population effect size by  
320 dividing the posterior of the group difference ( $\delta$ ) by the posterior of the population standard  
321 deviation ( $\sigma$ ).

322 As shown in Table 1, the children with dyslexia were on average slightly older and of  
323 lower IQ than the typically developing children. As pre-registered, the first author (CM) ran a  
324 default Bayesian t-test using the BayesFactor R package (Morey & Rouder, 2018) which  
325 revealed weak, inconclusive evidence for the absence of group differences in age (BF in  
326 support of group differences = 0.33; Jeffreys, 1961). As we know that diffusion model  
327 parameters change with age (Manning et al., 2021a), and as we couldn’t conclusively rule  
328 out group differences in age, we also ran models which partialled out the effects of age from  
329 all of the parameters (using the residuals from the line of best fit between age and each of  
330 the parameters), in addition to our standard models. In our pre-registered analysis plan we  
331 decided not to control for performance IQ as it may relate to both group membership and  
332 decision-making in cognitively relevant ways (Dennis et al., 2009). The analysis files were  
333 posted on the Open Science Framework prior to unblinding (<https://osf.io/nwwf7/>), at which  
334 point all models were re-run on the unblinded dataset with correct group membership.

335

### 336 *EEG analysis for joint modelling*

337 We ran exploratory analysis on the unblinded dataset to investigate links between  
338 drift-rate and EEG activity. EEG data were band-pass filtered between 0.3 and 40 Hz in

339 NetStation and then exported for further processing in MATLAB using EEGLAB functions  
340 (Delorme & Makeig, 2004). We downsampled each participant's data to 250 Hz and selected  
341 only the data between the first fixation onset and the last offset period. We then bandpass-  
342 filtered between 0.3 and 40 Hz (due to insufficient attenuation of low frequencies by  
343 NetStation filters, Manning et al., 2019) and used EEGLAB's 'clean\_artifacts' function to  
344 remove bad channels, identify data segments with standard deviations over 15 and correct  
345 them using artifact subspace reconstruction (ASR; Chang et al., 2018). Missing channels  
346 were then interpolated. We then ran independent components analysis on 3000 ms epochs  
347 starting at fixation onset using an Infomax algorithm and subtracted ocular components from  
348 the continuous data. Finally, we average re-referenced the data. In line with the behavioural  
349 analyses, we excluded triggers for response events made <200 ms or >2500 ms after  
350 stimulus onset.

351         Following previous work, we used a data-driven component decomposition technique  
352 to identify spatiotemporally reliable patterns of activity across trials, which has the effect of  
353 maximising signal-to-noise ratio (Reliable Components Analysis, Dmochowski et al., 2012;  
354 Dmochowski & Norcia, 2015; Manning et al., 2019, 2021a). To do this, we epoched each  
355 participant's preprocessed continuous data from -600 ms to 200 ms around each response,  
356 and we baselined the data to the last 100 ms of the random motion period. We submitted the  
357 baselined epochs for participants in both groups to Reliable Components analysis for each  
358 task separately. The forward-model projections of the weights for the most reliable  
359 component for each task (which explained 28.7% and 27.1% of the reliability in the motion  
360 coherence and direction integration tasks, respectively) are shown in Figure 3. This  
361 component resembled the most reliable component found in our previous work (Manning et  
362 al., 2021a), which in turn resembles the centro-parietal positivity (O'Connell et al., 2012;  
363 Kelly and O'Connell, 2013). Build-up of activity in this component has been linked to drift-  
364 rate in typically developing children (Manning et al., 2021a). To investigate links with drift-  
365 rate in the current dataset, we projected each participant's continuous data through the

366 spatial weights for this component to yield a single component waveform for each participant  
367 for each task.

368 *[insert Figure 3 about here]*

369

370 In our paradigm, stimulus-locked and response-locked activity overlap temporally,  
371 with the degree of overlap relating to the participant's reaction time. Importantly, the extent of  
372 overlap could vary between groups and/or conditions (Ehinger & Dimigen, 2019). Thus, in  
373 order to obtain an EEG measure for inclusion in our model that reflects the decision-making  
374 process as purely as possible, and fully separate the contributions of stimulus-locked and  
375 response-locked activity, we used a linear deconvolution method to unmix overlapping  
376 stimulus-locked and response-locked activity in our component waveform using the Unfold  
377 toolbox (Ehinger & Dimigen, 2019). We modelled the continuous waveform for each  
378 participant by selecting a time window of -1000 ms to 1000 ms around each stimulus event  
379 or response event. We specified a design matrix with predictors for each difficulty level  
380 (difficult, easy) for each event type (stimulus, response). We then time-expanded the design  
381 matrix by adding a predictor for each timepoint sampled (i.e., every 4 ms from -1000 ms to  
382 1000 ms) for each event type. The reason for this 'time-expansion' is that each regressor in  
383 the resulting design matrix models the evoked response (either stimulus-locked or response-  
384 locked) at a particular point in time (Smith & Kutas, 2015; Ehinger & Dimigen, 2019); this is  
385 equivalent to the 'finite impulse response' approach to analysis of fMRI timeseries (Henson,  
386 Rugg and Friston, 2001). The predictors are therefore simply 'boxcar' functions at each point  
387 in time, rather than information relating to the stimulus display. Having constructed the  
388 design matrix, we identified segments with amplitudes above  $\pm 250$   $\mu\text{V}$  using a sliding 2000  
389 ms segment in 100 ms steps, and excluded these segments from the design matrix (mean  
390 2.72% of the data for each participant, range: 0 to 43%). We then fit the deconvolution model  
391 resulting in regression weights (betas) for each of the 2 event types, 2 difficulty levels and  
392 500 timepoints, which we used to construct regression waveforms (see Figures 4 and 5).  
393 Comparing the left and middle columns of Figures 4 and 5 shows that deconvolution led to

394 reduced amplitudes (which is expected as the non-deconvolved waveform contains a mix of  
395 overlapping stimulus-locked and response-locked activity).

396 *[insert Figures 4 & 5 about here]*

397

398 The non-deconvolved waveforms showed amplitude differences between difficult and  
399 easy levels (Figures 4 and 5, left column), as to be expected for an EEG measure which  
400 reflects the decision-making process. However, these differences across difficulty levels  
401 were not evident in the deconvolved waveforms (Figures 4 and 5, central column). The fact  
402 that the difference between difficulty levels changed as a result of deconvolution could  
403 suggest that the overlap between stimulus- and response-locked activity differs between  
404 difficulty levels, due to different RT distributions in each difficulty level. However, we found a  
405 difficulty level difference in the non-deconvolved waveforms even when matching the RT  
406 distributions for the easy and difficult levels, so that difficulty level differences could not be  
407 purely attributed to different RT distributions. We therefore suspected that the beta estimates  
408 may be noisy and that the deconvolution technique was overfitting the noise. Therefore, in  
409 the final step where we selected EEG measures for inclusion in the diffusion model, we re-  
410 ran the deconvolution model using a regularisation method which penalises the squared  
411 magnitude of the regression coefficients (ridge regression; see Kristensen et al., 2017) to  
412 minimise noise. Using this approach retained the difficulty level differences while minimising  
413 the noise in the waveforms (see right column of Figures 4 and 5). Specifically, we found the  
414 best regularisation parameter for each participant using cross-validation, and then took the  
415 mode across all participants and constrained the regularisation parameter to ensure that  
416 differences in regularisation did not contribute to group differences in resulting waveforms.  
417 The modal parameter value was 10 for the motion coherence task (5.5 and 10 for the  
418 typically developing children and children with dyslexia, separately) and 5 for the direction  
419 integration task (5 and 4.5 for the typically developing children and children with dyslexia,  
420 separately). We then fit a regression slope to each participant's average deconvolved

421 waveform for each difficulty level between -200 ms to 0 ms around the time of the response  
 422 to obtain a slope measure which we entered into the diffusion model and related to drift-rate.

423 To assess the relationship between drift-rate and the EEG component discussed  
 424 above, we used a joint modelling approach (Turner et al., 2013, 2015, 2016, Evans et al.,  
 425 2018; Knowles et al., 2019). Specifically, we estimated additional hyper-parameters for the  
 426 correlation between the *v.mean* parameter and the average of the EEG measure (slope of  
 427 centro-parietal component activity between -200 ms to 0 ms before response) over difficulty  
 428 levels (*EEG.mean*), and between the *v.diff* parameter and the difference in the EEG  
 429 measure between difficulty levels (*EEG.diff*). Specifically, this meant that the structure of the  
 430 original hierarchical model (with age partialled out) was only different for the drift-rate  
 431 parameter, which was now a bivariate normal with the EEG measure:

$$\begin{aligned}
 & [v_{p1} - v_{p2}, EEG_{p1} - EEG_{p2}] \sim \\
 & BN([\mu_{v.diff} \pm \delta_{v.diff}, \mu_{EEG.diff} \pm \delta_{EEG.diff}], [\sigma_{v.diff}^2, \sigma_{v.diff}\sigma_{EEG.diff}\rho, \sigma_{EEG.diff}\sigma_{v.diff}\rho, \sigma_{EEG.diff}^2]) \\
 & [(v_{p1} + v_{p2})/2, (EEG_{p1} + EEG_{p2})/2] \sim \\
 & BN([\mu_{v.mean} \pm \delta_{v.mean}, \mu_{EEG.mean} \pm \delta_{EEG.mean}], [\sigma_{v.mean}^2, \sigma_{v.mean}\sigma_{EEG.mean}\rho, \sigma_{EEG.mean}\sigma_{v.mean}\rho, \sigma_{EEG.mean}^2]) \\
 & \mu_{EEG.diff} \sim N(0,0.5) \\
 & \mu_{EEG.mean} \sim N(0,1) \\
 & \sigma_{EEG.diff}, \sigma_{EEG.mean} \sim \Gamma(1,1) \\
 & \delta_{EEG.diff}, \delta_{EEG.mean} \sim N(0,0.01) \\
 & \rho \sim U(-1,1)
 \end{aligned}$$

432 where  $\rho$  refers to the correlation between drift-rate and the EEG measure. Note that we  
 433 again used DE-MCMC with 15 interacting chains to sample from the posterior of the joint  
 434 model, though due to the greater computational burden of the model we used 3000  
 435 iterations, of which the first 1000 were discarded as burn-in and no migration algorithm was  
 436 implemented. Furthermore, we estimated two different variants of this joint model: one where  
 437 the correlations were constrained to be the same across groups, which would allow for the  
 438 estimation of more precise posteriors due to the limited sample size, and another less  
 439 constrained version where the correlations were estimated separately for each group.

440 *Data and code availability*

441 Analysis scripts and output files are available at: <https://osf.io/nvwf7/>. Data will be made  
442 available on the UK Data Service after the manuscript has been accepted for publication.

443

444 **Results**

445 *[insert Figure 6 about here]*

446

447 *Diffusion modelling of behavioural data*

448 Figure 6 summarises the accuracy and response time data subjected to diffusion  
449 modelling. This figure shows that the children with dyslexia had slightly slower median  
450 response times compared to typically developing children, on average, and were slightly less  
451 accurate in the direction integration task, particularly on the difficult trials. However, there  
452 was substantial overlap between the groups with considerable variability within each group.  
453 These behavioural data were well-fit by our diffusion models, as shown by the cumulative  
454 density functions in Figure 7. All chains were well-converged, as reflected by Gelman-Rubin  
455 diagnostic values (Gelman & Rubin, 1992) close to 1 ( $M = 1.00$ , range = 1.00 – 1.07).

456

457 *[insert Figure 7 about here]*

458

459

460 Figure 8 shows the prior and posterior distributions for the group-level parameters  
461 that reflect the difference between groups for each of the 5 parameters (*v.mean*, *a*, *ter*, *v.diff*,  
462 *beta*), along with Bayes factors. Bayes factors above 1 reflect more evidence for the  
463 alternative hypothesis of group differences compared to the null hypothesis, whereas Bayes  
464 factors below 1 reflect relatively more evidence for the null hypothesis than the alternative  
465 hypothesis. We use the heuristic that Bayes factors between 1/3 and 3 constitute only weak,  
466 inconclusive evidence (Jeffreys, 1961).

467

468 *[insert Figure 8 about here]*

469 In support of our first hypothesis, children with dyslexia had reduced drift-rates in the  
470 motion coherence task compared to typically developing children, as shown by the leftward  
471 shift in the posterior distribution of  $v$ . *mean* in Figure 8. When age was partialled out, there  
472 was moderate evidence in favour of group differences (BF = 4.57, population effect size  $M =$   
473  $-.18$ , 95% CI:  $[-.40, .02]$ ). The evidence was weaker when age was not partialled out (BF =  
474 1.75). Interestingly, the same pattern was found in support of our second hypothesis, with  
475 children with dyslexia also showing reduced drift-rates in the direction integration task  
476 compared to typically developing children. Again, there was moderate evidence for group  
477 differences when age was controlled for (BF = 4.28, population effect size  $M = -.21$ , 95% CI:  
478  $[-.45, .02]$ ), but weak evidence when age was not controlled for (BF = 1.71).

479 Our third hypothesis was that children with dyslexia would show increased boundary  
480 separation. Although children with dyslexia did have slightly higher boundary separation  
481 compared to typically developing children (indicated by a small rightward shift in the  
482 posterior distribution of  $a$  in Figure 8), particularly in the motion coherence task, the evidence  
483 remained inconclusive, even when controlling for age. Our final hypothesis was that there  
484 would be no group differences in non-decision time (*ter*) in either task. Figure 8 shows little  
485 difference between the groups in this parameter, but the Bayes factors are close to 1,  
486 suggesting inconclusive evidence. Therefore, more data would be required to make firm  
487 conclusions regarding these hypotheses.

488 These pre-registered analyses did not control for performance IQ because it could be  
489 meaningfully related to both decision-making parameters and group membership, and  
490 investigating its contribution to both was beyond the scope of our multi-level modelling  
491 approach. However, as there was an indication of a relationship between performance IQ  
492 and drift-rate (Figure 9), and as both performance IQ and drift-rate differed between the  
493 groups, we investigated these links further with an exploratory analysis which partialled out  
494 the effects of both age and performance IQ (Figure 10). In brief, BFs of 2.3 and 2.38 in the

495 two tasks continue to provide weak evidence for group differences in mean drift-rate when  
 496 both age and PIQ are controlled for.

497 *[insert Figures 9 and 10 about here]*

498

499 *Joint modelling of EEG and behavioural data*

500

501 *[insert Figure 11 about here]*

502

503 Figure 11 shows the distribution of slope measures that were extracted from each  
 504 participant's deconvolved (with regularisation) response-locked waveform, which were used  
 505 in joint modelling to explore links between EEG and model parameters. While there was  
 506 considerable between-participants variability, the children with dyslexia had shallower slopes  
 507 than the typical children, on average. A Bayesian repeated measures ANOVA in JASP  
 508 (JASP Team, 2020) showed that, in the motion coherence task, the best model of EEG  
 509 slope measures included both the within-participants factor of difficulty level, the between-  
 510 participants factor of group and an interaction term. When averaging across models, there  
 511 was strong evidence for including a main effect of group ( $BF_{\text{incl}} = 14.70$ ) and a group by  
 512 difficulty level interaction ( $BF_{\text{incl}} = 4.65$ ). Yet in the direction integration task, the best model  
 513 of EEG slope measures included only the within-participants factor of difficulty, with  
 514 inconclusive evidence for including a main effect of group ( $BF_{\text{incl}} = 0.70$ ) or a group by  
 515 difficulty level interaction ( $BF_{\text{incl}} = 0.49$ ). Therefore it seems that the build-up of activity in the  
 516 centro-parietal component is clearly reduced in children with dyslexia in the motion  
 517 coherence task, but the reduction is not compelling in the direction integration task.

518 Next we established whether this EEG measure was related to drift-rate across the  
 519 whole sample, estimating a single correlation for both groups, with the effects of age  
 520 partialled out. For both tasks, the EEG measure was positively related to both the mean drift-  
 521 rate across difficulty levels, though the evidence was only weak in the case of the direction  
 522 integration task (motion coherence: posterior mean  $r = .44$ , 95% credible intervals (CI) =

523 [.26, .6], BF = 8869.49; direction integration: posterior mean  $r = .25$ , CI = [.03, .45], BF =  
 524 1.65). The posterior means were in the direction of a positive relationship between the  
 525 difference in EEG measure and the difference in drift rate between difficulty levels, although  
 526 the evidence was inconclusive with relatively more evidence for the null hypothesis (motion  
 527 coherence: posterior mean  $r = .22$ , CI = [-.02, .44], BF = .73; direction integration: posterior  
 528 mean  $r = .17$ , CI = [-.08, .4], BF = 0.43; see Figure 12 for scatterplots).

529

530 *[insert Figure 12 about here]*

531

532 Next we fit joint models in which we estimated a separate correlation coefficient  
 533 between drift-rate and the EEG measure for the children with dyslexia and typical children  
 534 (Figure 13). Note that our intention was not to explicitly test for differences in correlations  
 535 between groups, but rather to see if the previous findings seem to hold for each group; any  
 536 separation between the groups below is intended to merely describe our estimated posterior  
 537 distributions. A positive correlation can be seen for both groups in the motion coherence task  
 538 for the mean drift-rate across difficulty levels (typical: posterior mean  $r = .41$ , CI = [.13, .63],  
 539 BF = 7.45; dyslexia: posterior mean  $r = .43$ , CI = [.15, .64], BF = 12.75). The posterior means  
 540 were in the direction of a positive relationship for the difference in drift-rate between difficulty  
 541 levels, but the evidence was inconclusive with relatively more evidence for the null  
 542 hypothesis (typical: posterior mean  $r = .18$ , CI = [-.2, .51], BF = .39; dyslexia: posterior mean  
 543  $r = .20$ , CI = [-.12, .49], BF = .46). The strength of correlations was weaker in the direction  
 544 integration task, particularly for the typical children, for whom the Bayes factors suggested  
 545 moderate evidence for no relationship (mean drift-rate across difficulty levels: posterior mean  
 546  $r = .10$ , CI = [-.22, .4], BF = .29; difference between difficulty levels: posterior mean  $r = .04$ ,  
 547 CI = [-.31, .38], BF = .24). The strength of the correlations in children with dyslexia were  
 548 slightly stronger than in the typical children, with the mean drift-rate across difficulty levels  
 549 showing weak evidence for a relationship, though the difference in drift-rate between  
 550 difficulty levels showed weak evidence for no relationship (mean drift-rate across difficulty

551 levels: posterior mean  $r = .34$ , CI = [.04, .58], BF = 2.59; difference between difficulty levels:  
552 posterior mean  $r = .24$ , CI = [-.09, .53], BF = .61).

553

554 *[insert Figure 13 about here]*

555

## 556 **Discussion (1500 words)**

557 We analysed the performance of children with dyslexia and typical children in two  
558 global motion tasks using diffusion modelling, to identify the processing stages that are  
559 altered in dyslexia. In both the motion coherence and direction integration tasks, children  
560 with dyslexia accumulated sensory evidence more slowly than typical children, on average,  
561 once controlling for age. Moreover, we found a neural correlate of this evidence  
562 accumulation process that was attenuated in dyslexia in the motion coherence task, thus  
563 linking brain and behavioural measures with a latent model parameter.

564 The finding of reduced evidence accumulation for children with dyslexia during the  
565 motion coherence task echoes O'Brien and Yeatman (2020) and helps to explain previous  
566 reports of elevated motion coherence thresholds in dyslexia (Benassi et al., 2010).  
567 Importantly, the current study goes further by showing that reduced evidence accumulation  
568 is also found in a direction integration task that does not require segregating signal dots from  
569 noise dots. This result suggests that dyslexic individuals have general difficulties with  
570 extracting global motion information, rather than solely difficulties with noise exclusion (cf.  
571 Conlon et al., 2012; Sperling et al., 2006) –in line with reports of atypical performance in an  
572 illusory motion task without noise exclusion requirements (Gori et al., 2015, 2016). These  
573 general difficulties could reflect reduced temporal and/or spatial integration of motion signals  
574 (Benassi et al., 2010; Hill & Raymond, 2002; Raymond & Sorensen, 1998). This conclusion  
575 does not negate the possibility that dyslexic individuals face additional difficulties when  
576 segregating signal from noise, as we suggested based on stimulus-locked analyses using a  
577 similar dataset (Toffoli et al., 2021).

578 By supplementing our diffusion modelling analysis with EEG, we identified a neural  
579 index of reduced evidence accumulation in dyslexia. Specifically, we used a data-driven  
580 component decomposition technique to find a centro-parietal component previously linked to  
581 decision-making (Kelly and O'Connell, 2013; O'Connell et al., 2012; Manning et al., 2021a),  
582 and then 'unmixed' overlapping stimulus- and response-locked activity. In the motion  
583 coherence task, we found that children with dyslexia showed a shallower build-up in the  
584 response-locked centro-parietal component compared to typical children, and the gradient of  
585 the build-up was positively correlated with drift-rate in the joint model. While the EEG  
586 analysis was exploratory, the results are consistent with an earlier study of typically  
587 developing children (Manning et al., 2021a) and follow our hypothesised pattern  
588 (<https://osf.io/enkwm>). Similarly, Stefanac et al. (2021) reported reduced centro-parietal  
589 build-up in children with dyslexia compared to chronological and reading age-matched  
590 controls. Yet, in our direction integration task, we found no compelling evidence for reduced  
591 centro-parietal build-up in children with dyslexia and the evidence for a relationship between  
592 this EEG measure and drift-rate was weaker. This suggests that the magnitude of the  
593 centro-parietal positivity and its association with drift-rate may be group- and task-  
594 dependent, to some extent (see also Lui et al., 2021).

595 Alongside reductions in drift-rate, we hypothesised that children with dyslexia would  
596 show wider boundary separation compared to typically developing children, reflecting more  
597 cautious responses, and no differences in non-decision time. We found some evidence for  
598 increased boundary separation in children with dyslexia in the motion coherence task, but  
599 this was inconclusive. There was also inconclusive evidence for group differences in non-  
600 decision time. These results are not at odds with O'Brien and Yeatman (2020), but suggest  
601 that more data are required to reach a firm conclusion regarding these parameters.  
602 Seemingly any group differences in these parameters are more subtle than group  
603 differences in drift-rate. We note that the inferential method used by O'Brien and Yeatman  
604 (2020) differed from our own: while they also fit a hierarchical Bayesian model, they then  
605 extracted point estimates of diffusion model parameters for each individual to draw statistical

606 inferences. Importantly, this means that O'Brien and Yeatman (2020) ignored the uncertainty  
607 in the individual-level parameters, which can inflate the evidence in favour of the winning  
608 model (Boehm et al., 2018; Evans & Wagenmakers, 2019).

609         Together with the results from stimulus-locked analyses using a similar dataset  
610 (Toffoli et al., 2021), our results suggest that early sensory encoding of motion information is  
611 not altered in children with dyslexia. While differences in drift-rate cannot completely tease  
612 apart sensory and decision-making processes, in the current study we found no evidence of  
613 group differences in non-decision time – a measure which includes the time taken for  
614 sensory encoding. Moreover, Toffoli et al. showed that early peaks reflecting motion-specific  
615 processing were similar in children with dyslexia and typically developing children, with  
616 differences arising only after ~430 ms following stimulus onset, specifically in the motion  
617 coherence task. The current analyses suggest that differences in dyslexia arise due to the  
618 efficiency with which evidence is extracted from global motion stimuli and integrated towards  
619 a decision bound, which is often attributed to parietal areas (Hanks et al., 2006; Shadlen &  
620 Newsome, 1996; 2001; de Lafuente et al., 2015). Without a comparable form task, it is  
621 unclear from the current study whether reduced evidence accumulation is restricted to tasks  
622 that tax the dorsal stream. However, we suggest that *within* the magnocellular/dorsal stream,  
623 early sensory processing is unaffected in dyslexia with group differences emerging only at  
624 later processing stages, including those involved in decision-making. While this conclusion  
625 contrasts studies indicating early alterations of the magnocellular pathway in dyslexia  
626 (Giraldo-Chica et al., 2015; Livingstone et al., 1991; Perani et al., 2021; Stein, 2001, 2019;  
627 Stein & Walsh, 1997), the global motion tasks used in the current study are not ideally  
628 placed to isolate magnocellular processes (Skottun, 2011; Skottun & Skoyles, 2006, 2008;  
629 Skottun, 2016). Future work will be required to determine how specific reduced evidence  
630 accumulation in dyslexia is to visual motion processing. Slower responses have been  
631 reported in dyslexia for other tasks (Catts et al., 2002, Nicolson & Fawcett, 1994) which  
632 could reflect pervasive reduced evidence accumulation, and reduced global integrative  
633 processes have been reported in static tasks in children with dyslexia (Franceschini et al.,

634 2017a). However, slowed responses could arise for different reasons (e.g., increased non-  
635 decision time, wider boundary separation), so diffusion model decompositions on various  
636 tasks are required.

637         A number of future research directions emerge. What cognitive skills other than  
638 magnocellular / dorsal stream processing contribute to reduced drift-rate in dyslexia?  
639 General processing speed is a unique predictor of word reading and comprehension  
640 (Christopher et al., 2012) and RAN is a recognized independent contributor to variation in  
641 reading ability, complementing phonological skills (e.g., O'Brien & Yeatman, 2020). Future  
642 work will need to establish the extent to which reduced processing speed and slower RAN  
643 associate with reduced drift-rate in dyslexia. Additionally, performance IQ varied across our  
644 two groups and was associated with drift-rate. Exploratory models revealed that, even when  
645 controlling for both age and performance IQ, there was still relatively more evidence for  
646 group differences in drift-rate than no group differences. Yet the evidence was weaker than  
647 in models controlling only for age. Importantly, partialling out differences in performance IQ  
648 could remove some of the variance related to the group differences we are interested in, as  
649 atypical development could lead to both dyslexia and reduced IQ (Dennis et al., 2009).  
650 Indeed, performance IQ has been shown to strongly predict reading skills, independently of  
651 phonological skills (O'Brien & Yeatman, 2020). Future work will need to investigate the  
652 contribution of processing speed and performance IQ to decision making across the  
653 spectrum of reading abilities. Future research will also be required to explain the  
654 considerable between-participants variability in model and EEG parameters in children with  
655 and without dyslexia.

656         By combining diffusion modelling and EEG measures that are sensitive to the  
657 multiple processes contributing to motion perception, we have uncovered differences  
658 between children with dyslexia and typically developing children that could not be observed  
659 in behavioural responses alone. Moreover, diffusion modelling allows motion sensitivity to be  
660 measured without confounding speed-accuracy tradeoffs. Given that reduced behavioural  
661 sensitivity to motion has been reported in a range of other disorders (Braddick et al., 2003;

662 Chen et al., 2003; McKendrick & Badcock, 2004), we suggest that diffusion modelling may  
663 provide a useful framework to identify convergence and divergence across different  
664 conditions, with implications for understanding the development of these conditions and their  
665 relationship to other cognitive processes.

666 Future work should establish whether differences in evidence accumulation of motion  
667 information contribute causally to the reading difficulties experienced by children with  
668 dyslexia. Some studies have suggested a causal relationship between motion perception  
669 and reading difficulties (e.g., Boets et al., 2011; Ebrahimi et al., 2019; Gori et al., 2016;  
670 Kevan & Pammer, 2009; Lawton, 2016; Qian & Bi, 2015), so it would be interesting to know  
671 if evidence accumulation processes can be trained to improve reading ability. In support of  
672 this possibility, action video game training has been shown to improve motion perception by  
673 acting on the evidence accumulation phase (Green et al., 2010) and action video game  
674 training has also been linked to improved reading skills in children with dyslexia  
675 (Franceschini et al., 2013; 2017b, Franceschini & Bertoni, 2019; Bertoni et al., 2019; 2021).  
676 Such causal links will need to be investigated in future work using training or intervention  
677 designs.

678

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938  
939

940 **Figure Legends**

941

942 **Figure 1. Schematic representation of the decision-making process in the diffusion**  
943 **model for a trial with rightward motion**

944 Decision-making process represented as a noisy accumulation of evidence from a starting  
945 point,  $z$ , towards one of two decision bounds. In our motion tasks, the decision bounds  
946 correspond to left and right responses. Boundary separation,  $a$ , represents the width  
947 between the two bounds and reflects response caution. Wider decision boundaries reflect  
948 that more evidence is required before making a decision (i.e., more cautious responses).  
949 Drift-rate,  $v$ , reflects the rate of evidence accumulation, which depends on both the  
950 individual's sensitivity to a stimulus and the stimulus strength. Non-decision time,  $t_{er}$ , is the  
951 time taken for sensory encoding processes prior to the decision-making process and  
952 response generation processes after a bound is reached.

953

954 **Figure 2. Schematic representation of trial procedure.**

955 The trial started with an initial *fixation* period that was followed by a *random motion* period  
956 consisting of random, incoherent moving dots, which was in turn followed by a *stimulus*  
957 containing leftward or rightward global motion. The child was asked to report the direction  
958 using a response box. After the response or after the maximum stimulus duration elapsed  
959 (2500 ms), the stimulus remained on the screen for a short *offset* period. Note that arrows  
960 (indicating movement) and dotted lines (marking the square stimulus region) are presented  
961 for illustration only. The stimulus shown here is from the motion coherence task, where a  
962 proportion of dots move coherently. In the direction integration task, dot directions were  
963 taken from a Gaussian distribution. Figure reproduced from <https://osf.io/wmtpx/> under a CC-  
964 BY4.0 license.

965

966 **Figure 3. Scalp topographies and temporal dynamics for the most reliable component**  
967 **in the motion coherence and direction integration tasks**

968 Topographic visualisations of the forward-model projections of the most reliable component  
969 (left) reflecting the weights given to each electrode following reliable components analysis  
970 (RCA) on data from all participants pooled across difficulty level, for the motion coherence  
971 task (upper) and direction integration task (lower). The waveforms (right) show the temporal  
972 dynamics of the component.

973

974 **Figure 4. Group average stimulus-locked and response-locked evoked potentials for**  
975 **the motion coherence task**

976 Average ( $\pm 1$ SEM) stimulus-locked (upper) and response-locked (lower) evoked potentials for  
977 typically developing children (grey) and children with dyslexia (blue) in the motion coherence  
978 task for difficult and easy levels. The left column shows non-deconvolved group average  
979 waveforms. The central column shows deconvolved group average waveforms (without  
980 regularisation). The right column shows deconvolved group average waveforms with  
981 regularisation (ridge regression). The vertical line at 0 ms indicates when the stimulus phase  
982 started (stimulus-locked) or when the response was made (response-locked).

983

984 **Figure 5. Group average stimulus-locked and response-locked evoked potentials for**  
985 **the direction integration task**

986 Average ( $\pm 1$ SEM) stimulus-locked (upper) and response-locked (lower) evoked potentials for  
987 typically developing children (grey) and children with dyslexia (blue) in the direction  
988 integration task for difficult and easy levels. The left column shows non-deconvolved group  
989 average waveforms. The central column shows deconvolved group average waveforms  
990 (without regularisation). The right column shows deconvolved group average waveforms with  
991 regularisation (ridge regression). The vertical line at 0 ms indicates when the stimulus phase  
992 started (stimulus-locked) or when the response was made (response-locked).

993

994

995 **Figure 6. Accuracy and median response time (RT) for correct trials**

996 Violin plots showing the kernel probability density for each group's accuracy (left) and  
 997 median RT (s) for correct trials (right) for each difficulty level and each task (upper: motion  
 998 coherence; lower: direction integration). Data for typically developing children and children  
 999 with dyslexia are presented in grey and blue, respectively. Dots and vertical lines represent  
 1000 the group mean and  $\pm 1$  SEM.

1001

1002 **Figure 7. Model fits**

1003 Defective cumulative density function plots for each of the four models, for typically  
 1004 developing children (upper rows) and children with dyslexia (bottom rows) for difficult and  
 1005 easy levels. Green represents correct responses and red represents error responses, at  
 1006 each of 9 quantiles. The dots reflect the observed data and crosses with connecting lines  
 1007 reflect the model fit. The dots and crosses at 2.5 seconds reflect the observed and model  
 1008 predicted misses.

1009

1010 **Figure 8. Prior and posterior density distributions**

1011 Prior (green) and posterior (purple) density distributions for the group-level parameters  
 1012 reflecting group differences in each of the 5 model parameters ( $v.mean$  = mean drift-rate  
 1013 across difficulty levels;  $a$  = boundary separation;  $ter$  = non-decision time;  $v.diff$  = difference in  
 1014 mean drift-rate between difficulty levels;  $z/a$  = relative starting point) for each task. The upper  
 1015 inset shows a schematic of the model parameters shown. The leftmost columns show the  
 1016 results of the standard model and the rightmost columns show the results of the model with  
 1017 age partialled out. Negative values reflect lower parameter values in the dyslexia group  
 1018 compared to the typically developing group. BF = Savage-Dickey Bayes factors in favour of  
 1019 the alternative hypothesis ( $H_1$ ) over the null hypothesis ( $H_0$ ).  $BF > 1$  support  $H_1$ .

1020

1021

1022 **Figure 9. Scatterplots plotting individual parameter estimates against performance IQ**

1023 Maximum likelihood estimates contained within the posterior for each participant's mean  
 1024 drift-rate across difficulty levels (*v.mean*), boundary separation (*a*), non-decision time (*ter*),  
 1025 difference in drift-rate between difficulty levels (*v.diff*), and starting point (*z/a*), plotted as a  
 1026 function of performance IQ (PIQ), for the motion coherence task (left column) and direction  
 1027 integration task (right column). Typically developing children are plotted in grey and children  
 1028 with dyslexia are plotted in blue.

1029

1030 **Figure 10. Exploratory analyses: prior and posterior density distributions for model**  
 1031 **with age and performance IQ partialled out**

1032 While our pre-registered analysis did not control for performance IQ, we conducted an  
 1033 exploratory analysis to investigate whether group differences in drift-rate were still apparent  
 1034 when controlling for performance IQ. The figure shows prior (green) and posterior (purple)  
 1035 density distributions for the group-level parameters reflecting group differences in each of  
 1036 the 5 model parameters (*v.mean* = mean drift-rate across difficulty levels; *a* = boundary  
 1037 separation; *ter* = non-decision time; *v.diff* = difference in mean drift-rate between difficulty  
 1038 levels; *z/a* = relative starting point) for each task, when both age, performance IQ (PIQ) and  
 1039 their interaction are partialled out. Negative values reflect lower parameter values in the  
 1040 dyslexia group compared to the typically developing group. BF = Savage-Dickey Bayes  
 1041 factors in favour of the alternative hypothesis ( $H_1$ ) over the null hypothesis ( $H_0$ ).  $BF > 1$   
 1042 support  $H_1$ . As in Figure 8, the posterior distribution for *v.mean* is shifted leftwards, reflecting  
 1043 lower mean drift-rate in the dyslexia group than the typically developing group. The  
 1044 corresponding Bayes factors are smaller in these analyses, indicating weaker evidence for  
 1045 group differences. As we reflect on in the Discussion of the main manuscript, the decision to  
 1046 partial out PIQ should not be taken lightly, as PIQ seems to contribute to both decision  
 1047 making variables (drift-rate) and group differences, so it is likely that partialling out PIQ  
 1048 removes some of the variance related to the group differences we are interested in.

1049

1050 **Figure 11. EEG slope measure extracted for inclusion in the joint model**

1051 Violin plots showing the kernel probability density for the EEG slope measure extracted for  
 1052 inclusion in the joint model for each group (typically developing: grey; dyslexia: blue) for  
 1053 each difficulty level. The extracted measure was the slope of a linear regression line fitted to  
 1054 each participant's deconvolved (with regularisation) response-locked waveform, from 200 ms  
 1055 prior to the response to the response (see shaded area of schematic response-locked  
 1056 waveform in inset). The dotted line reflects a flat slope. Dots and vertical lines represent the  
 1057 group mean and  $\pm 1$  SEM.

1058

1059 **Figure 12. Scatterplots showing relationship between drift-rate and EEG**

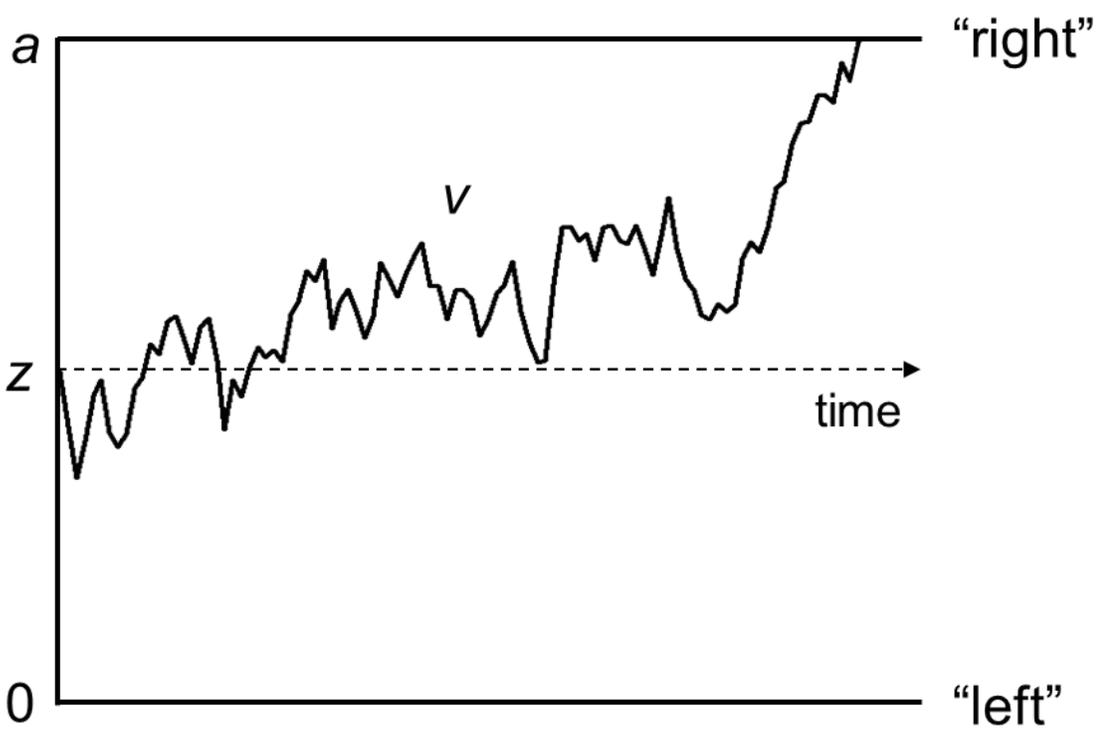
1060 Left panels show maximum likelihood estimates contained within the posterior for each  
 1061 participant's mean drift-rate across difficulty levels (*v.mean*) plotted against the slope of EEG  
 1062 activity averaged across difficulty levels (*EEG.mean*) for the motion coherence (top) and  
 1063 direction integration (bottom) tasks. Right panels show point estimates for each participant's  
 1064 difference in drift-rate between difficulty levels (*v.diff*) plotted against the difference in slopes  
 1065 of EEG activity between the two difficulty levels (*EEG.diff*), for each task. Typically  
 1066 developing children are plotted in grey and children with dyslexia are plotted in blue.

1067

1068 **Figure 13. Posterior density plots showing the correlation between drift-rate and the**  
 1069 **EEG measure**

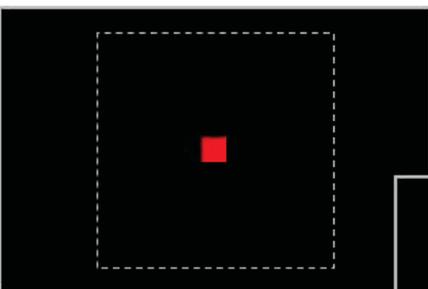
1070 Inset provides a schematic representation of the drift-rate parameter (*v*; left) and EEG  
 1071 measure (slope of response-locked waveform from -200 ms to 0 ms around the response;  
 1072 right) that were correlated in the joint model, where  $\rho$  represents the correlation. Posterior  
 1073 density plots in the left column reflect the correlation between the mean drift-rate across  
 1074 difficulty levels (*v.mean*) and the mean EEG slope measure across difficulty levels  
 1075 (*EEG.mean*). Posterior density plots in the right column reflect the correlation between the  
 1076 difference in drift-rate between difficulty levels (*v.diff*) and the difference in EEG slope  
 1077 measure between difficulty levels (*EEG.diff*). Plots for the motion coherence task are

1078 presented in the upper row and plots for the direction integration task are presented in the  
1079 lower row. The orange distribution shows the correlation across all participants, and the grey  
1080 and blue distributions show separate correlations estimated for typical children and children  
1081 with dyslexia, respectively.

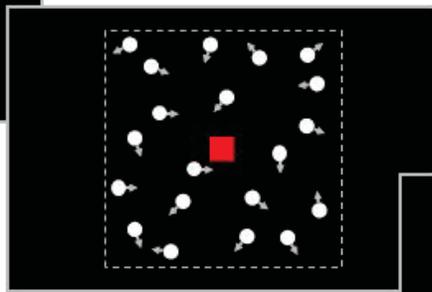


stimulus

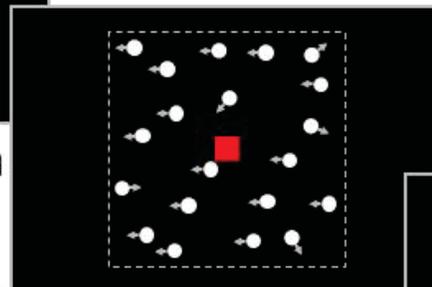
response



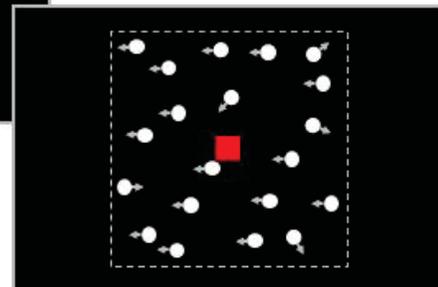
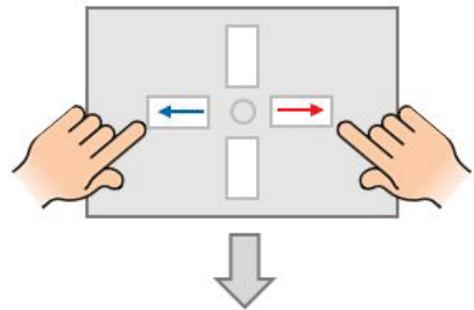
**Fixation**  
800-1000ms



**Random motion**  
800-1000ms

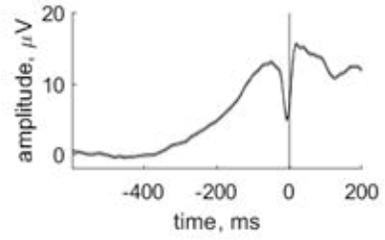
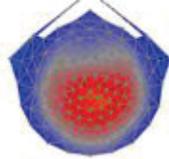


**Stimulus**  
until response  
OR 2500ms

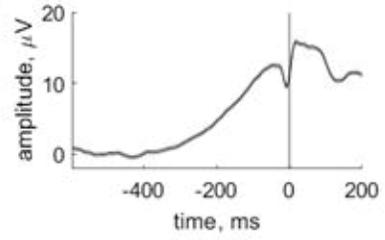
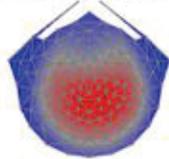


**Offset**  
200-400ms

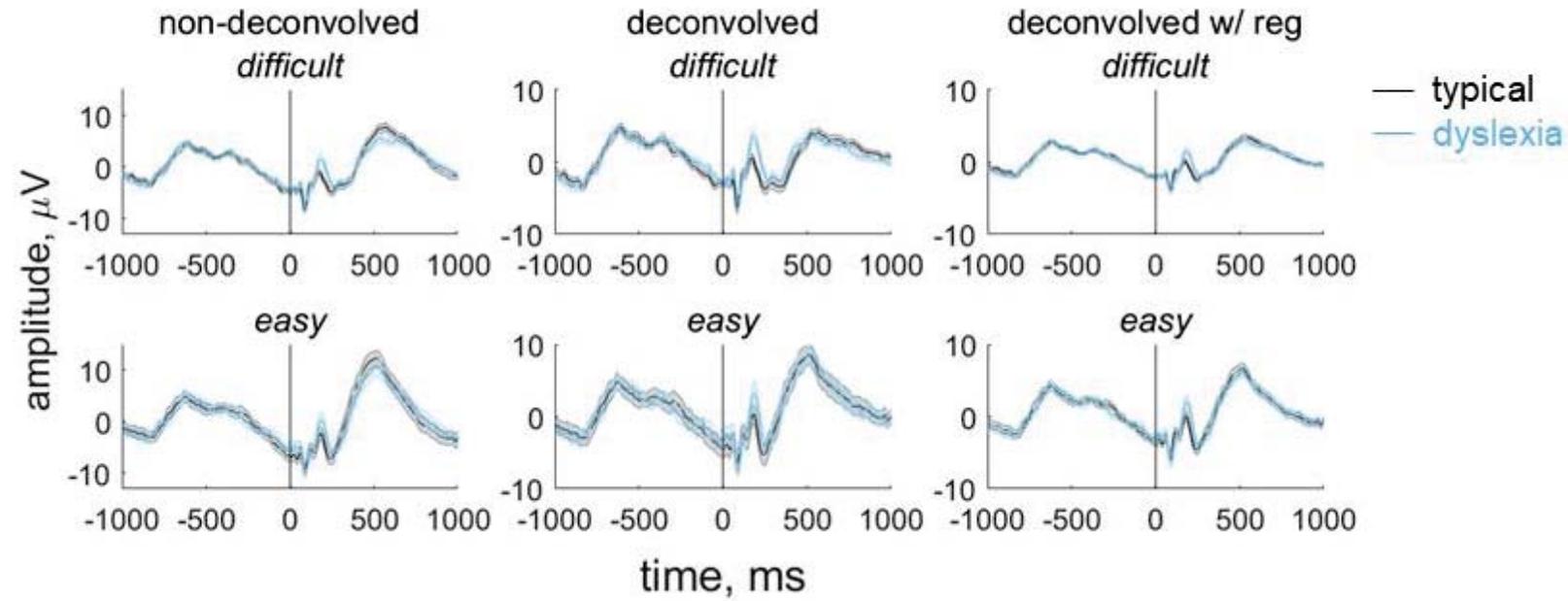
**motion coherence**



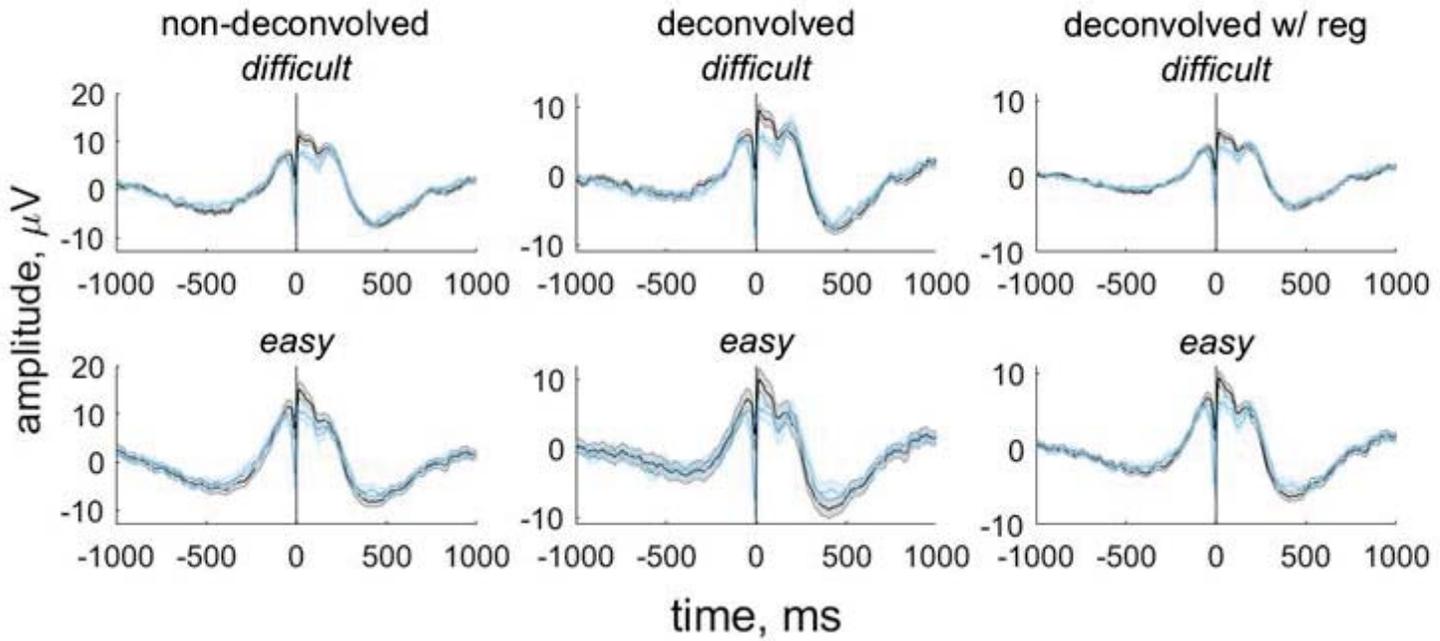
**direction integration**



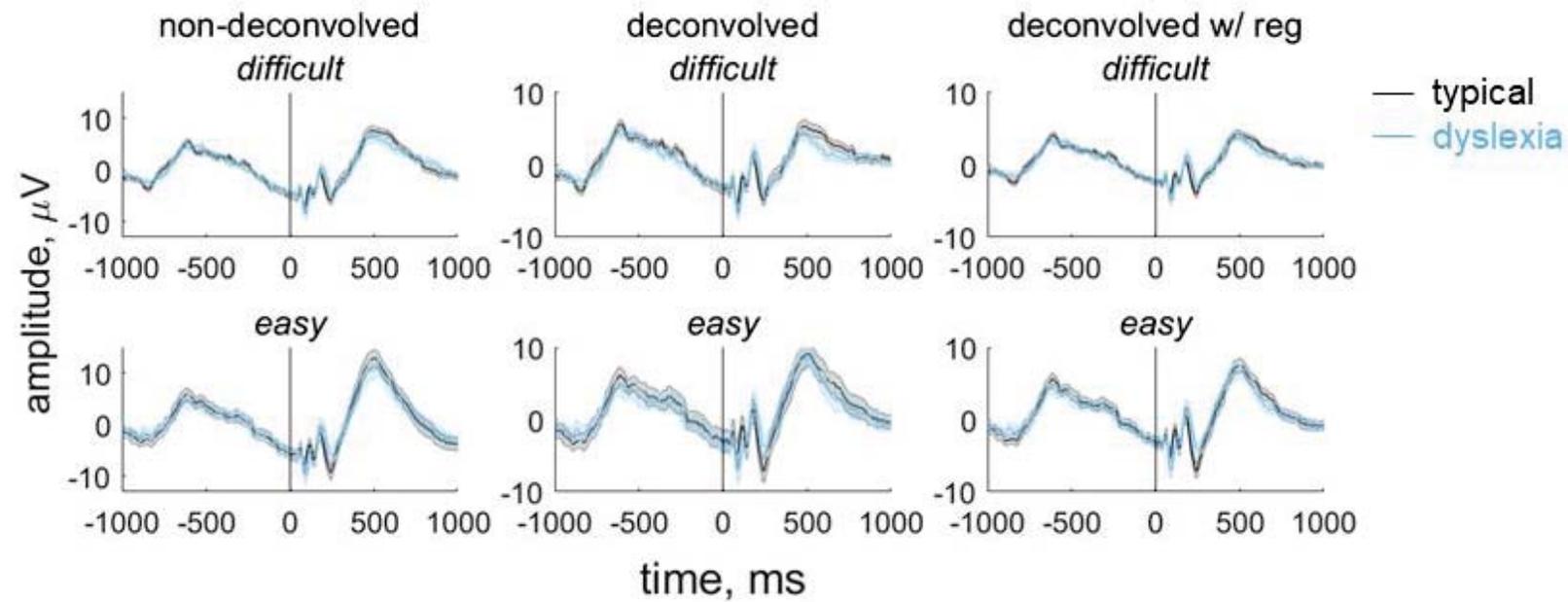
### stimulus-locked



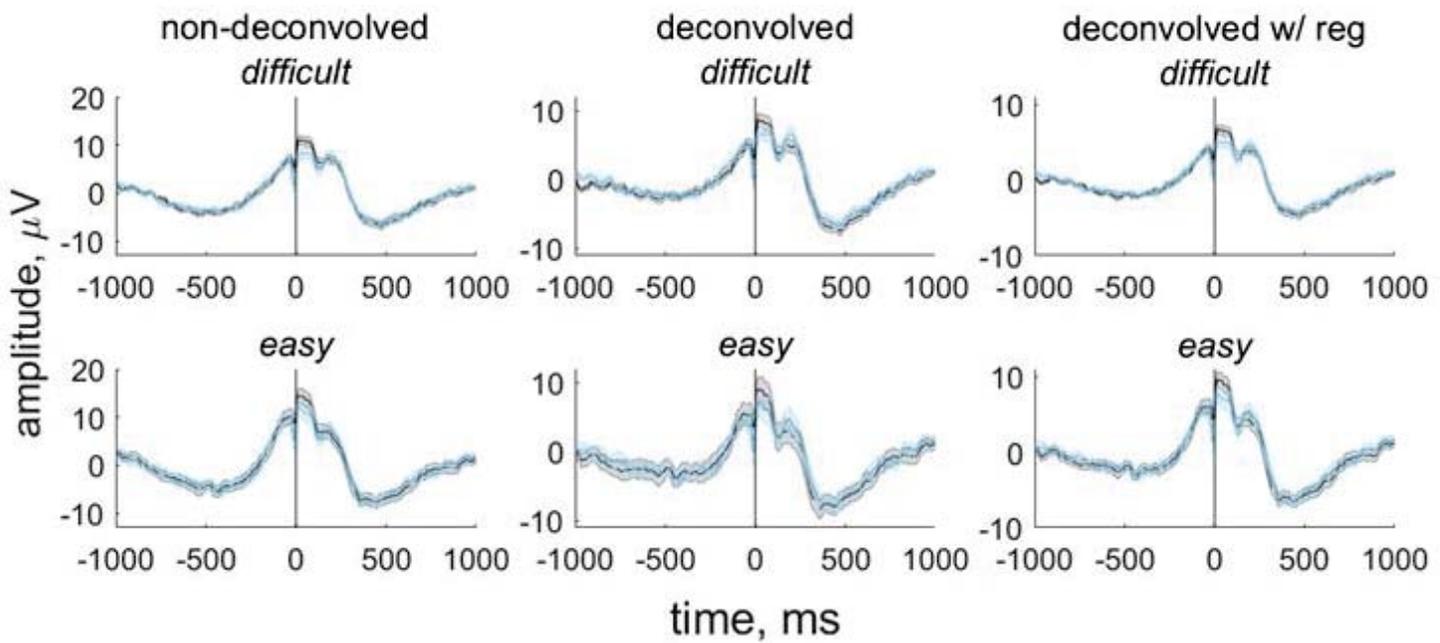
### response-locked



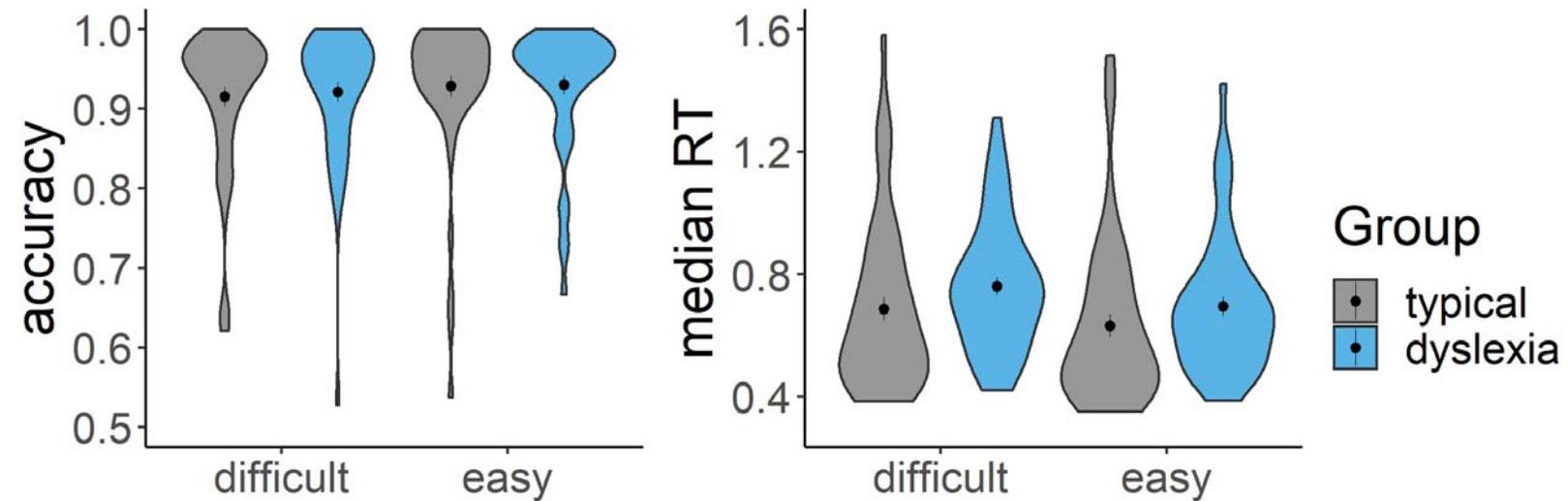
### stimulus-locked



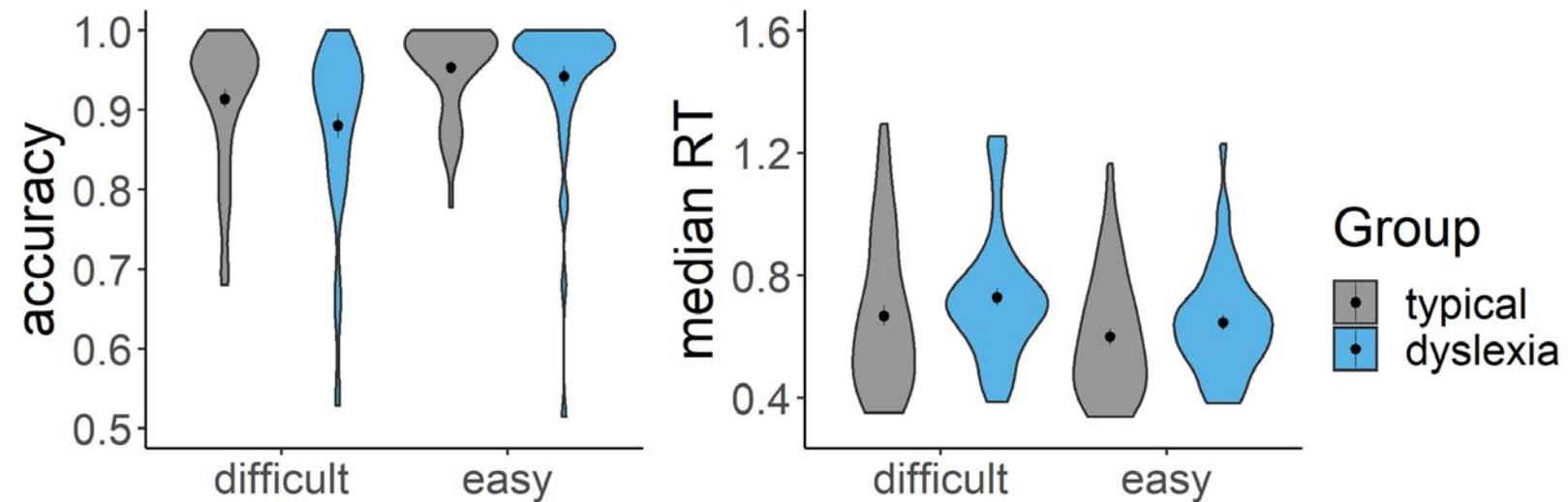
### response-locked

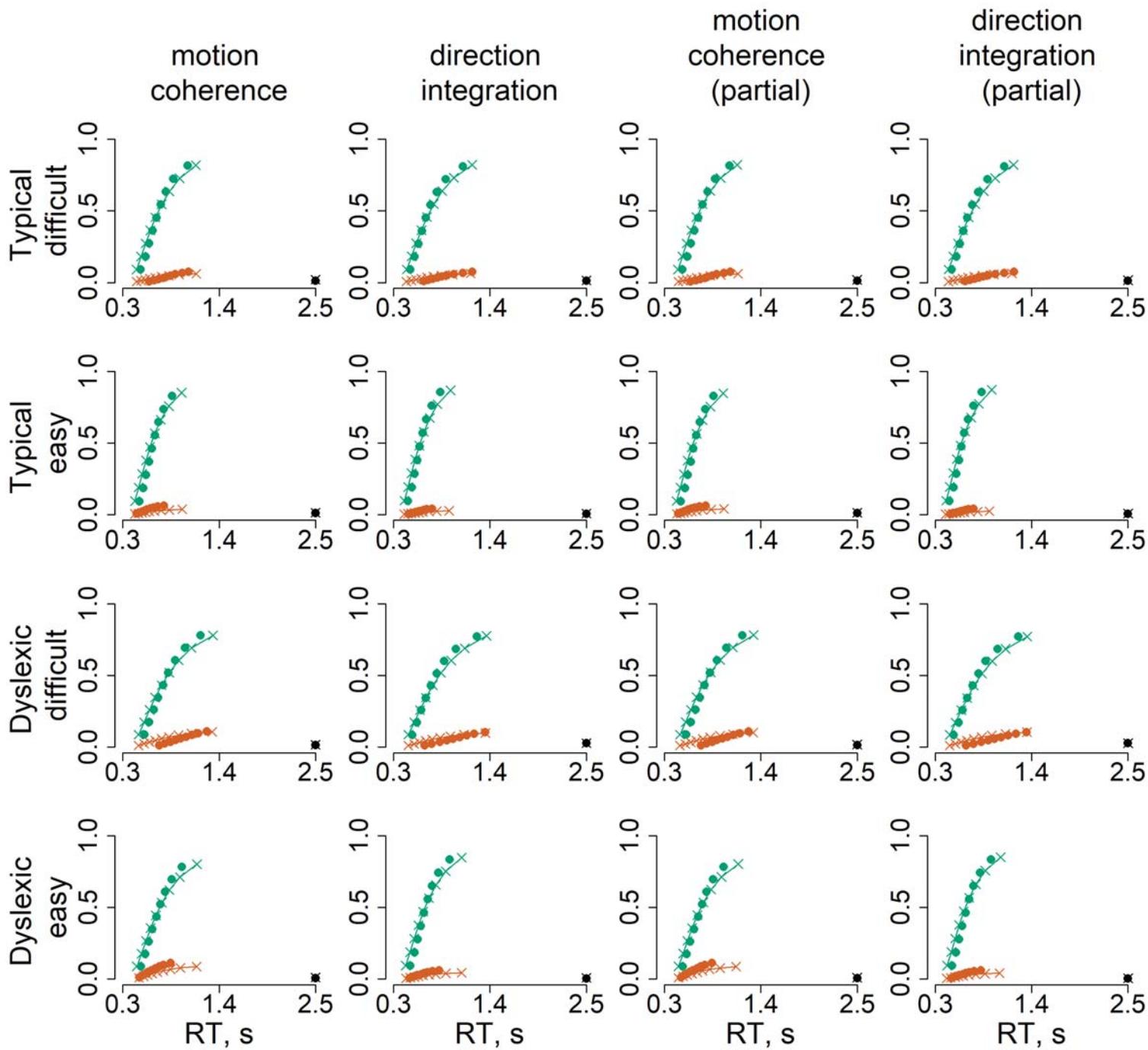


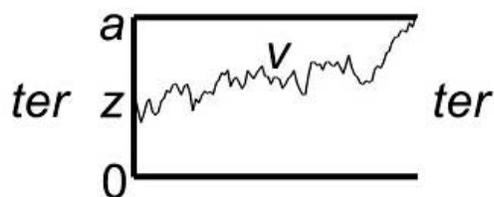
### motion coherence



### direction integration





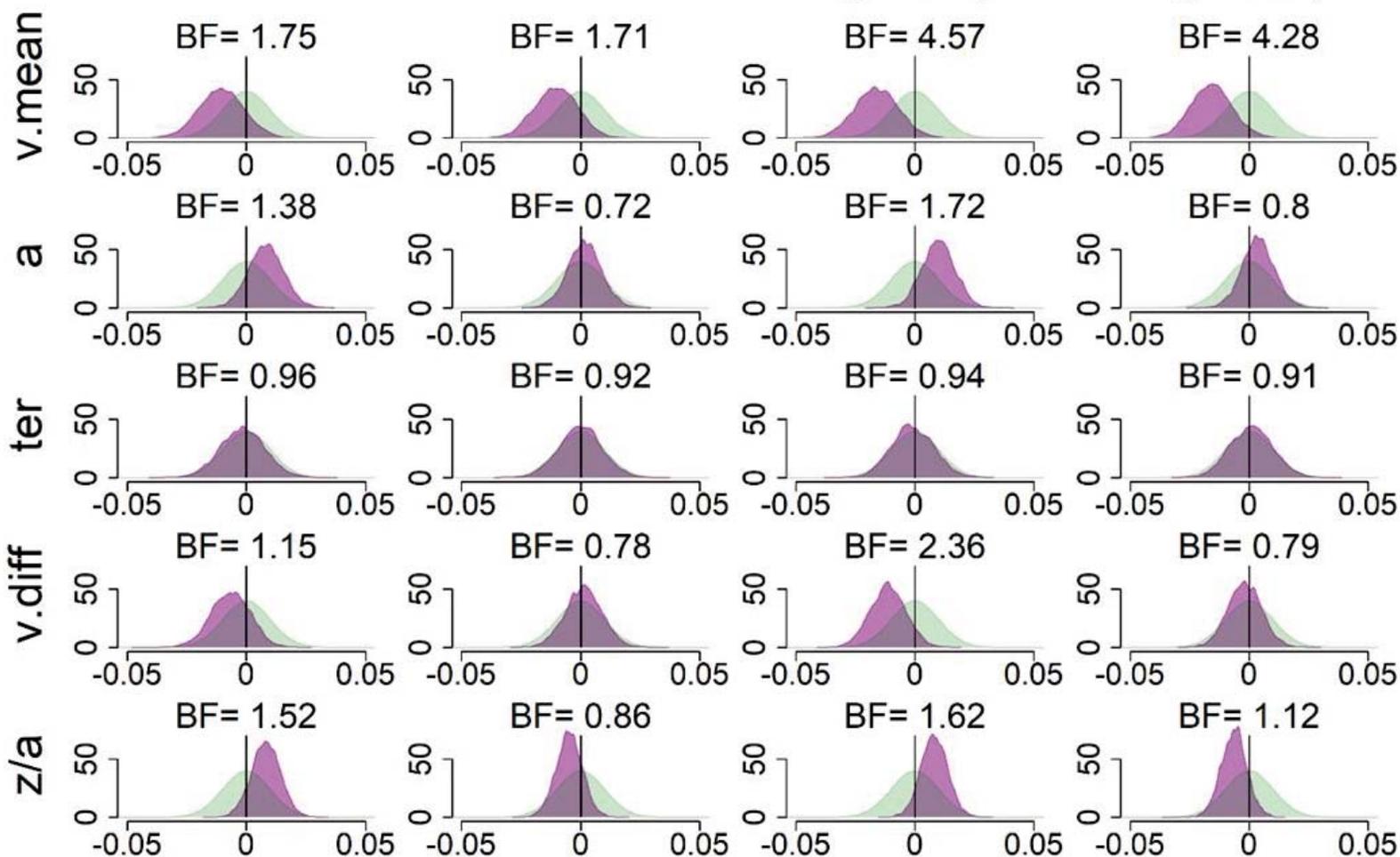


motion  
coherence

direction  
integration

motion  
coherence  
(partial)

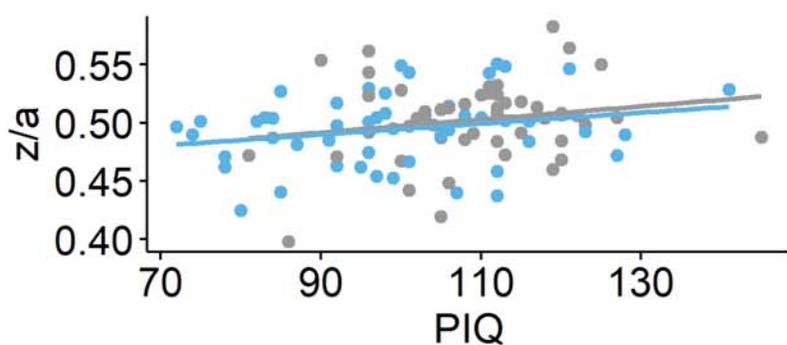
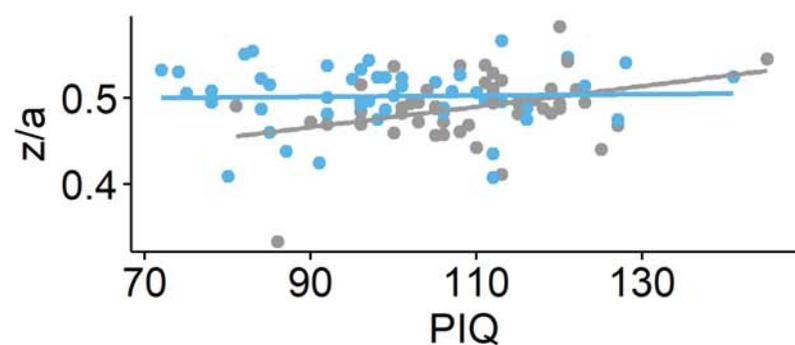
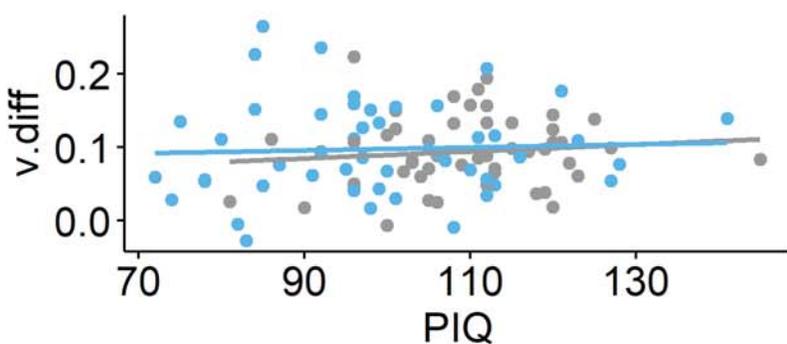
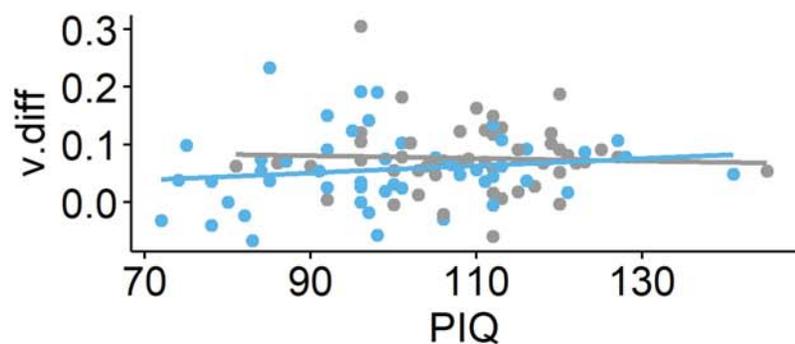
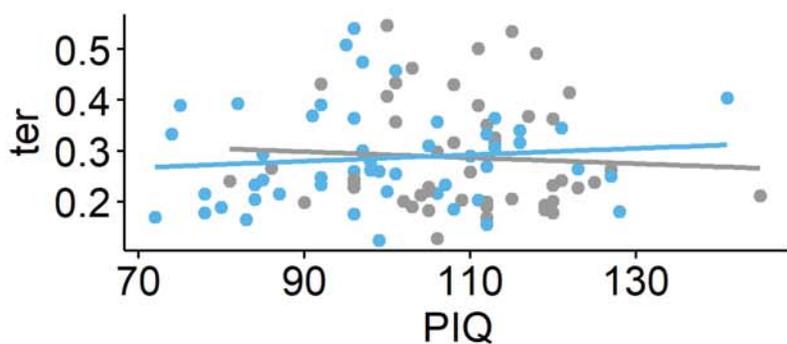
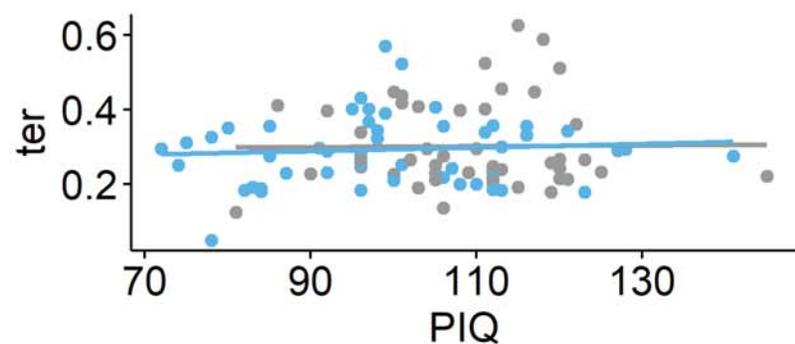
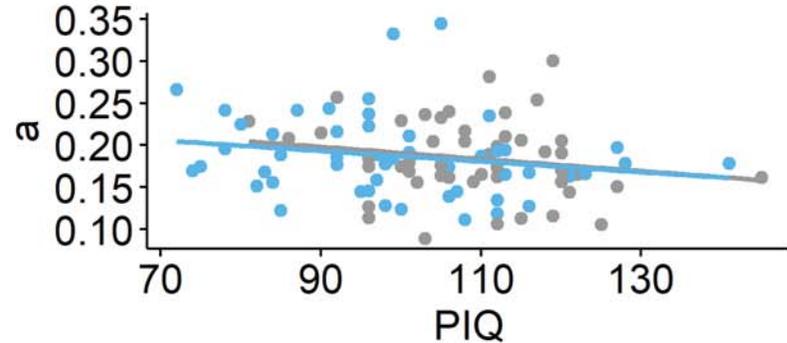
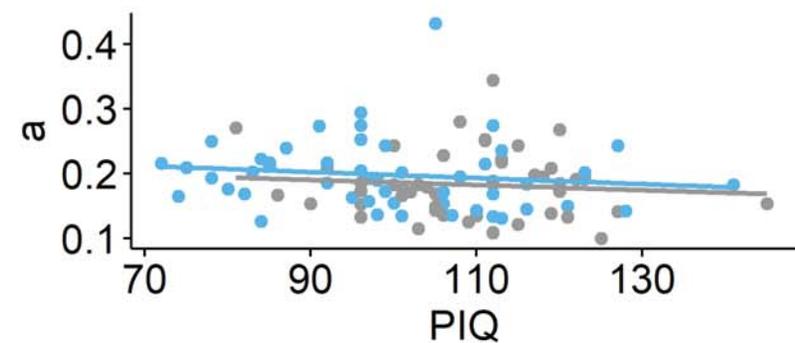
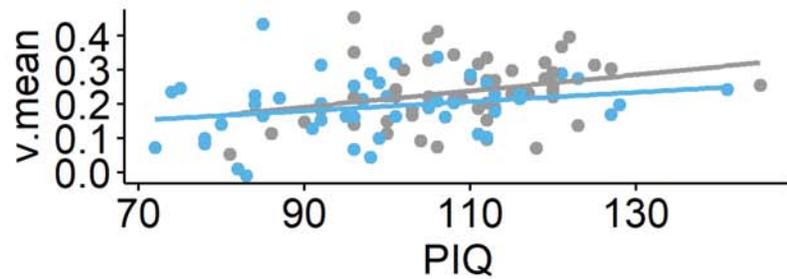
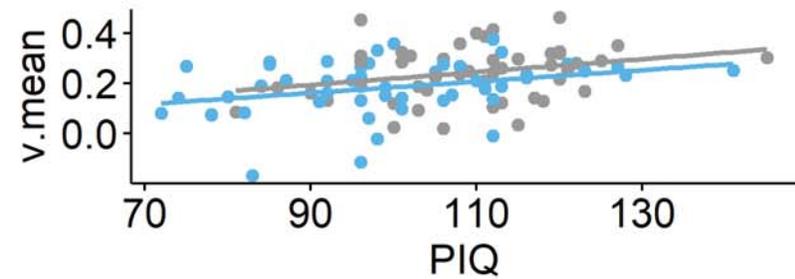
direction  
integration  
(partial)

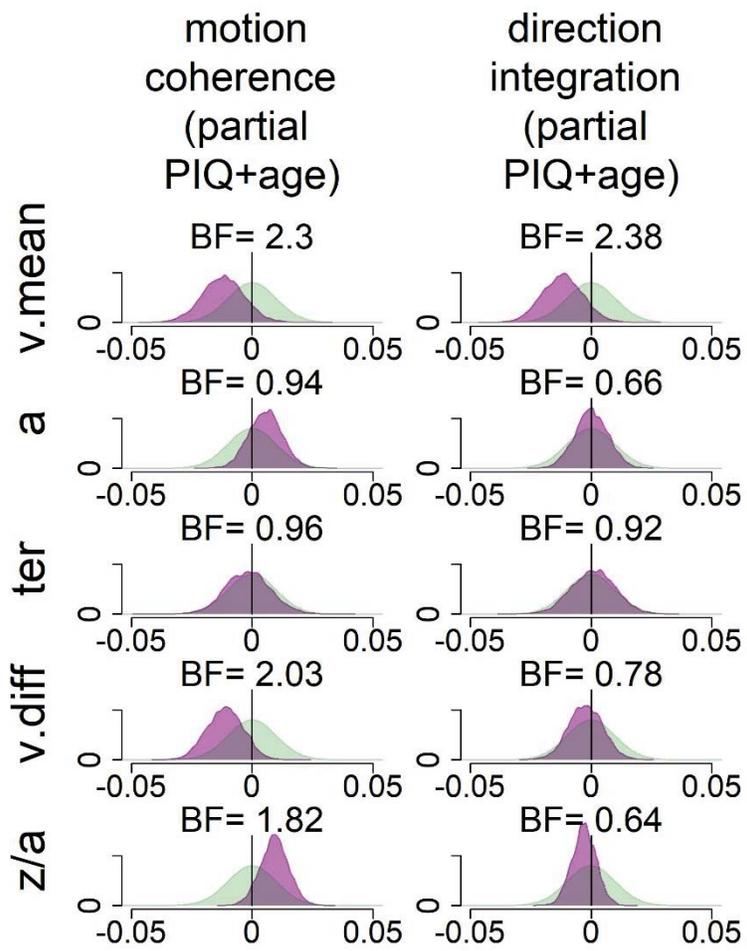


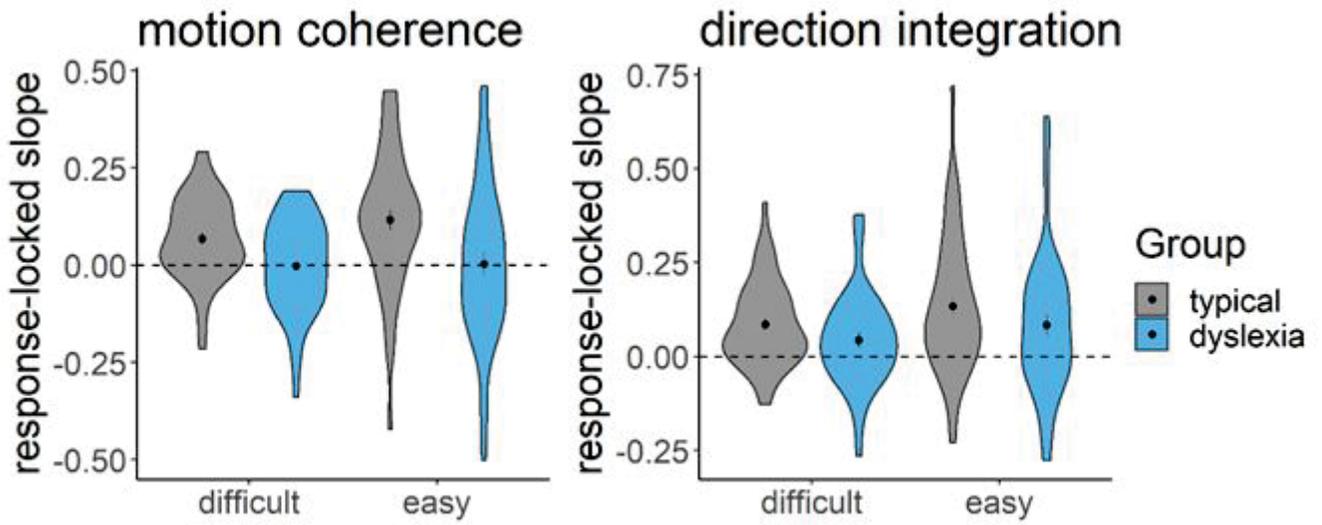
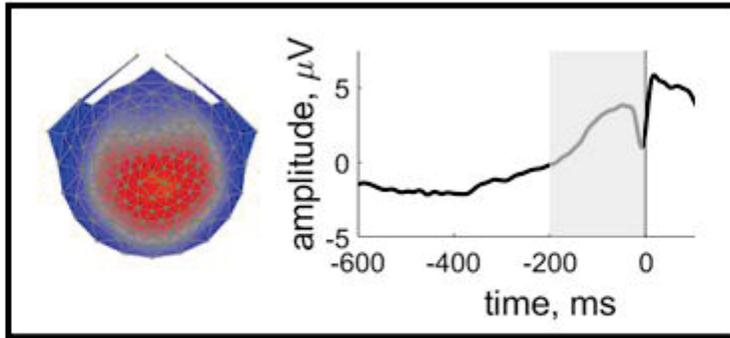
Group — typical — dyslexia

motion coherence

direction integration

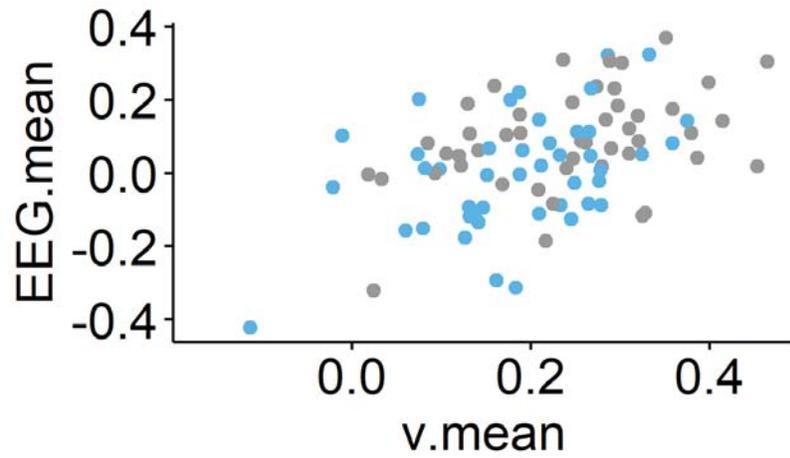




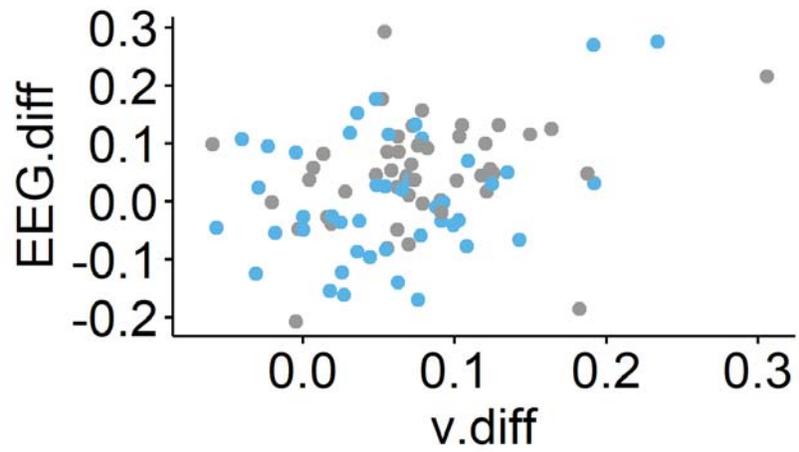


### motion coherence

Group • typical • dyslexia

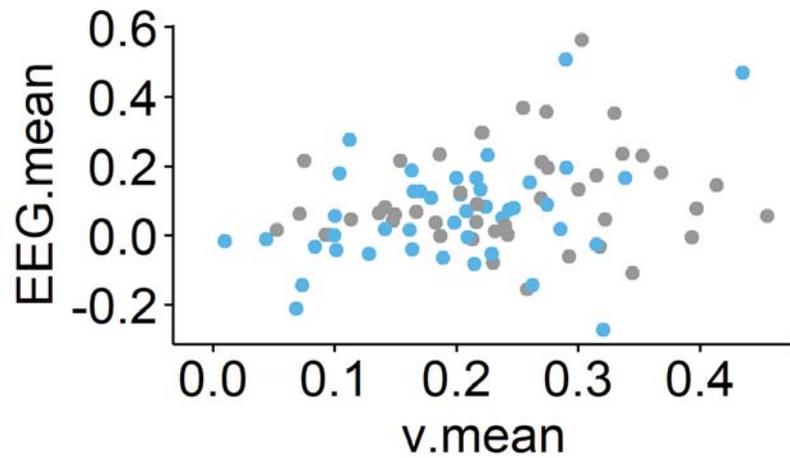


Group • typical • dyslexia

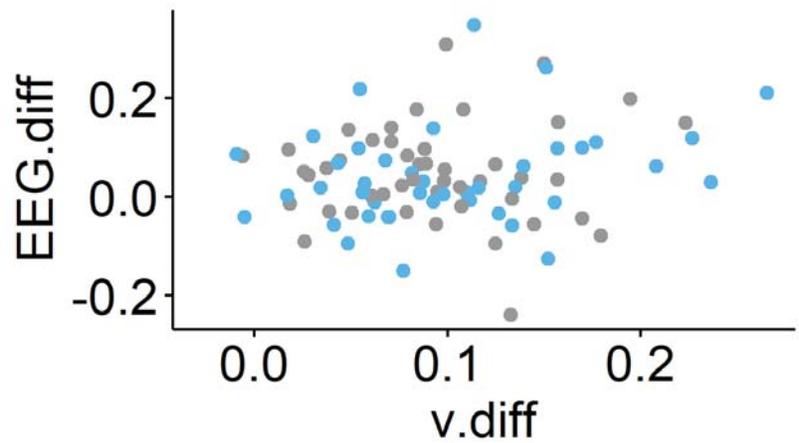


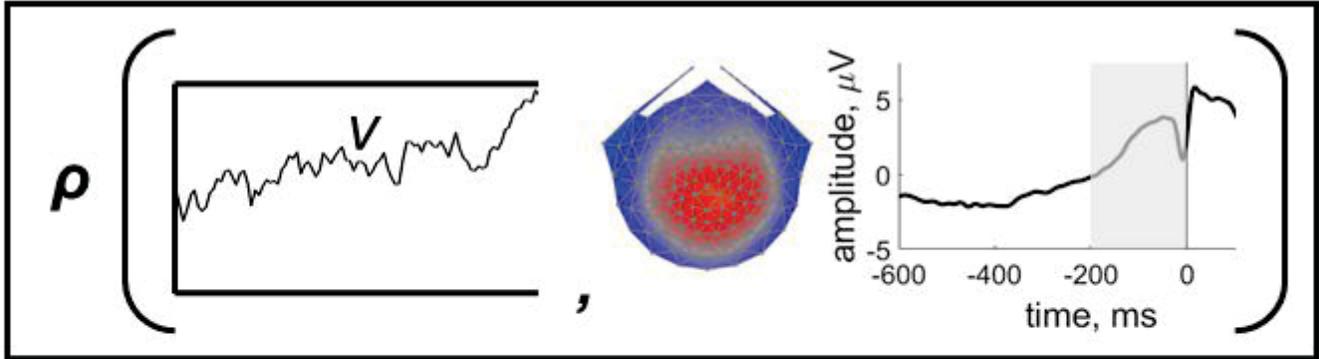
### direction integration

Group • typical • dyslexia



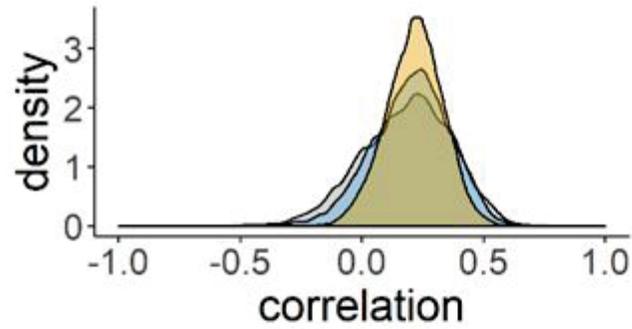
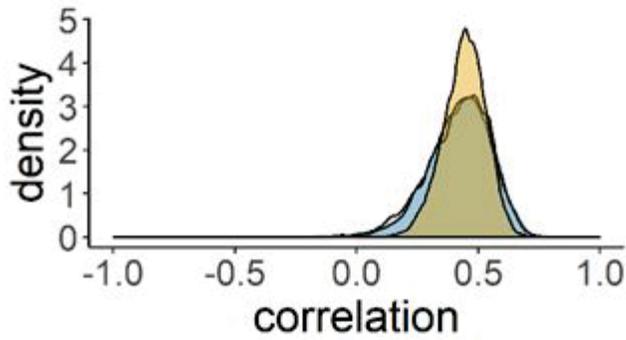
Group • typical • dyslexia





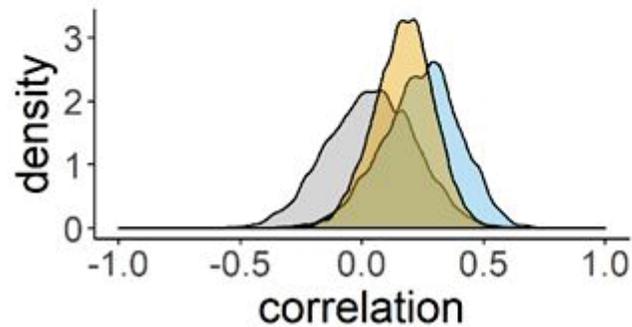
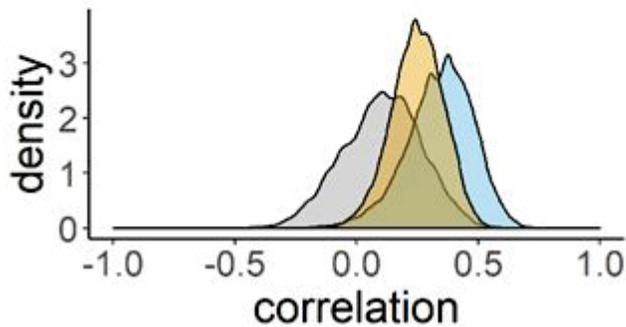
motion coherence  
mean

motion coherence  
difference



direction integration  
mean

direction integration  
difference



Group  
 typical  
 dyslexia  
 all

**Table 1. Demographics of participants included in final dataset**

	Typically developing (n = 50)	Dyslexia (n = 50)
Age	10.65 (2.34) 6.55 – 14.98	11.08 (1.87) 7.81 – 14.53
Performance IQ	109.26 (11.53) 81 – 145	99.40 (15.29) 72 – 141
Verbal IQ	110.60 (8.42) 95 – 127	98.56 (10.60) 77 – 118
Full-scale IQ	111.36 (9.02) 89 – 132	98.70 (12.85) 75 – 132
TOWRE-2 PDE	111.18 (16.53) 81 – 153	79.16 (9.45) 51 – 99
WIAT-Spelling	105.74 (10.21) 80 – 127	77.86 (7.96) 58 – 99
Composite score	108.46 (12.15) 89.5 – 138.0	78.51 (7.46) 54.5 – 89.0

Note. Data are presented as M (SD) Range.