Temporal event-structure coding in developmental dyslexia: evidence from explicit and implicit temporal processes

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As an alternative to theories positing visual or phonological deficits it has been suggested that the aetiology of dyslexia takes the form of a temporal processing deficit that may refer to impairment in the functional connectivity of the processes involved in reading. Here we investigated this idea in an experimental task designed to measure simultaneity thresholds. Fifteen children diagnosed with developmental dyslexia, alongside a matched sample of 13 normal readers undertook a series of threshold determination procedures designed to locate visual simultaneity thresholds and to assess the influence of subthreshold synchrony or asynchrony upon these thresholds. While there were no significant differences in simultaneity thresholds between dyslexic and normal readers, indicating no evidence of an altered perception, or temporal quantization of events, the dyslexic readers reported simultaneity significantly less frequently than normal readers, with the reduction largely attributable presentation of a subthreshold asynchrony. The results are discussed in terms of a whole systems approach to maintaining information processing integrity.

Keywords: *Dyslexia, event-structure coding, event simultaneity/asynchrony, visual perception*

Developmental dyslexia is typically revealed by large deviations in reading and spelling performance relative to age- and/or IQ-based norms, which cannot be explained by poor intellectual ability, inadequate instruction or problems such as poor eyesight. While originally considered a form of congenital word blindness, major symptoms of dyslexic language performance are difficulty in decoding and transforming language into the phonological code that relates letter units (graphemes) to speech sounds (phonemes) (Studdert-Kennedy & Mody, 1995). Dyslexia is also believed to present a general deficit in the accuracy and the segmental organization of the phonological representations of words in the mental lexicon (Swan & Goswami, 1997). Both ideas emphasize dyslexia as impairment at the level of phonological processing (e.g., Bradley & Bryant, 1978; Wagner, 1986; Stankovich, 1988).

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It has also been found that dyslexia is accompanied by sensory deficits that seem particularly marked during detection of rapidly changing stimuli (Lovegrove, Heddle, & Slaghuis, 1980; Reed, 1989; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; see also Farmer & Klein, 1995 for review). Auditory detection studies have shown that children with dyslexia are frequently less able than normal readers to distinguish either linguistic or non-linguistic stimuli that change rapidly in time (Tallal, 1980), including difficulties in phoneme discrimination (e.g. distinguishing /ba/ from /da/), when stimuli are presented in rapid succession (Tallal, 2004; Tallal & Piercy, 1974 although see Habib, 2000 for critical review). This has led to the suggestion that a general auditory deficit could underlie the difficulties dyslexic readers have with phonological decoding because phoneme discrimination often requires the detection of rapid and transient formant frequency changes (Tallal, 1980; Tallal, Millar, & Holly-Fitch, 1993; Masterton, Hazan, & Wijayatilake, 1995; Adlard & Hazan, 1998).

However, the exclusivity of a phonological explanation for dyslexia is brought into question by evidence pointing to impaired visual-transient channel function. Transient channels in vision originate in retinal systems sensitive to rapid contrast changes at low spatial frequencies and which project to magnocellular layers in the lateral geniculate nucleus (LGN) and from there to dorsal anterior brain regions, from visual to parietal cortex. A transient-channel disorder is an interpretation for dyslexia based upon observations that dyslexic subjects exhibit reduced contrast sensitivity for low spatial frequencies, increased visible persistence and a reduction in sensitivity to coherent motion (Lovegrove et al., 1980; Lovegrove, Martin, & Slaghuis, 1986; Slaghuis & Ryan, 1999). Indeed, the incidence of these disorders when considered alongside evidence for relatively normal sustained-channel performance (i.e. separate retinal system relatively insensitive to motion and which project through parvocellular layers of the LGN to ventral anterior regions of cortex, is very high, presenting in around 75% of dyslexic subjects (Slaghuis & Lovegrove, 1985).

If a functional link between transient channel, magnocellular and by extension dorsal visual-stream pathways is assumed, then the visual disorders experienced by dyslexic readers' concern both the sensory systems engaged in processing transient information as well as brain systems associated with focal attentional deployment. It has recently been suggested that the high incidence of visuo-spatial disorder in dyslexia is brought about as a specific consequence of disordered attentional mechanisms controlled by the dorsal visual stream (Vidyasagar & Pammer, 2009). Attentional deficits are argued to be core, impairing grapheme – phoneme conversion and, due to the high temporal sensitivity of the visual channels concerned, presenting as a general temporal processing deficit. Within this framework it is argued that apparently purely phonological impairments arising as a developmental consequence of impaired phonemic awareness. An alternative, but not mutually exclusive theory proposes dyslexia as a generalized temporal-processing deficit (Farmer & Klein, 1995; Habib, 2000; Lachmann, 2002; Stein & Walsh, 1997). Dyslexic performance is essentially multicausal in nature with reading impaired by faulty mechanisms in

any one of several auditory, visual or executive processes via the discoordination of those processes. Evidence in support of this theory may be inferred from difficulties in sequencing and temporal perception, reported not only with auditory but also with visual, tactile, and cross-modal sensory stimuli (Laasonen, Service, & Virsu, 2002). However that dyslexia may be symptomatic of a more generalised timing problem is suggested by performance on tasks that are not necessarily directly related to sensory coding. These include impaired duration judgements in dyslexic subjects (Walker, Shinn, Cranford, Givens, & Holbert, 2002) and rhythm reproduction, which requires at least the coordination of motor as well as auditory-perceptual mechanisms. In this case, the pre-schooler's ability to reproduce auditory rhythms has been found to be strongly predictive of later reading performance when performance on attentional and visuo-spatial tasks are accounted for by means of statistical partialling (Dellatolas, Watier, Le Normand, Lubart, & Cevrie-Muller, 2009).

The research described here seeks to examine evidence for a temporal processing deficit through two measures of visual event-structure coding: The first measure is the visual simultaneity threshold for repeating stimuli. The original research locates this threshold within the range 50-60 milliseconds (ms) (see Brecher, 1932), while more recent studies are corroborative in that thresholds following repeating visual stimuli are generally found between 50 – 60 ms (Elliott, Shi, & Sürer, 2007; Giersch, Lalanne, Corves, Seubert, Shi, Foucher, & Elliott, 2009; Schmidt, MacFarland, Ahmed, McDonald, & Elliott, 2009; Martyn, Antonijević, & Elliott, 2009). In fact, Martyn et al., using an adaptive version of the paradigm employed here, report the performance of samples of children with dyslexia and Specific Language Impairment (SLI) to be non-significantly different to that of normal readers. However in their experiment, which used method of limits as compared with the method of constant stimuli used in the other studies, thresholds for the three samples are located at ISIs almost double those reported previously (indicating the results to be sensitive to the experimental procedure), while the normal readers were taken for a sample of young adults, and were not matched to the reading impaired samples for age.

Unlike duration discrimination tasks, which are designed to refer to the operation of internal timing mechanisms, this simultaneity threshold corresponds to the interval of time over which scene dynamics are fused into a single perceptual event, i.e. a perceptual quantum in which events occurring within approximately 55 ms. will be judged to have occurred at the same time. Variations in this threshold in dyslexic relative to normal readers may be symptomatic of variations in information processing consequent upon disturbed reading dynamics in dyslexia.

The second measure employed examines the effect of a subthreshold synchrony signal on subsequent simultaneity judgment. Interest in this measure is premised on an established link between subthreshold synchrony and perceived simultaneity, for example, Elliott et al., (2007) showed that an increase in the frequency of simultaneity judgments followed the prior presentation of matching stimuli for subthreshold asynchronies in the range 0 – 15-21 milliseconds

(ms). The lower estimate of this range is close agreement with the inter-spike interval accompanying visual-cortical neuronal synchronization in response to coherent stimulus motion (see e.g. Gray, König, Engel, & Singer, 1989 for data and Singer, 1999 for review). Evidence for an altered relationship between subthreshold synchrony and the simultaneity judgments would be indicative of an altered relationship between oscillatory systems responsible for the binding dynamic sensory information and dyslexic performance. From this measure, corroboration might be inferred concerning the brain mechanisms concerned alongside summary hypotheses concerning the nature and integrity of the neural dynamics underlying temporal processing deficits in dyslexia.

Method

Participants: A total of 15 dyslexic participants (12 male, all normal or corrected-to-normal vision) completed the study, and were recruited from the Galway and Athlone dyslexic associations, and local national and secondary schools in Galway, Athlone, Ballinasloe and Moate. Thirteen control participants (10 male, all normal or corrected-to-normal vision) competed the study, recruited from local national and secondary schools in Galway and Athlone. The participant's ages ranged from 7 to 15 years, (mean age 11 years, 6.3 months) and groups were matched according to age and intelligence. For each participant, the level of general intelligence was measured by means of the Ravens Standard Progressive Matrices (Raven, 1958) and their reading age was measured by the blue and tan reading subtests of the Wide Range Achievement test (Jastak & Wilkinson, 1984). Both tests were administered on the same day as the experiment was undertaken. Each of the participants was found to have at least normal intelligence and no significant differences were found between the ages or intelligence of the groups (see Table 1) while dyslexic readers were found to be significantly poorer readers than the controls.

Table 1: Comparison of age, intelligence and reading ability
between normal and dyslexic readers

	Normal Readers	Dyslexic Readers	t	p
	X(min, max)	X(min, max)	t	p
Age	11(7,15)	12.28(7,15)	-1.714	.099
IQ	4,2(3,5)	4.2(3,6)	0.52	.929
Reading Ability	4.2(3,5)	2.7(2,3)	-6.54	.000

A diagnosis of dyslexia had been given to all members of the dyslexia group according to the discrepancy definition of the diagnostic manual ICD/ICD10 (World Health Organisation, 1992) by a professional educational or clinical psychologist between 1 and 6 years before the study. Such an assessment would typically include tests of reading and spelling for both contextualised and isolated letters and words, tests of phonological encoding ability, phoneme segmentation, phonological and visual recognition as well of tests of ability to learn mathematical procedures. The WISC III is also commonly used in such assessments to measure general intelligence and investigate discrepancies in the abilities the various subtests the WISC III measure. A diagnosis of dyslexia following such an assessment is legally recognised

by the Department of Education in the Republic of Ireland, and entitles dyslexic readers to additional educational assistance throughout their school years and allowances in state examinations. All dyslexic participants conformed to the definition of developmental dyslexia as opposed to acquired dyslexia and each participant produced the relevant documentation on the day of the experiment to ensure this. Finally all participants had normal or corrected to normal vision and received € (Euro) 15.00 for their participation. The dyslexic associations, the participant's teachers and parents gave their informed consent to the participation of the children in the present study while the children gave their assent and were informed that they could discontinue participation at any time during the experiment.

Apparatus and Stimuli: All stimuli were generated and the experimental procedure executed by means of custom software driving a Cambridge Research Systems ViSaGe graphics card, installed in an IBM compatible PC running Microsoft Windows XP. Stimuli were presented on a 21" monitor with the vertical refresh rate set to 100 Hz. The experimental programs were custom built in the visual C++ programming language. The target stimuli were two parallel, vertical gray bars which changed luminance on a black background. Luminance changes were Gaussian enveloped from a background of 0.02 cd/m^2 to a peak luminance of 12 cd/m^2 after 40 ms post-stimulus onset to reduce the effects of stimulus transients. The bars were separated 13° of visual angle at a viewing distance of 100 cm and at this viewing distance each bar subtended 3° x 10° of visual angle. Stimulus presentation occurred in an environment of low intensity ambient light (0.1 cd/m^2) to reduce the impact of onscreen persistence. The resolution of possible SOAs (stimulus onset asynchronies) was defined by the monitor refresh rate, which at 100 Hz was equivalent to a succession of frames of 10 ms duration.
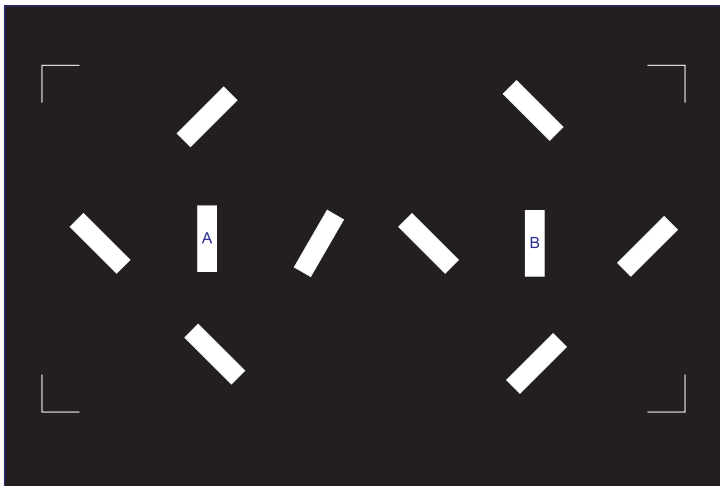

Figure 1: Schematic presentation display. Two central bars (here marked as 'A' and 'B' – there are no letters inside the bars in the experiment proper) change luminance amidst the changing luminance of each of 8 flankers, (bars rotated at either 45 or -45 degrees and surrounding the target bars). The flankers change luminance in sequence and the change in luminance of the central bars are embedded within this sequence such that the physical simultaneity/ asynchrony is non detectable. After the disappearance of the flankers the target bars increase luminance a second time with various target SOAs. Participants were asked to report whether they perceive this increase in luminance simultaneously across the two targets or whether the two targets increase in luminance asynchronously (Elliott et al, 2007).

In the main experiment there were two changes in luminance with the first masked by the rapid and sequenced onset of eight flankers (this paradigm is illustrated figure 1). The flankers were organized such that each of the targets had a 4-flanker mask and the flankers were positioned in a circle around the targets at horizontal angles of 0°, 90°, 180° and 270° and separated by 20 degrees of visual angle. Each flanker was the same size as the target but was oriented pseudo-randomly at either 45° or -45° relative to the horizontal meridian.

Design and Procedure: The experimental protocol was approved by an ad hoc ethics committee of the School of Psychology, the National University of Ireland Galway. The experiment was carried out in two parts for each participant. The first part comprised two pilot experiments that were intended to rapidly determine the lower and upper simultaneity thresholds for simple luminance changes of the two target bars. On each trial, participants were asked to indicate whether, in their opinion the target bars had changed luminance at the same time (simultaneously) or at a different time (i.e. with an asynchrony). Simultaneity thresholds were expected to be found at stimulus-onset asynchronies (SOA) of greater than 0 ms for their determination the stochastic approximation adaptive procedure was employed as described by Treutwein, (1995). Using this method, the adaptive procedure started with an initial asynchrony 40 ms and, following response, a new target-target asynchrony was calculated on a trial by trial basis using the following formula:

$$SOA_{n+1} = SOA_n \pm \frac{C}{T_n}$$

where C was a constant set to 20 ms and T_n was the number of reversals (i.e. the number of changes in response in the response sequence). When SOA reduced to below a single frame i.e. < 10 ms, or when the number of reversals was greater than 6 the adaptive procedure stopped and the final SOA was taken as the estimated threshold.

The upper simultaneity thresholds were determined by embedding the target bars in a sequence of on- and off-setting flankers (see Figure 1). The lower simultaneity thresholds were determined in the absence of flankers. Thus, the lower simultaneity threshold was determined in absence of perceptual noise. It was assumed that this was in fact the 'lower asynchrony threshold' because below this SOA two stimuli would always be seen as appearing simultaneously. The addition of flankers was expected to interfere with simultaneity perception and thus increase the simultaneity threshold. By adding flankers it was thus possible to determine an ,upper simultaneity threshold', under which it might be assumed the asynchrony of the two target bars could not be reliably reported.

These procedures were undertaken on a participant by participant basis in order to calculate individual asynchrony ranges (from lower to upper thresholds) to be used in the main experiment. Participants were asked to avoid eye movements or blinks during experimental trials and to make their response using a two key keypad as accurately as possible.

The associated lower and upper threshold values then determined the range of subthreshold asynchronies that were employed in the main experiment. The main experiment aimed to measure simultaneity threshold to two target bars changing luminance after presentation of the flanker/target bar sequence. The major modification in the main experiment was that the two target bars changed luminance twice: the first change (in fact the onset of the target bars) occurred within the matrix of rapidly onsetting flankers and this change occurred either synchronously (SB$_S$)

or with an asynchrony (SB$_A$) between the two target bars set at a pseudo-randomly determined value between the upper and lower simultaneity thresholds (previously determined by the adaptive procedures described above). Given this modification, it was assumed that, at onset, the temporal relationship between target bars was not detectable by observers. Flankers remained on screen for 80 ms, increasing and then decreasing in luminance nonlinearly over 40 ms intervals. Targets were presented as the 6th (combined) or the 6th and 7th stimuli in the target – flanker sequence for the SB$_S$ and SB$_A$ conditions respectively.
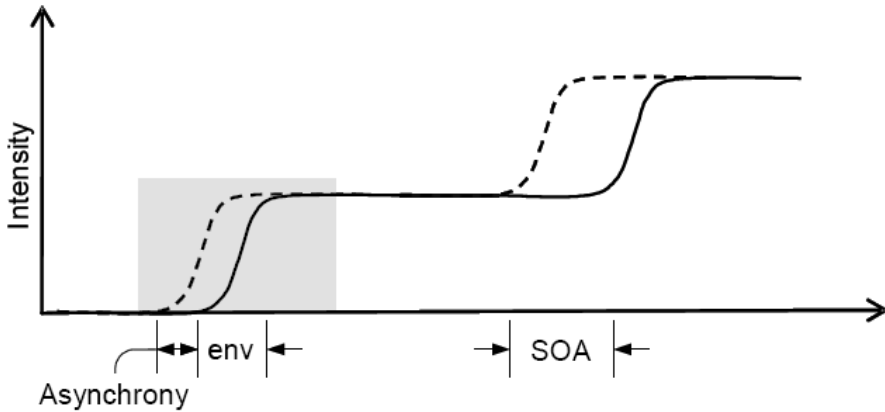

Figure 2: Luminance change as a function of time the dashed and solid lines represent the luminance of two target bars. In order to avoid an abrupt increase in luminance, which would encourage stimulus transients and possibly the engagement of attentional resources to the first of the two changing targets the luminance increases were enveloped by a half Gaussian function of 80 ms duration. The Grey region represents the period of time during which the dynamic presentation of flankers masked the first change in target luminance. During this interval the subthreshold luminance increases occurred either simultaneously or within a range of lower-upper asynchrony thresholds determined during the first part of the experiment. After the disappearance of the flankers the target bars remained on screen with same luminance for 150 ms which was then increased a second time with various target SOAs. Participants were asked to report whether they perceive this increase in luminance simultaneously across the two targets or whether the two targets increase luminance one after the other. Although the luminance of the targets increased twice during the experimental trial, due to the masking effects of the flankers only the second increase was perceived by the participants.

Exactly 150 ms after SB$_S$ onset or after onset of the second of the two target bars in the SB$_A$ condition, the two target bars increased in luminance although on this occasion no flankers were present. This luminance change took place at each of a set of SOAs that were equally distributed in 10 ms intervals over the range 0 to 110ms. After the second increase in luminance the targets bars were displayed for two seconds and participants were asked to judge whether the bars had changed luminance simultaneously or with an asynchrony (the full procedure is illustrated schematically in Figure 2). The second part of the experiment consisted of 10 blocks comprising 60 trials per block and the experimental conditions were pseudo-randomly ordered separately for each participant to control for possible order effects.

Results

For all participants the lower simultaneity thresholds were reliably estimated at lower SOAs than the upper simultaneity thresholds. For the dyslexic readers, the range of lower simultaneity thresholds ranged from 10 ms to 70 ms (mean: 38 ms [standard deviation = 18 ms]) the upper simultaneity thresholds ranged from 50 ms to 160 ms (mean: 92 ms [SD 39 ms]). For the control participants, the range of lower simultaneity thresholds extended from 20 ms to 80 ms (mean: 40 ms [SD 18 ms]) and the upper simultaneity thresholds extended from 40 ms to 190 ms (mean: 85 ms [SD 55 ms]). Independent-samples t-tests showed that the difference for both lower and higher thresholds was non significant between the two groups [$t(25)$ = -.227, **Cohen's d** = -0.098 and $t(25)$ = .334, **Cohen's d** = 0.04583, respectively].

In the main experiment, psychometric functions (PFs) were calculated individually using a least squares method and thresholds were calculated individually by a method of interpolation. In contrast to previous studies in which a correction was applied to correct for bias, i.e. a score of less than 100% when bar SOA was 0 ms, the presence of bias in this experiment (illustrated in Figures 3 & 4) was considered of potential theoretical interest and so the uncorrected data were analysed.
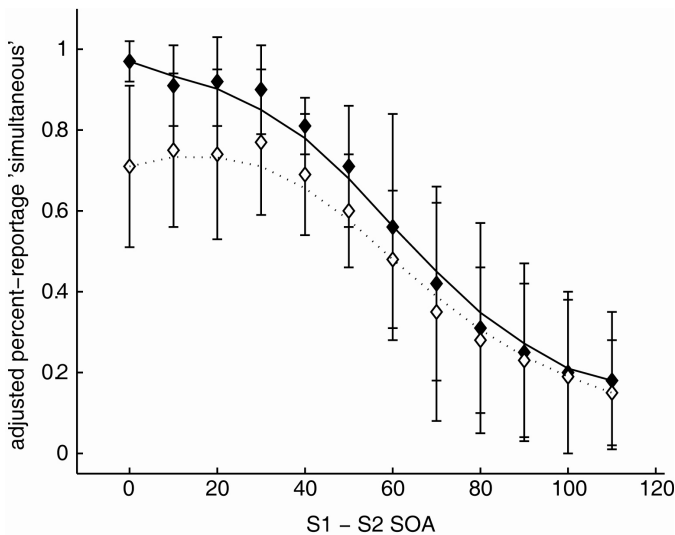

Figure 3. The mean psychometric function for simultaneity judgments for normal readers. The y axis represents percentage of simultaneity reportage while the x axis represents stimulus onset asynchronies between the two target bars in milliseconds. Filled symbols denote SB_S while unfilled SB_A (see text) Error bars are standard deviations. The functions are smooths calculated form the data using a least-squares method.

The mean simultaneity threshold for dyslexic children following exposure to SB$_S$ was located at 62 ms (SD 11 ms) and following exposure of SB$_A$ at 58 ms (SD 26 ms). These thresholds compare with mean thresholds for normal readers which were located at 69 ms following exposure to SB$_S$ (SD 19 ms) and 57 ms following exposure to SB$_A$ (SD 18 ms). Thus and quite in contrast to our expectation thresholds were slightly higher for normal readers although independent-samples t-tests showed that there was no significant differences between the thresholds in either SB$_S$ or SB$_A$ conditions or between the two groups [$t(25)$ = 2.578, **Cohen's d** = 0.9512, and $t(25)$ = .113, **Cohen's d** = 0.0452, respectively].
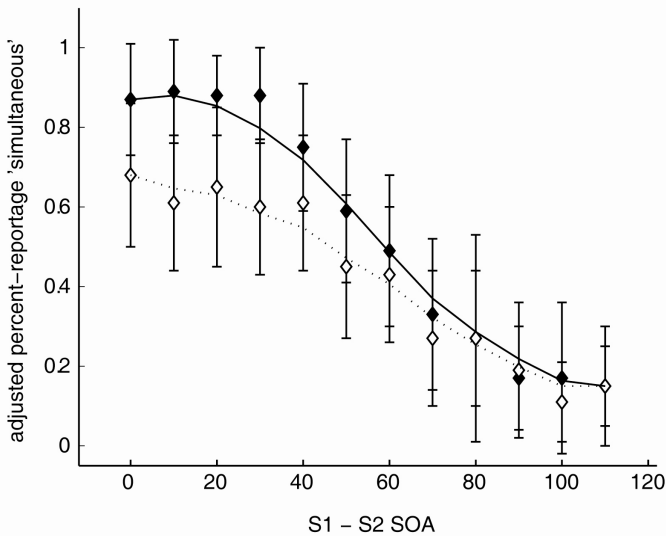

Figure 4. The mean psychometric function for simultaneity judgments for dyslexic readers. The y axis represents percentage of simultaneity reportage while the x axis represents stimulus onset asynchronies between the two target bars in milliseconds. Filled symbols denote SB$_S$ while unfilled SB$_A$ (see text) Error bars are standard deviations. The functions are smooths calculated form the data using a least-squares method.

Elliott et al. (2007) found simultaneity reports were more frequent for SB$_S$ relative to SB$_A$ across a small range of SOAs from 0 – (15 – 21) ms. We sought here to examine whether this finding repeated in the present data, i.e. whether there were any significant differences between the two groups in the number of simultaneity reports following SB$_S$ relative to SB$_A$ presentations. Figure 3 reveals the mean PFs separately for SB$_S$ and SB$_A$ produced by the dyslexic participants; Figure 4 shows the normal readers mean PF. As reported by Elliott et al., differences between the effects of SB$_S$ and SB$_A$ appear below

simultaneity thresholds for both groups with a relative reduction in the frequency of simultaneity reportage for the dyslexic participants (i.e. the potential effect of a report bias). To substantiate any differences between conditions and across groups, the data were examined by means of a univariate three-way analysis of variance (ANOVA). This analysis included the factors Group (dyslexic and normal readers), Subthreshold Synchrony Condition (SSC: SB_S and SB_A) and SOA with 12 levels, (0 ms, 10 ms, 20 ms, 30 ms, 40 ms, 50 ms, 60 ms, 70 ms, 80 ms, 90 ms, 100 ms, 110 ms).
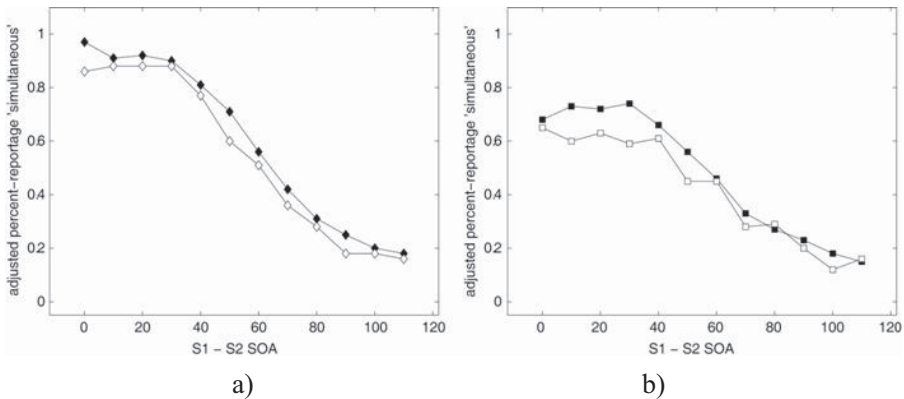

Figure 5. Filled symbols represent normal readers and unfilled represent the percentage reportage simultaneity for dyslexic readers following (a) SB_S and (b) SB_A respectively

There was a statistically significant main effect for group ($F(1, 600) =$ 15.199, $p < .01$, $\eta_p^2 = .025$ arising because dyslexic readers reported simultaneity overall less frequently than the normal readers which was more pronounced at shorter SOAs (see Figure 5) while at 0 ms SOA, dyslexic readers reported simultaneity 86% of the time following SB_S and 67% of the time following SB_A, while normal readers reported simultaneity 97% of the time following SB_S and 72% of the time following SB_A. This result is indicative of a stronger bias against simultaneity reportage in the dyslexic group. However, strong confirmation of this hypothesis was not provided by virtue of non-significant group x subthreshold synchrony or three-way interactions. Inspection of Figure 5 reveals a further characteristic peculiar to the SB_A condition: in contrast to the two SB_S conditions, for which the PFs and slopes appear almost identical (-.0107 vs. -.0118 for normal and dyslexic readers, respectively)[1] the two SB_A conditions also exhibit very similar slopes (-.006 vs. -.0079), but with PFs differing in one key characteristic: the critical SOA at which the PFs begin to decrease with

1   Slopes were averaged from individual calculations taken from smoothed PFs and where possible between interpolated values for 25% and 75% simultaneity reportage, or alternatively taken the highest or lowest report percentage when either 75% and/or 25% reportage was not achieved. Slopes were examined by means of an ANOVA which revealed no differences between groups for either SBS or SBA conditions.

increasing SOA. In the case of normal readers this decrease begins at around 30 ms while for dyslexic readers at 40 ms. Note that the difference of around 10 ms is sufficient to render the slope of the two PFs in closer proximity than would be the case if the dyslexic reader's PF began to decrease at the same SOA as that of the normal readers.

The ANOVA also revealed the expected significant main effect of SOA, $(F(11, 600) = 123.15, p < .01, \eta_p^2 = .693)$, reflecting the negative trajectory of the PF (i.e. simultaneity reportage decreased in frequency with increasing SOA). There was also a statistically significant SSC main effect $(F(1, 600) = 82.431 \; p < .01, \eta_p^2 = .121)$. This was because both groups reported simultaneity more frequently after the prior presentation of SB_S relative to SB_A. A significant SSC x SOA interaction $(F(11, 600) = 4.058, p < .01, \eta_p^2 = .062)$ was explored by means of simple-main effects analyses of SB_S and SB_A at each level of SOA: significant or borderline-significant differences between the frequency of SB_S and SB_A reportage were found for SOAs up to 50 ms which were not found for SOAs of 60 – 110 ms, (significant difference were: $F(1, 624) = 23.233, p < .01$; $F(1, 624) = 21.875, p < .01$; $F(1, 624) = 19.803, p < .01$; $F(1, 624) = 19.803, p < .01$; $F(1, 624) = 8.350, p < .05$ and $F(1, 624) = 7.593, p = .06$ for SOAs of 0 ms, 10 ms, 20 ms, 30 ms, 40 ms, and 50 ms respectively). It is interesting to note that simultaneity reportage significantly differed in frequency following the presentation of SB_S relative to SB_A up until an SOA that might be assumed to lay close to the lower boundary of the simultaneity threshold. This suggests the threshold is related to the shape and distribution of the PFs, which are in turn symptomatic of the performance of event-structure-coding in mechanisms earlier than those rendering a directly reportable experience.

DISCUSSION

These results indicate that it is not variation in the location of the threshold across groups (which is non-significantly different), but instead the consistency of that threshold that is important in this study. We believe that in dyslexia, thresholds may be maintained within the 50 – 60 ms range – resulting in a bias against simultaneity reportage – to allow perceptual quantization, and the judgment of event-structures, to be occur in as normal a fashion as possible. We propose that this happens by virtue of compensation within early binding mechanisms and that this proposal is strongly supported both by differences in distribution of PFs between the SB_S and SB_A conditions and the very close convergence of these functions at the threshold.

We propose this for two reasons: firstly, because in this study, a similar but stronger bias was observed to that reported by Elliott et al., (2007). In that study, Elliott et al. found that SB_A biases simultaneity judgments over SOAs of between 0 – 15-21 ms: a narrow range of critical SOAs that has been assumed to relate to temporal quantization at the neural level, e.g. in neural synchronization

and the binding of visuo-spatial material. By contrast, in the present study dyslexic participants were biased against a simultaneity report when previously presented with SB_A over SOAs extending to 50 or 60 ms (see Figure 4). This would suggest that the distribution of PFs indicate a difference between dyslexic and normal readers in the way in which event structures are coded in early visual mechanisms. A paradigmatic assumption is of these mechanisms as below threshold and so not directly contributive to conscious perception and report. We arrive at the conclusion that these are early visual and binding-related partly on the basis of the research of He, Cavanagh, & Intriligator (1996) who showed that the outcomes of processes as late as visual cortex are not available for conscious report. However, whether the differences in distribution are characteristic of a distribution in the timing of those processes, or in the amount of neural activity recruited by the process from the mechanism concerned, or both, is not clear, indicating that investigation of the neural correlates with these findings is one issue for future, brain oriented research.

Our proposal is thus of a compensation between early binding and later temporal quantization (i.e. perceptual integration over time), based upon the idea that the interval over which perceptual quantization occurs (i.e. that interval maintained below the simultaneity threshold) is maintained at the same levels in dyslexic as in normal readers by broadening the resolution and thereby reducing the efficiency of binding processes. This would provide an explanation for the apparent bias against simultaneity reportage following SB_A presentation (as well as the general bias) as lack of resolution in binding is likely to result in systems noise that may impair the efficiency of subsequent integration processes and lead to perceptual noise.

Our explanation is entirely consistent with the idea that simultaneity judgments are brought about by compensations within a multicausal processing architecture. As such, this explanation identifies separate mechanisms for binding and temporal quantization and that dyslexia represents a fundamental impairment in temporal quantization that is compensated for by a lack of resolution in binding. This does seem counter intuitive for at least two reasons; it runs counter to our hypotheses and is an interpretation indirectly related to the data. However it must be stressed that the costs associated with this strategy may be relatively low given the similar trend in normal readers (although see more restricted differences over subthreshold SOAs in the data of Schmidt et al., 2009), while the cost associated with leaving quantization relatively unconstrained may be high, particularly given schizophrenia, a very serious disorder, in which simultaneity thresholds are increased very significantly relative to healthy controls (Giersch et al., 2009; Schmidt et al., 2009).

We conclude that the normal relationship between early binding processes and perceptual quantization in time (i.e. the integration of events into conscious or at least reportable episodes) is maintained in dyslexia by a form of compensation in which the processes mediating binding lose fidelity and as a consequence

temporal quantization maintains over intervals like those recorded in normal readers. This conclusion emphasizes a facultative interaction of processes across a processing hierarchy and as such, it is strongly supportive of the notion of dyslexia as a multi-causal, functional connectivity disorder as proposed by Lachmann (2002).

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