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Visual and auditory attentional capture are both sluggish in children with developmental dyslexia

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Abstract. Automatic multimodal spatial attention was studied in 12 dyslexic children (SRD), 18 chronological age matched (CA) and 9 reading level matched (RL) normally reading children by measuring reaction times (RTs) to lateralized visual and auditory stimuli in cued detection tasks. The results show a slower time course of focused multimodal attention (FMA) in SRD children than in both CA and RL controls. Specifically, no cueing effect (i.e., RTs difference between cued–uncued) was found in SRD children at 100 ms cue-target delay, while it was present at 250 ms cue-target delay. In contrast, in both CA and RL controls, a cueing effect was found at the shorter cue-target delay but it disappeared at the longer cue-target delay, as predicted by theories of automatic capture of attention. Our results suggest that FMA may be crucial for learning to read, and we propose a possible causal explanation of how a FMA deficit leads to specific reading disability, suggesting that sluggish FMA in dyslexic children could be caused by a specific parietal dysfunction.

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INTRODUCTION

Dyslexia or specific reading disability (SRD) is often defined as a deficit in reading and spelling despite adequate intelligence and access to conventional instruction (American Psychiatric Association 1994). Although there are a wide variety of theories which attempt to account for SRD, two general approaches have received particular interest.

The first approach posits that SRD as well as specific language impairment (SLI) arise from deficits in systems that are linguistic in nature. Specifically, the phonological deficit theory suggests that SRD arises from deficits in phonological processing and memory (e.g., Goswami 2000, Ramus 2003, Snowling 2000, for a review of normal language neuroimaging see Heim 2005, this issue).

On the other hand, a second theory claims that deficits in underlying non-linguistic sensory mechanisms are the real core deficits in SRD (e.g., Stein and Walsh 1997 for visual deficits; Wright et al. 2000 for auditory deficit). This theory, known as the magnocellular (M) theory of dyslexia, is an exhaustive, albeit controversial (e.g., Skottun 2000) account, starting from the observation that many reading disabled children are impaired in the specific visual abilities that utilize the M pathway (e.g., Stein and Walsh 1997). The multimodal (i.e., visual and auditory) version of the M theory, called the "temporal processing hypothesis", suggests that children with SRD (and also children with SLI) have specific deficits in processing rapidly presented or brief sensory stimuli within the visual and auditory domains (for a review see Farmer and Klein 1995). Of course, linguistic and sensory deficits are not mutually exclusive. More importantly, the M hypothesis explicitly claims that phonological deficits in SRD children arise from visual and auditory impairments, which in turn lead to the language disorder.

However, a recent study by Amitay and coauthors (2002), investigating both visual and auditory M functions (i.e., flicker detection, detection of drifting gratings at low spatial frequencies, speed discrimination and detection of coherent dot motion), showed that "pure" M deficits were present only in six out of 30 SRD adults. In addition, disabled readers showed "impaired performance in both visual and auditory non-M tasks requiring fine frequency discriminations. The stimuli used in these tasks were neither modulated in time nor briefly presented" (Amitay et al. 2002, p. 2272). Thus, the authors concluded that dyslexics have a generally inefficient multimodal processing of perceptual stimuli.

To explain the multimodal perceptual deficit in SRD (and SLI) children, Hartley and Moore (2002) presented a model based on processing efficiency. The model suggests that masking effects (i.e., spatial and temporal signal interference induced by "near" noise) can be better explained by a "processing efficiency hypothesis" rather than a temporal processing hypothesis. Processing efficiency refers to all factors, aside from temporal and spectral resolution, that affect the ability to detect visual and acoustic signals in noise (i.e., threshold signal-to-noise ratio).

We suggest that focused spatial attention (FSA) is one crucial factor affecting multimodal perceptual processing efficiency. Despite the great amount of information flooding the scenes, we are able to focus attention on one spatial location (or/and object) and to process the relevant information. The major effect on perceptual functions is that FSA appears to enhance the neural representation of the attended stimuli. This signal enhancement manifests itself in a variety of ways, including faster reaction times (RTs), improved sensitivity (thresholds), as well as reduced interactions with flanking stimuli. FSA allows decisions to be based on the selected stimulus alone and thus any distracting stimuli which may be present can be disregarded (e.g., Braun 2002, Carrasco and McElree 2001). On the basis of these perceptual effects, FSA influences the contents of all the post-perceptual processes such as short-term memory, perceptual decisions and voluntary responses.

In fact, sluggish FSA (i.e., prolongation of input chunks) appears to be crucial not only for a variety of subtle sensory and motor deficits but also for specifically impaired reading skills in dyslexic subjects. "Sluggish attentional shifting" (SAS) in SRD children and adults can account for the generally inefficient multimodal processing of perceptual stimuli (Hari and Renvall 2001, Facoetti et al. 2003b). Accordingly, several studies have shown deficits in both visual and auditory shifting and focusing of attention in SRD subjects.

Brannan and Williams (1987) demonstrated that, compared to subjects reading normally, poor readers were not able to rapidly focus visual attention. A series of studies conducted by our group has shown sluggish and asymmetric focusing in dyslexic children, which affects automatic control of visual attention (e.g., Facoetti et al. 2000b, 2001, 2003a). In addition, the visual attentional blink (i.e., transient blindness to the second

target in a dual task consisting of two targets) was longer in dyslexic than in normally reading adults (Hari et al. 1999). Both a temporal order judgment between visual hemifields and a line motion illusion task were applied to test whether dyslexics have difficulties in their automatic attentional capture. Dyslexics showed slower processing in the left than in the right visual hemifield (i.e., asymmetric distribution of attention); moreover, their attentional capture was sluggish in both hemifields (Hari et al. 2001). Despite this evidence, the issue of whether visual attention deficits are causally linked to reading disorders in dyslexic children is still hotly debated (for a recent review see Ramus 2003).

Evidence for an auditory spatial attentional deficit in SRD subjects was initially provided by Asbjornsen and Bryden (1998). Deficits in dyslexia often manifest themselves in the auditory modality with problems in speech-sound perception (phoneme discrimination) in the presence of background noise (e.g., Cunningham et al. 2001). SRD children also have difficulties in discriminating between acoustically similar sounds (e.g., Tallal 1980) and in processing rapid sound sequences (e.g., Helenius et al. 1999). These auditory perception deficits are likely related to an inability to rapidly shift and focus auditory attention in order to properly discriminate the features of the sound (Renvall and Hari 2002). In fact, several studies conducted on non-impaired individuals demonstrated that phoneme identification may be substantially increased when auditory spatial attention is focused (e.g., Mondor and Bryden 1991) providing strong evidence that selective spatial attention may act to facilitate auditory perception.

Thus the generally inefficient multimodal perceptual processing in SRD subjects could be due to a loss in the temporal resolution of spatial attention to transient events (Facoetti et al. 2003b).

Nonetheless, direct evidence of both visual and auditory (i.e., multimodal) SAS in the same sample of SRD subjects has not yet been reported in the literature.

In a previous study we investigated the automatic focusing of visual and auditory attention in the same SRD children. The results showed both visual and auditory deficits in the automatic focusing of spatial attention in the SRD children (Facoetti et al. 2003b). However, the two different cue-target delays used in the visual (i.e., 250 and 400 ms) and auditory (i.e., 100 and 250 ms) attentional tasks could not allow a direct comparison of the time course of spatial attentional capture in the two modalities.

Thus, the aim of the present study was to precisely establish the time course of visual and auditory spatial attention in control and SRD children by using the same cue-target delays (i.e., 100 and 250 ms) for both the visual and auditory modalities.

In order to complete the experimental design (as correctly recommended by Goswami 2003, for developmental studies), a further control group matched on reading level (RL) was added. RL controls are typically normally reading children 2–3 years younger than the SRD children. The RL control group (which was also matched with SRD children on IQ level) enables causal hypotheses to be generated (Goswami 2003). Thus, if SRD children show deficits (i.e., sluggish attentional capture) in spatial attention compared to both chronological age (CA) controls and to RL controls, these deficits cannot be interpreted as a consequence of the reading deficits, and a causal link between spatial attention deficit and dyslexia may be suggested. Further support for the causal hypothesis can be found in a rehabilitation study showing that SRD children's ability to read improves following a specific training program that also improves their visual attentional focusing (Facoetti et al. 2003c).

In the present study, we measured the covert (i.e., without eye movements) automatic capture (e.g., Posner 1980) of both visual and auditory attention in 12 children diagnosed with SRD, 18 control children with normal IQ and reading skills, who were matched for chronological age (CA controls), and 9 younger reading-level (RL) control children with normal IQ and reading skills.

In Experiment 1, we measured the time course of focused visual spatial attention. Participants fixated the central point of a display. A non-informative peripheral visual cue preceded the onset of a subsequent target in the left or right visual field. In Experiment 2 the same children fixated the central point of a visual display, and a non-informative peripheral auditory cue, delivered by headphones, preceded the onset of a subsequent target tone in the left or right ear. After variable intervals (100 and 250 ms) from the onset of the spatial cue a target stimulus was presented at the cued or uncued location.

Faster responding to cued targets at the shorter interval (about 100 ms) reflects the facilitatory effect of automatic focusing of attention towards the cue (attentional facilitation). During the automatic capture of spatial attention (i.e., peripheral and non-predictive spatial cueing) this early facilitatory effect of attentional focus-

ing is no longer observed and it is replaced by a later inhibitory effect. Indeed, at longer cue-target intervals (about 250–350 ms), slower responding to the target at the cued location could reflect inhibition of return (IOR). This inhibitory effect is attributed to the withdrawal of attention and favors focusing towards novel locations (for a recent review see Klein 2000).

MATERIALS AND METHODS

Participants

Twelve children with SRD, ranging in age between 10 and 13 years, were selected from a sample of children referred to the Scientific Institute "E. Medea" because of learning difficulties. The children (mean age 11.9 years old; full scale IQ 97; verbal IQ 93; performance IQ 102) had been diagnosed as SRD based on standard criteria (American Psychiatric Association 1994). Their performance in reading aloud a text and/or single words and/or single non-words was 2 SDs below the mean on age-standardized Italian tests (Cornoldi et al. 1981, Sartori et al. 1995). SRD participants were 10 males and 2 females selected on the basis of: (i) a full-scale IQ greater than 85 as measured by the Wechsler Intelligence Scale for Children – Revised (WISC-R) (Wechsler 1974); (ii) normal or corrected-to-normal vision and hearing; (iii) the absence of attention deficit disorders with hyperactivity (ADHD) (American Psychiatric Association 1994); and (iv) right handedness.

Eighteen CA matched control children (mean age 11.7 years old) were selected. They had been recommended as normal readers by their teachers. They were at or above the norm with *z* scores of +0.44 (accuracy) and +0.46 (speed) on Italian Age-Standardized Single Word Reading Tests (Sartori et al. 1995). CA controls were of at least average intelligence, as measured by two WISC-R (Wechsler 1974) sub-tests (Vocabulary 12.3 standard score and Block Design 13.1 standard score).

In addition, 9 RL matched control children were selected (mean age 8.8 years old). RL controls were younger than SRD (3.1 years, $P<0.05$) and they were not different than SRD in both speed and accuracy ($Ps>0.05$) of word reading (Sartori et al. 1995). They were also of at least average intelligence, as measured by two WISC-R (Wechsler 1974) sub-tests (Vocabulary 12.1 standard score and Block Design 11.7 standard score). Table I shows descriptive data for the three groups of participants. All participants' parents gave informed consent.

Apparatus and stimuli

Tests were carried out in a dimly lit (luminance of 1.5 cd/m^2) and quiet room (approximately 50 dB SPL). Participants sat in front of a monitor screen (15 inches and

Table I

Descriptive data (means and SDs) of the three groups of participants

	Dyslexics (*n*=12)	CA Controls (*n*=18)	RL Controls (*n*=9)
Age (years)	11.9 (± 0.9)	11.7 (± 1.1)	8.8 (± 0.7)
Full IQ	97 (± 7)	/	/
Verbal IQ	93 (± 10)	12.3# (0.9)	12.1# (1.1)
Performance IQ	102 (± 11)	13.1§ (1.2)	11.7§ (0.8)
Word speed (*z* score)	-3.8 (± 2.5)	+0.5 (± 0.7)	+0.4 (± 0.8)
Word accuracy (*z* score)	-3.0 (± 2.8)	+0.4 (± 0.5)	-0.1 (± 0.7)
Word accuracy (number of errors)	7.5 (5)	/	6.2 (2)
Word speed (seconds)	183.7 (74.5)	/	131.5 (36.1)
Non-word speed (*z* score)	-3.1 (± 1.7)	/	/
Word accuracy (*z* score)	-2.3 (± 1.6)	/	/
Text speed (*z* score)	-3.6 (± 2.7)	/	/
Text accuracy (*z* score)	-2.6 (± 2.9)	/	/

(#) Vocabulary sub-test of the WISC-R, standard score; (§) Block Design sub-test of the WISC-R (Wechsler 1986), standard score

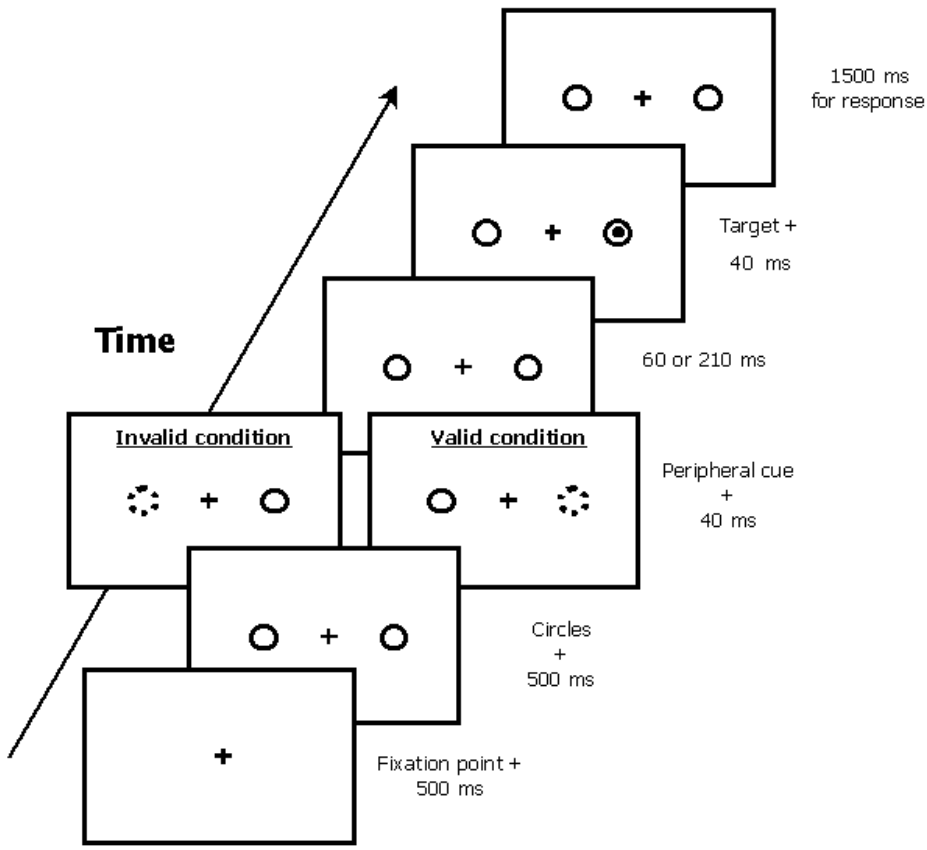

Fig.1. Schematic representation of the display used in the visual cued detection task (Experiment 1)

with a background luminance of 0.5 cd/m^2), with their head positioned on a headrest so that the eye-screen distance was 40 cm. The fixation point consisted of a cross (1° of visual angle) appearing at the center of the screen.

EXPERIMENT 1: VISUAL FOCUSED ATTENTION

Two circles (2.5°) were presented peripherally (8° of eccentricity), one to the left and one to the right of the fixation point. The peripheral cue consisted of the offset (40 ms in duration) and then the onset of one of the circles. A dot (0.5°) in the center of one of the two circles was the target stimulus (40 ms in duration). Stimuli were white and had a luminance of 24 cd/m^2 (see Fig. 1).

EXPERIMENT 2: AUDITORY FOCUSED ATTENTION

The sounds were presented over Sennheiser HD270 headphones. A single pure tone of 1 000 Hz was used as auditory cue and a single pure tone of 800 Hz was used as the target. The cue and target sounds were presented for 40 ms at approximately 65 dB SPL (see Fig. 2).

Procedure

Participants were instructed to keep their eyes on the fixation point throughout the duration of the trial. Eye movements were monitored by means of a video-camera system. Any eye movement larger than 1° was detected by the system and the corresponding trial was discarded but not replaced. Each trial started with the onset of the fixation point. In the auditory focused attention task, the cue was presented after 500 ms. In the visual focused attention task, after 500 ms, the two circles were displayed peripherally, and 500 ms later the cue was shown. The target was presented after one of two cue-target delays or stimulus onset asynchronies (SOA, 100 or 250 ms).

On each response trial a location cue presented in either the left or the right location was followed by a target presented in either the left or the right location. In contrast, on catch trials the target was not presented and participants did not have to respond. Catch trials were intermingled with response trials. On response trials, the probability that the target would appear in the same

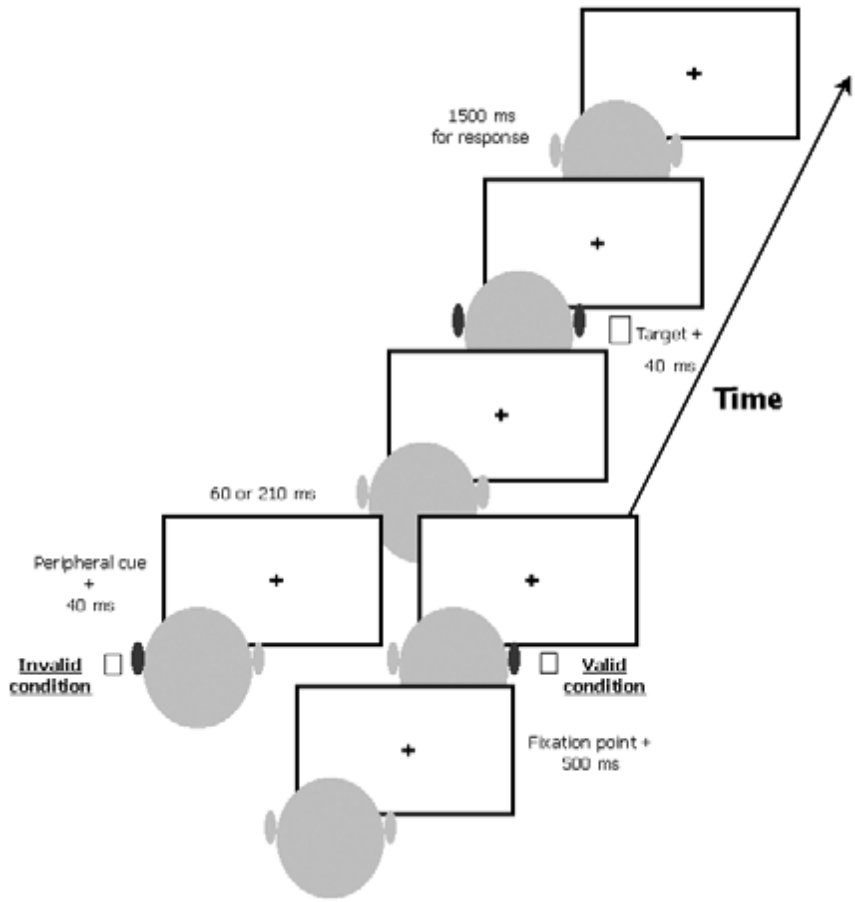

Fig. 2. Schematic representation of the display used in the auditory cued detection task (Experiment 2)

location as (a valid trial) or in a different location from (an invalid trial) the cue was 50% (i.e., there were an equal number of valid and invalid trials: cue location was non-predictive of target location).

Participants were instructed to react as quickly as possible to the onset of the target by pressing the spacebar on the computer keyboard. Both simple RTs and error rates were recorded by the computer. The maximum time allowed to respond was 1 500 ms. The inter-trial interval was 1 000 ms. The experimental session consisted of 160 trials divided into two blocks of 80 trials each. Trials were distributed as follows: 32 valid trials (16 for each cue-target delay), 32 invalid trials (16 for each cue-target delay), and 16 catch trials (20% of total trials). The administration sequence of the two Experiments was counterbalanced across subjects.

RESULTS

Errors in Experiment 1 (Visual Focused Attention), that is responses on catch trials and missed responses, were less than 3% and were not analyzed. Outliers were defined as RTs faster than 150 ms or more than 2.5 standard deviations above the mean and were excluded from the data sets before the analyses were carried out. This resulted in the removal of approximately 2% of all observations. Trials discarded because of eye movements were about 4% of total trials.

Errors in Experiment 2 (Auditory Focused Attention) were less than 2% and were not analyzed. Outliers were excluded from the data before the analyses were carried out. This resulted in the removal of approximately 2% of all observations. Trials discarded because of eye movements were about 1% of total trials.

Mean correct RTs were analyzed with a mixed ANOVA in which the three within-subject factors were stimulus mode (visual and auditory), cue condition (valid and invalid) and SOA (100 and 250 ms). The between-subject factor was group (CA controls, RL controls and SRD).

The main effect of SOA was significant, $F_{1,36}$=37.73, P<0.0001; RTs were faster at 250 ms (404 ms) than at 100 ms SOA (425 ms). The cue condition main effect

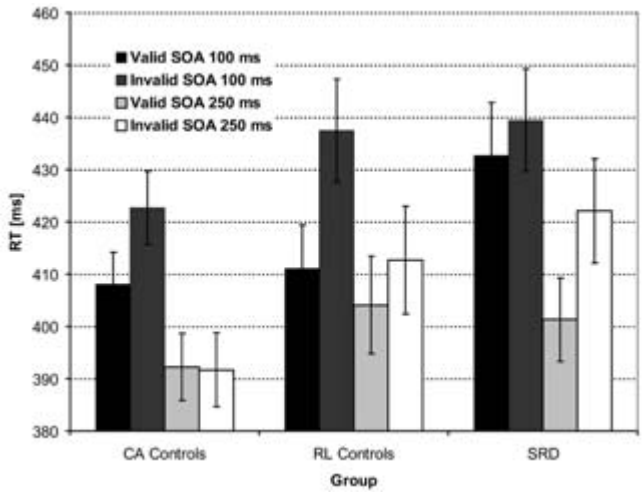

Fig. 3. Mean reaction times (RTs) as a function of group (SRD children, chronological age – CA controls, and reading level – RL controls), cue condition (valid and invalid) and cue-target delay (100 and 250 ms). Bar error represent ± one standard error.

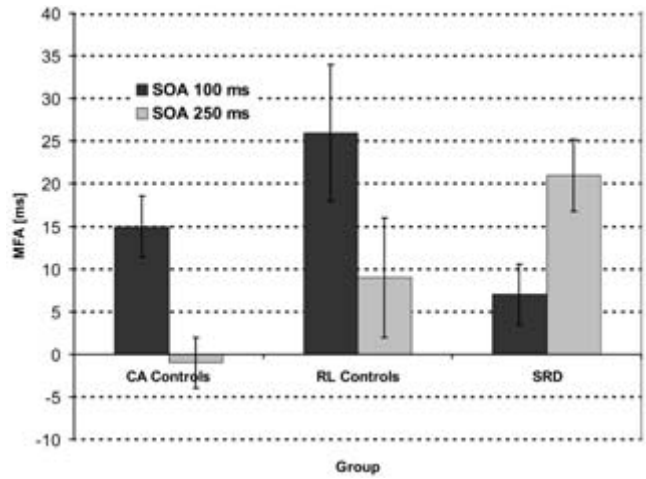

Fig. 4. Multimodal focused attention (MFA, i.e., visual and auditory mean of invalid-valid difference) as a function of cue-target delay (100 and 250 ms) and group (SRD children, chronological age – CA controls, and reading level – RL controls). Bar error represent ± one standard error.

was also significant, $F_{1,36}$=18.5, P<0.0002; RTs were faster for the valid cue condition (408 ms) than for the invalid cue condition (421 ms).

The stimulus mode × SOA interaction was significant, $F_{1,36}$=14.57, P<0.001, indicating that the SOA-warning effect varied across stimulus modalities. In the visual mode the SOA-warning effect was 10 ms (100 ms SOA = 422 ms; 250 ms SOA = 412 ms) whereas in the auditory mode the SOA-warning effect was 32 ms (100 ms SOA = 428 ms; 250 ms SOA = 396 ms). The stimulus mode × SOA × cue condition interaction was also significant, $F_{1,36}$=6.67, P<0.02, indicating that the time course of attentional capture varied across stimulus modalities. Specifically, at an SOA of 250 ms, in the visual mode the cue condition effect (i.e., attentional focusing) was significant (16 ms; invalid = 420 and valid = 404) whereas in the auditory mode it was not significant (3 ms; invalid = 397 and valid = 394), suggesting a faster withdrawal of attention in the auditory mode than in the visual mode.

More crucially, the group × SOA × cue condition interaction was also significant, $F_{2,36}$=6.88, P<0.005 (see Fig. 3), indicating that the time course of attentional capture varied across groups. Indeed, at the 100 ms SOA, both CA and RL controls showed a significant effect of attentional focusing (CA 15 ms, invalid = 423 and valid = 408; RL 26 ms, invalid = 437 and valid = 411) whereas the effect for SRD children was non-significant (6 ms, invalid = 439 and valid = 433). In contrast, at the 250 ms SOA, SRD children showed a significant effect of attentional focusing (21 ms, invalid = 422 and valid = 401) whereas for both CA and RL controls the effect was non-significant (CA 0 ms, invalid=392 and valid = 392; RL 9 ms, invalid = 413 and valid = 404). Since the group × stimulus mode × SOA × cue condition interaction was not significant (F<1), it can be suggested that the time course of attentional capture was not significantly different between different groups across the visual and auditory modalities.

To further study the SAS hypothesis, attentional focusing (i.e., invalid – valid cue conditions) was analyzed by means of a mixed ANOVA in which the two within-subject factors were stimulus mode (visual and auditory) and SOA (100 and 250 ms). The between-subject factor was group (CA controls, RL controls and SRD). Importantly, the group × SOA interaction was significant, $F_{2,36}$=6.87, P<0.005 (see Fig. 4), clearly indicating that attentional focusing at the two different SOAs varied across groups. Specifically, planned comparisons showed that attentional focusing significantly decreased from 100 to 250 ms of cue-target delays both in CA (from 14 to -1 ms; P<0.05) and in RL (from 26 to 9 ms; P<0.05) controls. In contrast, attentional focusing significantly increased from 100 to 250 ms SOA in SRD children (from 7 to 21 ms; P<0.05).

DISCUSSION

Dual-route (DR) models propose that skilled readers use two interactive procedures for converting print into

speech (for a recent review, see Coltheart et al. 2001): the sub-lexical route (i.e., phonological procedure) that involves a letter-to-sound mapping of the most frequent relationships between graphemes and phonemes in a given language allowing readers to read unfamiliar words and non-words, and the lexical route (i.e., orthographic procedure) that relies on whole-word recognition and retrieval of stored phonological codes and produces fast and efficient reading of familiar words.

It is crucial to note that for a beginning reader all real words are at first non-words because the lexical route is still to be developed. Accordingly, most developmental models based on the DR account assume that the two procedures are acquired serially, with beginning readers initially relying on the phonological route and only later shifting to the lexical route (e.g., Frith 1986). Other developmental models propose that a single phonological route is involved for learning to read both non-words and irregular words (e.g., Perfetti 1992, Share 1995). Indeed, most longitudinal studies have shown that beginning readers use primarily the phonological route for both reading aloud and silent reading (for a recent review see Sprenger-Charolles et al. 2003). This suggests that phonological processing may be gradually replaced by lexical processing.

One of the two major competing views of dyslexia maintains that dyslexics have normal auditory perception but have a specific deficit in coding linguistic input into phonological information. Indeed, there is much evidence that phonological processing deficits are linked to difficulties in learning to read: (i) phonological performances predict later reading skills; (ii) phonological processing deficits markedly distinguish children with dyslexia from children with normal reading skills; and (iii) phonological training has been shown to improve reading ability (e.g., Snowling 2000, Goswami 2000, for recent reviews see Goswami 2003, and Ramus 2003). Nevertheless, no study has provided unequivocal evidence, controlling for existing literacy skills in their participants, that there is a causal link from competence in phonological processing to success in reading and spelling acquisition (Castles and Coltheart 2004).

The competing view (i.e., the temporal processing deficit hypothesis) maintains that a more general auditory processing deficit characterizes dyslexia. Specifically, the difficulties in processing brief and rapid auditory cues impair the ability to perceive accurately crucial features in the speech stream, degrading the development of phonological codes (e.g., Farmer and Klein 1995, for a recent review see Wittman and Fink 2004). However, recent evidence suggests that the basic deficit is not specific to auditory stimuli modulated in time or briefly presented (e.g., Amitay et al. 2002). Instead, auditory "processing efficiency theory" suggests that SRD individuals have problems in the ability to detect acoustic signals in noise (Hartley and Moore 2002). In fact, nonlinguistic auditory perceptual deficits in dyslexia involve: (i) difficulties in discrimination between acoustically similar sounds; (ii) impaired ability to hear differences in sound frequency; (iii) problems in speech–sound perception (i.e., phoneme discrimination) in the presence of background noise; and (iv) deficits in processing rapid sound sequences (for a recent review see Wright et al. 2000). It has been suggested that all these nonlinguistic auditory perception deficits are likely to be related to an inability to focus auditory attention in order to discriminate properly and rapidly the features of the relevant signal sound (e.g., Vidyasagar 1999, Hari and Renvall 2001). Direct evidence for an auditory focused attention deficit in dyslexics is now provided by several studies (e.g., Asbjornsen and Bryden 1998, Facoetti et al. 2003b, Renvall and Hari 2002).

Hari and Renvall (2001) proposed that the causal link between reading deficits and phonological problems involves the capture of auditory and visual automatic attention. It has been suggested that the automatic capture of auditory attention could be directly linked to phonemic perception. Indeed, auditory focused attention may act to facilitate phoneme discrimination (e.g., Mondor and Bryden 1991). Phoneme discrimination is also related to pure phonological processes involved in the sub-lexical route (i.e., grapheme-to-phoneme correspondences and phonological short-term memory). SRD children have also been shown to be impaired in perceiving the rhythmic timing of speech, a deficit that is likely to affect the detection of "perceptual-centers" and therefore the segmentation of syllables into onsets and rhymes (e.g., Goswami 2003). Thus, we suggest that auditory attention might be crucial not only for phonemic but also for syllabic segmentation of the speech signal.

For decades researchers have been approaching reading and dyslexia from the standpoint not only of auditory and phonological contributions but also of visual contributions. In fact, many studies have shown a specific deficit of the magnocellular (M) visual system in dyslexia (e.g., Eden et al. 1996, Galaburda and Living-

stone 1993, for a review see Stein and Walsh 1997). However, the role of M deficit in dyslexia is hotly debated, mainly because of the lack of a clear causal link between M processing and impaired reading of isolated words and non-words. To complicate the picture, impaired performance on M-processing tasks appears to be associated mostly with the phonological subtype of dyslexia (e.g., Borsting et al. 1996, Cestnick and Coltheart 1999, Talcott et al. 1998).

It is widely assumed that the phonological route requires a primary graphemic parsing process, that is the segmentation of a grapheme string into its constituent graphemes (e.g., Cestnick and Colthart 1999, Colthart et al. 2001). Thus, it is clear that phonological assembly *via* the phonological route involves not only appropriate phonological skills (i.e., grapheme-to-phoneme correspondences and phonological short-term memory) but also visual spatial processing. Focused visuo-spatial attention is likely to be extremely important for letter parsing and segmentation. It is well known that focused spatial attention enhances visual processing not only in terms of processing speed but also of improved sensitivity (i.e., spatial resolution), and reduced interactions with "near" stimuli (spatial and temporal masking) (e.g., Braun 2002, Carrasco and McElree 2001). Therefore, we argue that a multimodal deficit in dyslexia is a much more plausible scenario than a single deficit in the visual or auditory processing domain (e.g., Cestnick 2001). Accordingly, the main result of the present study is that dyslexic children show a slower time course of both visual and auditory (i.e., multimodal) attentional capture than both CA and RL normally reading children. This result supports the multimodal SAS theory (Hari and Renvall 2001). It could be suggested that attention in SRD individuals tends to be distributed (e.g., Facoetti et al. 2000a), and thus to be affected by interfering spatial (e.g., Geiger and Lettvin 1999) and temporal stimuli (e.g., Di Lollo et al. 1983), because of this specific deficit (i.e., slowing down) of multimodal attentional focusing. This "distributed perception", involving both visual and auditory (and also tactile, see Grant et al. 1999) modalities, could cause a general inefficient processing of stimuli in any task requiring "vision with scrutiny" (i.e., focused spatial attention).

Since the time course of multimodal attention has been shown to be sluggish in SRD children in comparison with RL normally reading children, a possible causal link between spatial attentional deficits and reading disability can be suggested. In order to confirm this possible causal link, it would be necessary to show that a specific treatment of visual and auditory attention enhances SRD children's reading skills. Some support for this hypothesis can indeed be found in rehabilitation studies of dyslexia (e.g., Facoetti et al. 2003c, Geiger and Lettvin 1999, Kujala et al. 2001).

In conclusion, it is suggested that sluggish multimodal attentional focusing in dyslexic children may distort the development of phonological and orthographic representations that are crucial for learning to read.

What is the neurobiological substrate of multimodal sluggish attentional focusing?

The information processed by the M system ends in the posterior parietal cortex (PPC), which is the basic area controlling multimodal spatial attention (e.g., Downar et al. 2000). There is evidence of a supramodal spatial representation in the PPC with convergence of both auditory and visual inputs (e.g., Farah et al. 1989), and the existence of crossmodal cells has been documented in the PPC (e.g., Anderson et al. 1995). The PPC may be involved in spatial selection independently of modality through a multimodal map that would be used for orienting-focusing attention in both the auditory and visual modalities (e.g., Vidyasagar 1999).

A decreased M input to the dorsal visual stream would result in a bilateral dysfunction of the PPC. Although Skottun (2000), in a review of the studies of contrast sensitivity in dyslexia, found evidence both for and against the M theory, the M deficit could still influence higher visual processing stages through the dorsal pathway and therefore lead to reading difficulties *via* attentional mechanisms (e.g., Facoetti et al. 2003a, Hari and Renvall 2001, Vidyasagar 1999, for a recent review se Jaśkowski and Rusiak 2005, this issue).

Note that sluggish attention focusing is compatible with another possible interpretation. A mild dysfunction of the right parietal lobe might also underlie the sluggish focusing shown by dyslexic children and adults (e.g., Facoetti et al. 2000b, Hari et al. 2001). Schulte-Korne and coauthors (1999), using event-related potential recordings, revealed a decreased P200 response in the right posterior region in dyslexics, consistent with a right PPC impairment. In addition, Mazzotta and Gallai (1992) found a decreased P300 response in the right hemisphere of phonological dyslexics. Finally, poor readers showed lower N100 amplitudes in response to non-words, but not

in response to words at central sites of the right hemisphere (Wimmer et al. 2002).

In addition, patients with right parietal damage also show a severe loss in the perception of apparent motion in their "good" RVF (Battelli et al. 2001). This deficit is probably due to a bilateral loss in the "temporal resolution of spatial attention" to transient events that drive the apparent motion percept (e.g., Yantis and Gibson 1994). It is interesting to note that this motion perception deficit is similar to that shown in phonological dyslexics children (e.g., Cestnick and Coltheart 1999).

CONCLUSIONS

In summary, at the beginning of reading acquisition, the orthographic lexicon is not yet operating, so children learn to read *via* the phonological route. Among the processes that are necessary to the phonological route, graphemic parsing may be linked to visuo-spatial attention. In contrast, auditory spatial attention could be linked to phonemic perceptual processing, that is a basic skill essential for grapheme-to-phoneme correspondences, as well as for phonological short-term memory. In the present study, a slower multimodal focusing of attention was shown in SRD children as compared to both CA and RL controls. Finally, it is suggested that this sluggish attentional focusing could be interpreted as a parietal dysfunction.

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REFERENCES

American Psychiatric Association (1994) Diagnostic and Statistical Manual of Mental Disorders (4th Ed.). American Psychiatric Press, Washington DC.

Amitay S, Ben-Yehudah G, Banai K, Ahissar M (2002) Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. Brain 125: 2272–2285.

Anderson RA, Essick GK, Siegel RM (1995) Encoding of spatial location by posterior parietal neurons. Science 230: 456–458.

Asbjornsen AE, Bryden MP (1998) Auditory attention shifts in reading disabled students: Quantification of attentional effectiveness by the attentional shift index. Neuropsychologia 36: 143–148.

Battelli L, Cavanagh P, Intriligator J, Tramo MJ, Hennaf M, Michel F, Barton JJS (2001) Unilateral right parietal damage leads to bilateral deficit for high level motion. Neuron 32: 985–995.

Borsting E, Riddel WH, Dudeck K, Kelley C, Matsui L, Motoyama J (1996) The presence of a magnocellular deficit depends of the type of dyslexia. Vis Res 36: 1047–1053.

Brannan J, Williams M (1987) Allocation of visual attention in good and poor readers. Percept Psychophys 41: 23–28.

Braun J (2002) Visual attention: Light enters the jungle. Curr Biol 12: R599–R601.

Carrasco M, McElree B (2001) Covert attention accelerates the rate of visual information processing. Proc Natl Acad Sci U S A 98: 5363–5367.

Castles A, Coltheart M (2004) Is there a causal link from phonological awareness to success in learning to read? Cognition 91: 77–111.

Cestnick L (2001) Cross-modality temporal processing deficits in developmental phonological dyslexics. Brain Cogn 46: 319–325.

Cestnick L, Coltheart M (1999) The relationship between language-processing and visual-processing deficit in developmental dyslexia. Cognition 71: 231–255.

Coltheart M, Rastle K, Perry C, Langdon R, Ziegler J (2001) The DRC model: a model of visual word recognition and reading aloud. Psychol Rev 108: 204–258.

Cornoldi C, Colpo G, Gruppo MT (1981) MT Reading Test (in Italian). O.S. Organizzazioni Speciali, Florence.

Cunningham J, Nicol T, Zecker SG, Bradlow A, Kraus N (2001) Neurobiologic responses to speech in noise in children with learning problems: Deficits and strategies for improvement. Clin Neurophysiol 112: 758–767.

Di Lollo V, Hanson D, McIntyre JS (1983) Initial stages of visual information processing in dyslexia. J Exp Psychol Hum Percept Perform 6: 623–935.

Downar J, Crawley AP, Mikulis DJ, Davis KD (2000) A multimodal cortical network for the detection of changes in the sensory environment, Nat Neurosci 3: 277–283.

Eden GF, Vanmeter J, Rumsey J, Maisog J, Woods R, Zeffiro T (1996) Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature 382: 66–69.

Facoetti A, Paganoni P, Lorusso ML (2000a) The spatial distribution of visual attention in developmental dyslexia. Exp Brain Res 132: 531–538.

Facoetti A, Paganoni P, Turatto M, Marzola V, Mascetti GG (2000b) Visuospatial attention in developmental dyslexia. Cortex 36: 109–123.

Facoetti A, Turatto M, Lorusso ML, Mascetti GG (2001) Orienting of visual attention in dyslexia: Evidence for asym-

metric hemispheric control of attention. Exp Brain Res 138: 46–53.
Facoetti A, Lorusso ML, Paganoni P, Cattaneo C, Galli R, Mascetti GG (2003a) The time course of attentional focusing in dyslexic and normally reading children. Brain Cogn 53: 181–184.
Facoetti A, Lorusso M.L, Paganoni P, Cattaneo C, Galli R, Umiltà C, Mascetti GG (2003b) Auditory and visual automatic attention deficits in developmental dyslexia. Cogn Brain Res 16: 185–191.
Facoetti A, Lorusso ML, Paganoni P, Umiltà C, Mascetti GG (2003c) The role of visual spatial attention in developmental dyslexia: Evidence from a rehabilitation study. Cogn Brain Res 15: 154–164.
Farah MJ, Wong AB, Wong MA, Monheit MA, Morrow LA (1989) Parietal lobe mechanisms of spatial attention: Modality-specific or supramodal? Neuropsychologia 27: 461–470.
Farmer ME, Klein RM (1995) The evidence for a temporal processing deficit-linked to dyslexia. Psychon Bull Rev 2: 469–493.
Frith U (1986) A developmental framework for developmental dyslexia. Ann Dyslexia 36: 81–96.
Galaburda A, Livingstone M (1993) Evidence for a magnocellular deficit in developmental dyslexia. Ann N Y Acad Sci 682: 70–82.
Geiger G, Lettvin JY (1999) How dyslexics see and learn to read well. In: Reading and Dyslexia: Visual and Attentional Processes (Everatt J, ed.). Routledge, London and New York, p. 64–90.
Goswami U (2000) Phonological representations, reading development and dyslexia: Towards a cross-linguistic theoretical framework. Dyslexia 6: 133–151.
Goswami U (2003) Why theories about developmental dyslexia require developmental designs. Trends Cogn Sci 7: 534–540.
Grant AC, Zangaladze A, Thiagarajah MC, Sathian K (1999) Tactile perception in developmental dyslexia: A psychophysical study using gratings. Neuropsychologia 37: 1201–1211.
Hari R, Renvall H (2001) Impaired processing of rapid stimulus sequences in dyslexia. Trends Cogn Sci 5: 525–532.
Hari R, Valta M, Uutella K (1999) Prolonged attentional dwell time in dyslexic adults. Neurosci Lett 271: 202–204.
Hari R, Renvall H, Tanskanen T (2001) Left minineglect in dyslexic adults. Brain 124: 1373–1380.
Hartley DEH, Moore DR (2002) Auditory processing efficiency deficits in children with developmental language impairments. J Acoust Soc Am 112: 2962–2966.
Heim S (2005) The structure and dynamics of normal language processing – insights from neuroimaging. Acta Neurobiol Exp (Wars) 65: 95–116
Helenius P, Uutela K, Hari R (1999) Auditory stream segregation in dyslexic adults. Brain 122: 907–913.
Jaśkowski P, Rusiak P (2005) Posterior parietal cortex and developmental dyslexia. Acta Neurobiol Exp (Wars) 65: 79–94.
Klein RM (2000) Inhibition of return. Trends Cogn Sci 4: 138–147.
Kujala T, Karma K, Ceponiene R, Belitz S, Turkkila P, Tervaniemi R, Naatanen M (2001) Plastic neural changes and reading improvement caused by audiovisual training in reading-impaired children. PNAS 98: 10510–10514
Mazzotta G, Gallai V (1992) Study of the P300 event-related potential through brain mapping in phonological dyslexics. Acta Neurol (Napoli) 14: 173–186.
Mondor TA, Bryden MP (1991) The influence of attention on the dichotic REA. Neuropsychologia 29: 1179–1190.
Perfetti C (1992) The representation problem in reading acquisition. In: Reading Acquisition (Gough P, Enri L, Treisman R, eds.). Erlbaum, Hillsdale, NJ, p.107–143.
Posner MI (1980) Orienting of attention. Quart J Exp Psychol 32: 3–25.
Ramus F (2003) Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? Curr Opin Neurobiol 13: 212–218.
Renvall A, Hari R (2002) Auditory cortical responses to speech–like stimuli in dyslexic adults. J Cognit Neurosci 14: 757–768.
Sartori G, Job R, Tressoldi P (1995) Battery for the assessment of developmental reading and spelling disorders (in Italian). O.S. Organizzazioni Speciali, Florence.
Share DL (1995) Phonological recoding and self-teaching: sine qua non of reading acquisition. Cognition 55: 151–218.
Schulte-Korne G, Bartling J, Deimel W, Remschmidt H (1999) Attenuated hemispheric lateralization in dyslexia: Evidence of a visual processing deficit. Neuroreport 10: 3697–3701.
Skottun BC (2000) The magnocellular deficit theory of dyslexia: The evidence from contrast sensitivity. Vis Res 40: 111–127.
Snowling MJ (2000) Dyslexia. Blackwell, Oxford.
Sprenger-Charolles L, Siegel LS, Béchennec D, Serniclaes W (2003) Development of phonological and orthographic processing in reading aloud, in silent reading, and spelling: A four-year longitudinal study. J Exp Child Psychol 84: 194–217.
Stein J, Walsh V (1997) To see but not to read: The magnocellular theory of dyslexia. Trends Neurosci 20: 147–152.
Talcott JB, Hansen PC, Willis-Owen C, McKinnell IW, Richardson AJ, Stein J (1998) Visual magnocellular impairment in adult developmental dyslexics. Neuro-Opthal 20: 187–201.
Tallal P (1980) Auditory temporal perception, phonetic, and reading disabilities in children. Brain Lang 9: 182–198.
Vidyasagar TR (1999) A neural model of attentional spotlight: Parietal guiding the temporal. Brain Res Rev 30: 66–76.

Wechsler D (1974). Wechsler Intelligence Scale for Children – Revised. The Psychological Corporation, New York.

Wimmer H, Hutzler F, Wiener C (2002) Children with dyslexia and right parietal lobe dysfunction: Event-related potentials in response to words and pseudowords. Neurosci Lett 331: 211–213.

Wittmann M, Fink M (2004) Time and language – critical remarks on diagnosis and training methods of temporal-order judgment. Acta Neurobiol Exp (Wars) 64: 341-348.

Wright BA, Bowen RW, Zecker SG (2000) Nonlinguistic perceptual deficits associated with reading and language disorders. Curr Opin Neurobiol 10: 482–486.

Yantis S, Gibson BS (1994) Object continuity in apparent motion and attention. Canad J Exp Psychol 48: 182–204.

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