



Behavioral and ERP evidence for amodal sluggish attentional shifting in developmental dyslexia

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ABSTRACT

The goal of this study was to examine the claim that amodal deficits in attentional shifting may be the source of reading acquisition disorders in phonological developmental dyslexia (sluggish attentional shifting, SAS, theory, Hari & Renvall, 2001). We investigated automatic attentional shifting in the auditory and visual modalities in 13 dyslexic young adults with a phonological awareness deficit and 13 control participants, matched for cognitive abilities, using both behavioral and ERP measures. We tested automatic attentional shifting using a stream segregation task (perception of rapid succession of visual and auditory stimuli as one or two streams). Results of Experiment 1 (behavioral) suggested that in order to process two successive stimuli separately dyslexic participants required a significantly longer inter-stimulus interval than controls regardless of sensory modality. In Experiment 2 (ERPs), the same participants were tested by means of an auditory and a visual oddball tasks involving variations in the tempo of the same alternating stimuli as Experiment 1. P3b amplitudes elicited by deviant tempos were differently modulated between groups, supporting predictions made on the basis of observations in Experiment 1. Overall, these results support the hypothesis that SAS in dyslexic participants might be responsible for their atypical perception of rapid sequential stimulus sequences in both the auditory and the visual modalities. Furthermore, these results bring new evidence supporting the link between amodal SAS and the phonological impairment in developmental dyslexia.

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1. Introduction

Developmental dyslexia is a specific deficit in written language acquisition that occurs despite normal intelligence and learning opportunities and in the absence of sensory or psychiatric disorders (Shaywitz & Shaywitz, 2005). Amongst many hypotheses regarding the possible origins of dyslexia, the phonological deficit hypothesis remains the most accepted and documented (Snowling, 2000; Vellutino, Fletcher, Snowling, & Scanlon, 2004 for a review). In this framework, a dysfunction in the build up of phonemic and phonological representations, necessary for adequate decoding skills (e.g., Hulme & Snowling, 1992), would lead to difficulties in acquiring automatic fluent reading (Share, 1995).

Several hypotheses have been put forward to explain the source of the phonological deficit itself. Focusing on the auditory component of phonological perception, a number of studies have suggested central auditory deficits in developmental dyslexia (Banai & Ahissar, 2006; Bailey & Snowling, 2002; Tallal, 1980). For instance, rapid auditory temporal processing deficits have been repeatedly reported in relation to phonological difficulties in dyslexic adults (Helenius, Uutela, & Hari, 1999; Lallier et al., 2009) and children (Boets, Wouters, van Wieringen, De Smedt, & Ghesquiere, 2008; Meng et al., 2005). In addition, research focusing on amodal temporal deficits in dyslexia (Farmer & Klein, 1995) has brought to light a possible involvement of both the visual transient magnocellular system and its auditory counterpart (Stein & Talcott, 1999; Van Ingelghem et al., 2001; Witton et al., 1998). However, the evidence for a role of magnocellular temporal processing in reading (Au & Lovegrove, 2008; Hulslander et al., 2004) and phonological processing (Boets et al., 2008; Heim et al., 2008; Kronbichler, Hutzler, & Wimmer, 2002; Ramus et al., 2003) remains inconsistent.

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Moving away from perceptual-level explanations but still compatible with a magnocellular deficit, Hari and Renvall (2001) acknowledge the proposition made by Stein and Walsh (1997) that a failure of attention subtended by a parietal lobe deficit could explain temporal impairments in developmental dyslexia. The magnocellular theory offers an attractive neurophysiological framework to explain developmental dyslexia, but a dorsal stream function (e.g., temporal perceptual processing) may not reliably reflect a magnocellular deficit since the dorsal pathway also receives parvocellular and koniocellular inputs (Skottun & Skoyles, 2006). Therefore, the temporal processing impaired in developmental dyslexia may not only be characterized by magnocells' functional properties, but also by that of the cerebral structure most affected by the magnocellular dysfunction, e.g., the parietal lobe (Vidyasagar & Pammer, 2010). With these considerations in mind, Hari and Renvall (2001) put forward the amodal sluggish attentional shifting (SAS) as the origin of rapid temporal processing deficits in dyslexia.

According to the SAS theory, when dyslexic individuals are faced with rapid stimulus sequences, their automatic attention system cannot disengage fast enough from one item to move to the next. The SAS theory is supported by studies conducted separately in the auditory (e.g., Hari, 1995; Hari & Kiesilä, 1996; Helenius et al., 1999) or the visual (e.g., Hari, Renvall, & Tanskanen, 2001; Hari, Valta, & Uutela, 1999) modality in different groups of dyslexic participants. However, few studies have examined an attentional deficit in both visual and auditory rapid serial presentation tasks in the same dyslexic participants. Using a spatial cueing paradigm, Facioetti, Lorusso, Cattaneo, Galli, and Molteni (2005) and Facioetti et al. (2010) have found that dyslexic children exhibit slower covert attentional orienting skills in both modalities. In addition, Lallier, Berger, Donnadieu, and Valdois (2010) showed that a dyslexic adult presenting with phonological problems was impaired in both visual and auditory attentional blink tasks similarly designed. Finally, Lallier et al. (2009) reported that dyslexic adults had higher thresholds in both auditory and visual stream segregation tasks whereas dyslexic children were impaired on the auditory task only.

Interestingly, stimulus stream integration/segregation deficits have been consistently found in relation to reading impairments in both children (Lallier et al., 2009; Ouimet & Balaban, 2010) and adults (Helenius et al., 1999; Lallier et al., 2009). Processing correctly acoustic cues at fast tempo in speech streams is crucial with respect to reading acquisition, and to language acquisition more generally (e.g., Pasquini, Corriveau, & Goswami, 2007; Schulte-Körne, Deimel, Bartling, & Remschmidt, 1999a). Thus, the phonological disorder which is a common source of literacy difficulties in developmental dyslexia may itself derive from an atypical perception of rapid auditory sequences in the dyslexic brain. The role of rapid visual processing in phonological skills development is less clear although it may relate to sequential visual attention processes required to analyze and segment the orthographic input before its conversion into a phonological code (Pammer & Vidyasagar, 2005).

So far, the possible role of SAS in developmental dyslexia has only been studied using behavioral measures, although Neville, Coffey, Holcomb, and Tallal (1993) have provided both behavioral and neurophysiological evidence for visual and auditory attentional deficits in language-impaired children. The goal of the present study was to strengthen and extend the current evidence for an amodal SAS deficit, by examining rapid stimulus stream perception using both behavioral measures and event-related potentials (ERPs) in the same participants. ERPs are particularly adapted to the study of fine temporal processing differences because of their high temporal resolution (data points every millisecond over a number of scalp sensors). Moreover, ERP data may reflect the perceptual experience elicited by rapid stimulus streams more directly

than behavioral measures which are more susceptible to strategic response biases or may lack sensitivity. As an illustration of this general point, Stoodley, Hill, Stein, and Bishop (2006) showed that dyslexic adults had normal auditory psychophysical thresholds but reduced ERP amplitudes as compared to controls, even though the same testing materials were used in the two methodologies. According to Stoodley et al. (2006) the fact that their participants were high-functioning dyslexic adults may explain why they did not exhibit deficits in behavioral tasks while still showing anomalies using more sensitive electrophysiological measures. Note that this study did not test the SAS theory (Hari & Renvall, 2001) since the frequency modulation detection task used did not involve rapid sequences of stimuli. Indeed, the SAS theory proposes that the temporal deficit in developmental dyslexia exclusively affects sequential processing by increasing the processing time between stimuli. Importantly, the SAS theory remains compatible with the magnocellular hypothesis of developmental dyslexia that hypothesizes temporal deficits to be both *transient*, i.e., affecting the processing of temporal variations within a single stimulus, and *sequential*.

The aim of the present study was to establish a link between behavioral (Experiment 1) and electrophysiological (Experiment 2) evidence for a sequential SAS deficit in dyslexic adults matched for cognitive abilities with control adults. The SAS deficit will be measured using stream segregation tasks (tested in adaptive and oddball paradigm contexts, respectively) in both the visual and the auditory modalities (Helenius et al., 1999; Lallier et al., 2009). Our hypothesis is that an amodal SAS deficit (Experiment 1) will be accompanied by an atypical perception of rapid stimulus sequences in both modalities (Experiment 2).

In both experiments, participants were presented with streams of alternating tones (high/low pitch) or alternating dots (above/below fixation). In Experiment 1, participants engaged in a stream segregation task as used in Lallier et al. (2009). This task measures the speed at which participants automatically disengage their attention from a given stimulus and reengage with the next. This is done by varying gradually the tempo of stimulus alternation according to whether participants report perceiving one or two streams, which allows us to establish individual SOA-driven segregation thresholds. According to the SAS theory, dyslexic participants should show higher visual and auditory segregation thresholds. Namely, they should require a longer time interval between stimuli in order to start perceiving successive auditory/visual stimuli as independent from one another.

In Experiment 2, the same participants performed visual and auditory oddball tasks involving stimulus sequences varying in SOA. Based on the segregation thresholds determined in Experiment 1, we measured P3b ERP responses elicited by the detection of deviant SOA targets (fast tempo and ambiguous tempo) embedded in standard SOA stimulus series (slow tempo). For both groups, the slow tempo (SOA 340 ms) allowed full disengagement/reengagement of attention with each stimulus and the fast tempo deviant prevented shifting attention back and forth between stimuli (SOA 90 ms). The ambiguous tempo deviant was intermediate (SOA 175 ms) and likely to allow disengagement for control participants only. Thus, differences between fast and ambiguous deviant tempos on amplitude in the P3b range were expected to arise between participants groups.

2. Methodological aspects common to Experiments 1 and 2

2.1. Participants

Twenty-six adult volunteers (13 dyslexics: 5 males, 1 left-handed, 20.4 ± 1.0 years old; 13 controls: 4 males, 1 left-handed,

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