



PAPER

Speech-perception-in-noise deficits in dyslexia

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Abstract

Speech perception deficits in developmental dyslexia were investigated in quiet and various noise conditions. Dyslexics exhibited clear speech perception deficits in noise but not in silence. Place-of-articulation was more affected than voicing or manner-of-articulation. Speech-perception-in-noise deficits persisted when performance of dyslexics was compared to that of much younger children matched on reading age, underscoring the fundamental nature of speech-perception-in-noise deficits. The deficits were not due to poor spectral or temporal resolution because dyslexics exhibited normal 'masking release' effects (i.e. better performance in fluctuating than in stationary noise). Moreover, speech-perception-in-noise predicted significant unique variance in reading even after controlling for low-level auditory, attentional, speech output, short-term memory and phonological awareness processes. Finally, the presence of external noise did not seem to be a necessary condition for speech perception deficits to occur because similar deficits were obtained when speech was degraded by eliminating temporal fine-structure cues without using external noise. In conclusion, the core deficit of dyslexics seems to be a lack of speech robustness in the presence of external or internal noise.

Introduction

About 5% of children in primary school exhibit severe and long-lasting problems in acquiring written language despite normal intelligence, adequate educational opportunities and in the absence of any obvious neurological or sensory deficiencies (Snowling, 2000). This disorder is referred to as *developmental dyslexia (DD)*. The hallmark of DD is extremely slow and error-prone reading, poor nonword decoding and weak spelling.

The causes of DD are still hotly debated (Demonet, Taylor & Chaix, 2004). While some authors see the causes of DD in visual-attentional deficits (Stein & Walsh, 1997) or sensori-motor dysfunction (Nicolson, Fawcett & Dean, 2001), the most influential theory attributes DD to a deficit in the use and representation of phonological information (Vellutino, Fletcher, Snowling & Scanlon, 2004). Because learning to read is fundamentally about mapping an orthographic code onto a fine-grained phonological code, poor and/or underspecified phonological representations will inevitably lead to deficits in learning to read (Ziegler & Goswami, 2006). Indeed, when the phonological deficit theory is directly compared to the competing visual or sensori-motor theories, it becomes quite clear that the majority of children with

DD suffer from phonological deficits (Ramus, Rosen, Dakin, Day, Castellote, White & Frith, 2003; White, Milne, Rosen, Hansen, Swettenham, Frith & Ramus, 2006).

One key question therefore is to what extent the phonological deficits stem from a more general deficit in auditory perception (Mody, Studdert-Kennedy & Brady, 1997; Rosen, 2003; Tallal, 2003). Indeed, previous research has often suggested that dyslexics show abnormal performance in a variety of auditory tasks, such as frequency discrimination (Ahissar, Protopapas, Reid & Merzenich, 2000; Banai & Ahissar, 2004; Witton, Stein, Stoodley, Rosner & Talcott, 2002), amplitude modulation detection (Goswami, Thomson, Richardson, Stainthorpe, Hughes, Rosen & Scott, 2002; Muneaux, Ziegler, Truc, Thomson & Goswami, 2004) or auditory stream segregation (Helenius, Uutela & Hari, 1999). However, the problem for a general auditory deficit theory is that (1) only a subgroup of children with DD seem to show robust auditory deficits, (2) some controls seem to show abnormal auditory processing and yet have not developed dyslexia, (3) deficits in auditory processing tend to correlate poorly with reading skills within the group of dyslexics, and (4) auditory deficits seem to disappear when dyslexics are compared with reading-level controls (for a summary of these arguments see Rosen, 2003).

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The fact that a general auditory deficit theory runs into the above problems has led many researchers to abandon the idea that low-level speech perception deficits may be at the origin of the phonological deficits seen in dyslexia (Ramus, White & Frith, 2006). However, speech perception is complex and relies on the simultaneous integration of a variety of acoustic cues across different time scales (Greenberg, 2006). Thus, the fact that a deficit in perceiving a single acoustic cue (e.g. rapid frequency changes) is not consistently associated with phonological deficits does not rule out the possibility that speech perception deficits are at the origin of poor phonological development.

A few studies have investigated speech perception deficits but the results are rather mixed. Brandt and Rosen (1980) failed to demonstrate speech perception problems in children with dyslexia. Dyslexics labelled and discriminated speech sounds much like normal-reading children and adults. Adlard and Hazan (1998) found that no more than 30% of the dyslexic children had speech perception deficits. Similarly, Manis, McBride-Chang, Seidenberg, Keating, Doi, Munson and Petersen (1997) found that the majority of dyslexics had normal categorical perception on a voicing continuum. In their study, only 28% of the dyslexics had a deficit and this deficit was not significant in comparison to reading-level controls. Maassen, Groenen, Crul, Assman-Hulsmans and Gabreëls (2001) reported categorical perception deficits but these deficits were present only in discrimination not in identification. Cornelissen, Hansen, Bradley and Stein (1996) found speech perception deficits but these deficits were restricted to very few phonetic contrasts. Finally, Blomert and Mitterer (2004) found speech perception deficits but these deficits were only present in synthetic not in natural speech. Indeed, only very few studies seemed to show robust speech perception deficits (Godfrey, Syrdal-Lasky, Millay & Knox, 1981; Lieberman, Meskill, Chatillon & Schupack, 1985; Mody *et al.*, 1997; Serniclaes, Sprenger-Charolles, Carre & Demonet, 2001; Serniclaes, Van Heghe, Mousty, Carre & Sprenger-Charolles, 2004).

One reason for the fragility of speech perception deficits in the above-mentioned studies might be that speech perception has typically been investigated in quiet conditions, that is in an optimal listening situation where deficient access to certain speech cues may be compensated for by normal access to other redundant speech cues. Indeed, perfect or nearly perfect speech perception in quiet can be achieved in normal-hearing listeners despite severely limited access to certain spectral or temporal cues (e.g. Lorenzi, Gilbert, Carn, Garnier & Moore, 2006; Shannon, Zeng, Kamath, Wygonski & Ekelid, 1995). However, in real life, speech perception generally occurs against a background of various sound sources (e.g. multiple talkers, steady or fluctuating noise). In such adverse listening situations, robust recognition relies heavily on the integration of corrupted (i.e. masked), but fortunately redundant, spectral and

temporal speech cues. As a consequence, any reduction in speech redundancy is likely to cause poorer-than-normal speech perception in noise. Consistent with this notion, a number of studies found clear speech perception deficits in children with developmental language disorders in noise but not in quiet (Bradlow, Kraus & Hayes, 2003; Brady, Shankweiler & Mann, 1983; Ziegler, Pech-Georgel, George, Alario & Lorenzi, 2005). Moreover, in children with language learning disabilities, the neurophysiological responses to repeated speech stimuli were found to be abnormal and asynchronous in noise but not in quiet (Wible, Nicol & Kraus, 2002).

The goal of the present study was to use a psychophysical approach to investigate speech perception deficits in dyslexia. We investigated speech perception in noise while manipulating the temporal properties of the noise (see Figures 1a and b). Indeed, in conditions of temporally fluctuating noise, unimpaired listeners experience *release from masking*, that is, better speech identification in fluctuating than in stationary noise (Duquesnoy, 1983; Festen & Plomp, 1990; Miller & Licklider, 1950). Masking release occurs when the auditory system is capable of taking advantage of relatively short temporal minima in the fluctuating background to detect speech cues. It therefore requires a certain degree of temporal and spectral resolution (e.g. Peters, Moore & Baer, 1998). Interestingly, patients with sensorineural (that is, cochlear) hearing loss show strongly reduced masking release (Duquesnoy, 1983; Gustafsson & Arlinger, 1994; Lorenzi, Husson, Ardoit & Debrulle, 2006; Peters *et al.*, 1998). Normal masking release therefore suggests that low-level auditory or peripheral processes are intact.

Using such a psychophysical approach, Ziegler *et al.* (2005) have shown that children with language learning disabilities exhibited poor speech perception in noise for fast as well as slow amplitude modulated noise conditions, suggesting that the deficit could not be reduced to a rapid temporal processing deficit (Tallal, 1980). Most importantly, these children showed a perfectly normal speech masking release, which suggested that (i) the deficit was not due to poor temporal or spectral resolution, and (ii) children with language learning disabilities showed normal peripheral processes. Finally, the greatest deficits were obtained for the *voicing* feature (e.g. /b/ versus /p/), which clearly contrasted with the general pattern of phonetic deficits reported in listeners with sensorineural hearing, for whom reception of *place-of-articulation* is mostly degraded whereas reception of *voicing* and *manner* are barely affected (Baer, Moore & Kluk, 2002; Vickers, Moore & Baer, 2001).

In the present study, we replicated the speech-perception-in-noise experiment by Ziegler *et al.* (2005) with both stationary and fluctuating speech-shaped noise (4, 32 and 128 Hz). We were interested in finding out whether dyslexics would exhibit speech perception deficits in noise but not in silence. If so, we wanted to investigate whether these deficits would persist with respect to

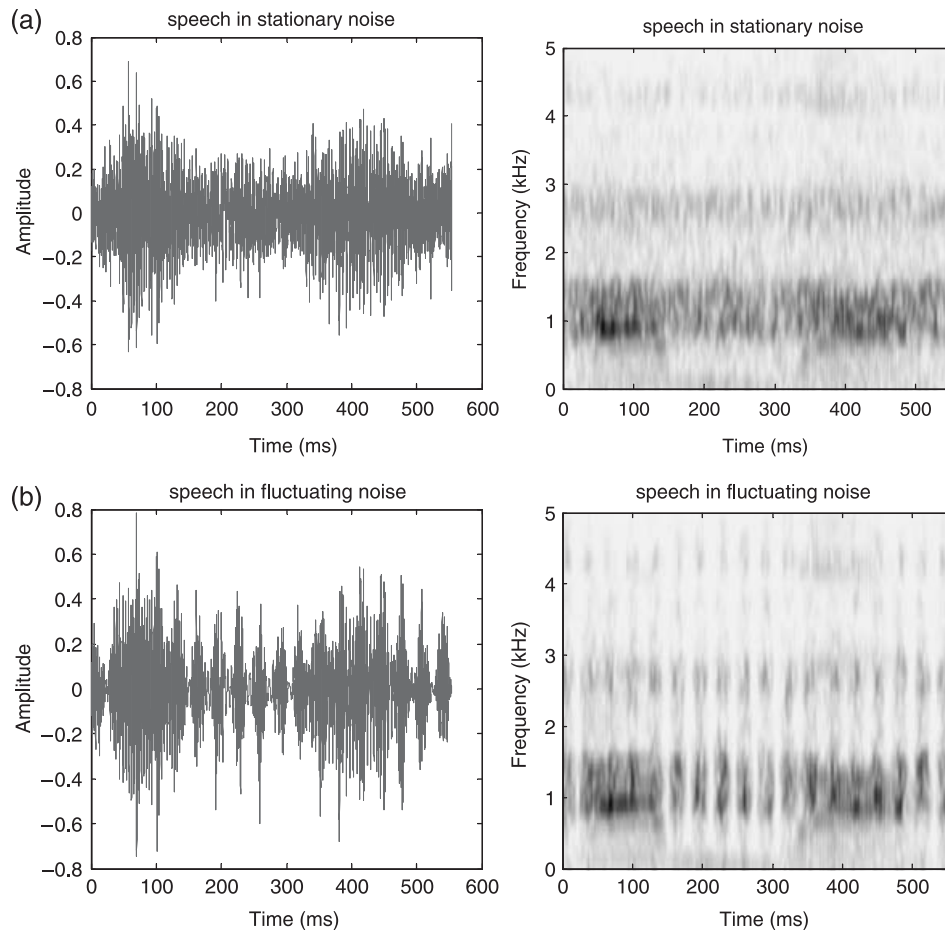


Figure 1 Critical conditions in the experiment in noise (left panels: waveforms; right panels: spectrograms). (a) intact VCV bisyllable (/aba/) in stationary speech-shaped noise, (b) intact VCV bisyllable (/aba/) in sinusoidally amplitude-modulated speech-shaped noise (three amplitude modulation rates are used: $fm = 4, 32,$ and 128 Hz). Here, a 32-Hz amplitude-modulation is used.

reading-level controls (e.g. Manis *et al.*, 1997). This comparison is crucial in order to show that the speech perception deficit is not simply a consequence of the reading deficit (e.g. Goswami, 2003). It was of major interest to investigate whether dyslexics showed a normal masking release effect. If so, this would suggest that low-level temporal or spectral processes are relatively spared, which would point to a central (i.e. post-cochlear) and specifically phonetic deficit (e.g. Mody *et al.*, 1997). Finally, we were interested in finding out whether dyslexics would show the same pattern of phonetic deficits as the language-impaired children in Ziegler *et al.* (2005), that is, greater deficits for *voicing* than for other phonetic features.

One important unsettled issue concerns the question whether the speech-perception-in-noise deficits are due to a general problem with *noise exclusion* or whether external noise is simply a sufficient but not necessary condition for speech deficits to occur. Indeed, a recent study suggested that the detection of visual impairments in dyslexia entirely depended on the presence of noise in the visual display (Sperling, Lu, Manis & Seidenberg, 2005). This finding led the authors to suggest that

dyslexics might suffer from a general noise exclusion problem (see also Sperling, Lu, Manis & Seidenberg, 2006). One way to test this prediction is to corrupt the speech signal – not by the addition of noise – but by degrading the temporal fine structure of the acoustic signal within a limited number of adjacent audio-frequency bands whose bandwidth was chosen to be broader than normal (cochlear) auditory filters (see Figure 2b). This degradation (by a so-called tone-excited envelope vocoder) is commonly used to reduce speech redundancy as found in sensorineural hearing loss. More specifically, the tone-excited vocoder removes temporal fine structure cues within each frequency band and degrades frequency resolution by a factor of 2, as found in moderate sensorineural hearing loss (Lorenzi, Gilbert *et al.*, 2006; Shannon *et al.*, 1995). If external noise is not a necessary condition for speech deficits to occur, we should observe deficits in *all* degraded-speech conditions. Such a deficit would suggest a *general lack of speech robustness* rather than a *noise exclusion deficit*.

Finally, to check whether speech-perception-in-noise deficits explained unique variance in reading, we added

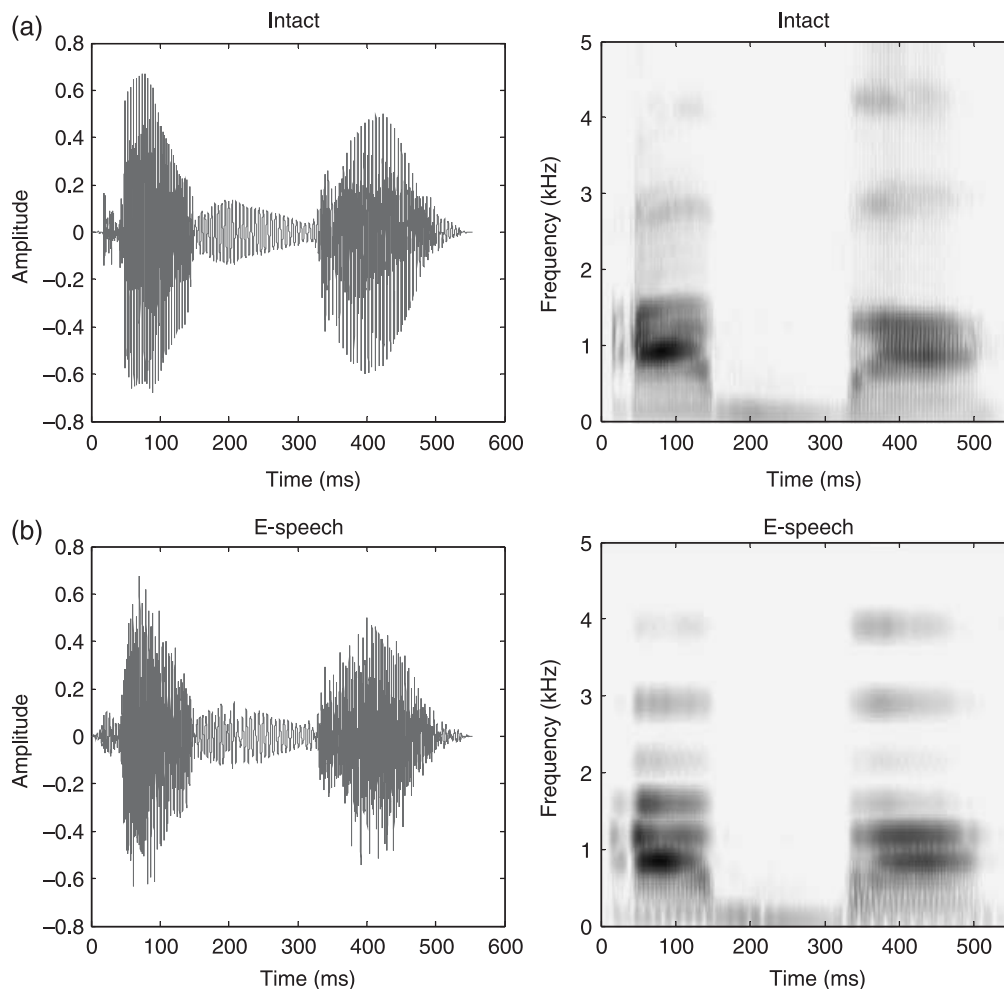


Figure 2 Critical conditions in the experiment in quiet (left panels: waveforms; right panels: spectrograms). (a) intact VCV bisyllable (/aba/) in quiet, (b) a tone-excited envelope vocoded VCV bisyllable (/aba/) in quiet.

different tasks to assess the possible contribution of general cognitive ability, verbal memory, low-level visual and auditory processes and sustained attention. Verbal memory was assessed with word and digit span, low-level visual and auditory processes were assessed with temporal order thresholds for pure tones and light flashes and sustained attention was assessed by an object cancellation task. In summary, the present study tried to answer the following five questions:

1. Is there evidence for a speech-perception-in-noise deficit in dyslexia?
2. If so, does the deficit persist in comparison with reading-level controls?
3. Is the deficit restricted to *voicing* or does it affect all phonetic features equally?
4. Is external noise a necessary or sufficient condition for speech deficits to occur?
5. Does speech-perception-in-noise predict reading performance beyond general cognitive ability, verbal memory, low-level auditory processing and sustained attention?

Methods

Participants

Nineteen dyslexic children were recruited from the University Hospital La Timone Marseille, France. They were aged between 8;6 and 12;1 years with an average of 10;4 years. Prior to the study, all dyslexics received a complete medical, psychological, neuropsychological and cognitive assessment. This assessment was done by an interdisciplinary team of psychologists, neurologists and speech therapists. Dyslexics were included in the study if their reading age was at least 18 months below the age norm on a standardized reading test (Alouette; Lefavrais, 1965) and if their performance IQ was above 80 on the Wechsler III intelligence scale (Wechsler, 1996). They were excluded from the study if their oral language skills were in the pathological range (i.e. formal diagnosis of SLI) on any of three standardized oral language tests, the L2MA (Chevrie-Muller, Simon, Fournier & Brochet, 1997), the NEEL (Chevrie-Muller & Plaza, 2001) and the ECOSEE (Lecocq, 1996). Prior to participating in the study, a

Table 1 Description of the population and performance in various cognitive tasks. Standard deviations in (). Min/max values in []

	Groups			Effect size (Cohen's <i>d</i>)	
	Dyslexics	CA controls	RA controls	Dys – CA	Dys – RA
Chronological age	10.4 (1.0) [8.5–12.1]	10.4 (1.0) [8.7–12.2]	7.2 (0.4) [6.5–8.2]	.10	4.20***
Reading age	7.2 (0.5) [6.5–8.2]	9.8 (1.3) [8.4–13.2]	7.5 (0.9) [5.9–9.4]	2.63***	.41
Nonverbal IQ	99.8 (12.6) [80–119]	108.2 (20.5) [68–148]	101.8 (18.2) [71–136]	.73	.13
Reading					
Regular words	91.0 (14.0) [60–100]	100 (0) [100–100]	94.2 (10.2) [65–100]	.90**	.26
Irregular words	67.9 (26.2) [10–100]	97.2 (4.6) [90–100]	76.7 (18.1) [40–95]	1.61***	.38
Nonwords	61.6 (21.0) [15–95]	96.9 (3.5) [90–100]	78.6 (11.2) [40–100]	2.28***	1.01**
Phonology					
Phono. awareness	41.0 (21.7) [0–85]	86.1 (10.4) [60–100]	72.1 (18.5) [0–85]	2.64***	1.54***
Word repetition	88.9 (8.2) [67–100]	98.5 (3.1) [90–100]	97.5 (3.9) [87.5–100]	1.54***	1.34***
RAN	953 (343) [430–1480]	481 (208) [350–1280]	986 (483) [450–2320]	1.66***	.04
Fluency	12.3 (6.3) [2–24]	17.8 (4.8) [9–28]	9.58 (4.2) [4–17]	.99**	.51
Memory					
Word span	3.7 (1.1) [2–5]	4.4 (1.1) [2–6]	3.7 (.47) [3–4]	.62	.05
Digit span	2.1 (0.8) [1–4]	3.4 (0.8) [2–5]	4.3 (1.2) [2–6]	1.70***	2.22***
Backward digit	1.5 (0.9) [0–3]	3.0 (1.3) [1–5]	1.7 (0.6) [1–3]	1.29***	.24
Attentional/visual/auditory processes					
Sustained attention	264 (207) [110–1020]	243 (121) [100–550]	312 (177) [169–1001]	.12	.24
Visual order thres	157 (120) [30–400]	62 (57) [28–280]	86 (70) [18–360]	1.01**	.72*
Auditory order thres	152 (107) [42–500]	73 (64) [18–280]	135 (70) [65–320]	.89*	.19

pure-tone audiogram was obtained for all dyslexic children, which showed that all of them had normal audiometric thresholds between 0.25 and 6 kHz (< 20 dB HL).

Two control groups were selected from nearby schools. The first consisted of 18 children matched on chronological age (CA controls). The second consisted of 19 children matched on reading age (RA controls). None of the controls reported a history of written or oral language impairment. More details are found in Table 1. The study was conducted with the understanding and consent of the participants and their parents.

Tasks

Reading

Reading age was obtained with a standardized reading test that takes into account both speed and accuracy (Alouette; Lefavrais, 1965). Reading and decoding skills were further assessed by having children read 10 regular words, 10 irregular words and 20 pseudowords. The items were taken from a recent study (Ziegler, Castel, Pech-Georgel, George, Alario & Perry, 2008).

Phonological tasks

Several tasks were used to assess phonological skills. In the *word repetition* task, the children had to repeat aloud a list of 10 complex words that were read aloud by the experimenter. The items were taken from a standardized test (Chevrie-Muller *et al.*, 1997). In the *rapid automatized naming* task (RAN), a child was asked to name as quickly as possible 50 object drawings of five repeatedly displayed objects (Castel, Pech-Georgel, George & Ziegler, 2008). In the phonological fluency task, children were given 1 minute to produce a maximum number of words starting with /p/ (first round) and /f/ (second round). The *phonological awareness* tasks consisted of two subtests (phoneme fusion and phoneme deletion) that were taken from the ODEDYS test battery (Jacquier-Roux, Valdois & Zorman, 2002). In the *phoneme fusion* task, a child was asked to extract the first phoneme of two spoken words and then blend the two phonemes into a new syllable (10 trials). In the *phoneme deletion* task, the child was asked to delete the first phoneme of a spoken word and pronounce the remaining part of the word (10 trials).

Memory

Three verbal memory tasks were used that were taken from the *L2MA* (Chevrie-Muller *et al.*, 1997): Immediate recall of six words that belonged to different semantic categories (10 trials), digit span and backward digit span.

Sustained attention

To measure sustained attention, the object cancellation task was used (Di Filippo, Brizzolara, Chilosi, De Luca, Judica, Pecini, Spinelli & Zoccolotti, 2005). The child received a sheet with 50 object drawings of five repeatedly displayed objects. Their task was to cross out as quickly as possible two of the objects.

Visual and auditory order thresholds

The thresholds were obtained with *Brainboy Universal Professional* (Meditech, Germany). Visual order thresholds correspond to the shortest interval necessary to discriminate two visual flashes. The two flashes are emitted by two diodes that are placed 3 cm to the right and left of a fixation point. The participant is given two response buttons (one for the right and one for the left hand). The participant indicates which of the two light flashes occurred first by pressing the corresponding button. The starting ISI was 400 ms. A two-down/one-up staircase procedure established the threshold after three consecutive errors (minimal value 10 ms). To measure auditory order thresholds, two 35-dB SPL noise bursts were presented, one to the participant's left ear and one to his/her right ear (via a Sennheiser MT-70 headset). The starting ISI was 400 ms. The staircase procedure and response modalities were the same as for the visual thresholds.

Speech perception in noise

The stimuli were identical to those used by Ziegler *et al.* (2005). That is, one set of 48 unprocessed Vowel-Consonant-Vowel (VCV) stimuli was recorded. These speech stimuli consisted of three exemplars of 16 possible /aCa/ utterances (C = /p,t,k,b,d,g,f,s,j,m,n,r,l,v,z,j/) read by a French female speaker in a quiet environment. Each signal was digitized via a 16-bit A/D converter at a 44.1 kHz sampling frequency. VCV identification was assessed in silence or noise. In the latter condition, a gated speech-shaped noise masker (i.e. a noise with the long-term power spectrum of running speech) was added to each utterance (and refreshed in each trial of a given session). This speech-shaped noise was either:

- i. stationary (i.e. unmodulated);
- ii. amplitude-modulated (AM) using a sine-wave modulator. The expression describing the sine-wave modulator $m(t)$ was:

$$m(t) = [1 + m \sin(2\pi f_m t + \phi)]n(t) \quad (1)$$

where $n(t)$ represents the speech-shaped noise. Modulation depth m was fixed at 1 (i.e. 100%); modulation frequency f_m was either 4 Hz (slow), 32 Hz or 128 Hz (fast). The starting phase of the modulation ϕ was randomized between 0 and 360° on each trial (for a more detailed description of the stimuli and methods, see Füllgrabe, Berthommier & Lorenzi, 2006).

In each experimental condition, the speech-shaped noise masker was added to each speech utterance at a 0-dB (rms) signal-to-noise ratio (S/N). This S/N ratio was determined in a preliminary experiment so as to yield a consonant identification performance of about 60–70% correct when the speech-shaped noise was steady in control children. In each utterance, signal and noise were of identical duration (mean duration = 648 ms; $SD = 46$ ms). Noise was shaped using a raised-cosine function with 50-ms rise/fall times.

Each stimulus was presented diotically to the listener through headphones (Sennheiser HD 565) and overall levels were calibrated to produce an average output level of 70 dB(A) for continuous speech.

The children were tested individually using a single-interval, 16-alternative procedure without feedback. In each experimental condition (e.g. quiet, stationary noise, etc.), the 48 VCV utterances were presented randomly. All children started with the 'silence' condition. Presentation of the other conditions was counterbalanced. The children were instructed to identify each stimulus. The children gave their responses orally. The experimenter entered the responses by clicking on one of the 16 options on the computer screen.

Perception of envelope-coded speech

In this additional experimental condition, the 48 VCV signals were band-pass filtered using zero-phase, third-order Butterworth filters into 16 adjacent 0.4-oct wide frequency bands spanning the range 80–8,020 Hz. The cutoff frequencies used and technical details regarding stimulus generation are given in Gilbert and Lorenzi (2006). These band-pass filtered signals were then processed in order to remove temporal fine structure information and smear spectral cues (that is, place of excitation on the basilar membrane in the cochlea) by a factor of 2. The envelope was extracted in each frequency band, using the Hilbert transform followed by low-pass filtering with a zero-phase, sixth-order Butterworth filter (cutoff frequency = 64 Hz). The filtered envelope was used to amplitude modulate a sine wave with a frequency equal to the centre frequency of the band, and with random starting phase. The 16 amplitude-modulated sine waves were summed over all frequency bands. All processed stimuli were equalized in terms of global rms value and presented for identification in quiet to listeners.

Table 2 Speech perception performance in quiet, amplitude-modulated (AM) noise, stationary noise, and with tone-excited envelope vocoded speech. Standard deviations in parentheses

	Dyslexics <i>N</i> = 19	CA controls <i>N</i> = 18	RA controls <i>N</i> = 19	Effect size (Cohen's <i>d</i>)	
				Dys – CA	Dys – RA
Silence	99.1 (1.7)	99.4 (.9)	98.7 (1.4)	.21 ns	.25 ns
Noise (average)	68.2 (8.1)	77.4 (6.2)	74.3 (7.4)	1.27***	.78*
4-Hz AM noise	69.2 (7.1)	79.5 (7.1)	74.2 (10.3)	1.44***	.57+
32-Hz AM noise	81.6 (8.5)	86.9 (5.2)	85.3 (9.6)	.75*	.41
128-Hz AM noise	64.6 (12.8)	74.5 (11.8)	70.3 (12.0)	.80*	.46
Stationary noise	57.2 (14.6)	68.5 (10.2)	67.2 (12.6)	.90**	.73*
Envelope-coded	72.6 (8.2)	81.2 (10.6)	77.3 (10.5)	.90**	.50+

Note: Dys = Dyslexics; CA = Chronological age controls; RA = Reading age controls.
 *** $p < .0001$; ** $p < .01$; * $p < .05$; + $p < .10$.

Results

Ancillary cognitive tasks

Performance on all ancillary cognitive tasks (IQ, reading, phonology, memory, attention, visual and auditory thresholds) is presented in Table 1 for dyslexics as well as the two controls groups. The differences between the groups were assessed using independent sample *t*-tests. Effect sizes were calculated using Cohen's *d*. As can be seen in Table 1, the dyslexics had a remarkable deficit in reading regular and irregular words as well as nonwords. The nonword reading deficit was significant with respect to RA controls. The dyslexics also showed strong deficits in phonology, especially in phonological awareness and word repetition (both significant with respect to RA controls). The memory deficit was restricted to digit span. Somewhat weaker but still significant deficits were obtained for visual and auditory thresholds.

Speech perception performance

Table 2 presents mean identification performance (percentage correct) for the different conditions: silence, fluctuating noise (4 Hz, 32 Hz, 128 Hz), stationary noise, and envelope-coded speech signals. For the statistical analyses, the percent correct scores were arcsine transformed (Studebaker, 1985).

Mean comparisons confirmed that there was a clear speech perception deficit in all noise conditions but not in silence (see Table 2 for effect sizes and significance levels). In the average and stationary noise conditions, the speech-perception-in-noise deficit was significant when dyslexics were compared with RA controls. Importantly, we found a significant deficit in the envelope-coded speech condition; this deficit was marginally significant when dyslexics were compared to RA controls.

To assess whether the size of the deficit varied with masker modulation frequency (4 Hz versus 32 Hz versus 128 Hz), we conducted an ANOVA with group (dyslexics versus controls) and modulation frequency as factors. The ANOVA showed a significant effect of group ($F(2,$

53) = 6.40, $p < .01$) and noise modulation frequency ($F(2, 106) = 56.35$, $p < .0001$). However, the interaction between these two effects was not significant ($F < 1$), confirming that the size of the deficit did not change with modulation frequency but was rather stable across the three noise conditions.

Masking release

The effect of masking release was calculated by comparing performance in fluctuating noise with performance in stationary noise. Figure 3 presents the effect of masking release for different noise modulation frequencies for the three groups of participants. For each of the fluctuating noise conditions (4 Hz, 32 Hz, 128 Hz), we conducted an ANOVA with Masking Release (Fluctuating versus Stationary Noise) and Group (Dyslexics versus Controls) as factors. When dyslexics were compared with CA controls, there was a significant Masking Release effect for each noise condition (4 Hz, $F(1, 35) = 35.69$, $p < .0001$; 32 Hz, $F(1, 35) = 101.42$, $p < .0001$; 128 Hz, $F(1, 35) = 11.46$, $p < .01$). More importantly, there was no significant interaction between Masking Release and Group in any of the noise conditions (all F s < 1). Similarly, when dyslexics were compared to RA controls, Masking Release was significant in each condition (4 Hz, $F(1, 36) = 19.10$, $p < .0001$; 32 Hz, $F(1, 36) = 81.57$, $p < .0001$; 128 Hz, $F(1, 36) = 5.86$, $p < .05$). Again, the interaction between Masking Release and Group failed to reach significance in any of the noise conditions (all F s < 1). The absence of a significant interaction between Masking Release and Group confirms that the dyslexics showed a perfectly normal masking release effect both with respect to CA and RA controls. Inspection of Figure 3 shows that, if anything, the masking release effect was bigger for dyslexics than controls.

Phonetic feature transmission

The specific reception of three speech features (voicing, place, manner) was evaluated by information transmission analyses (Miller & Nicely, 1955) that were performed on the basis of individual confusion matrices obtained

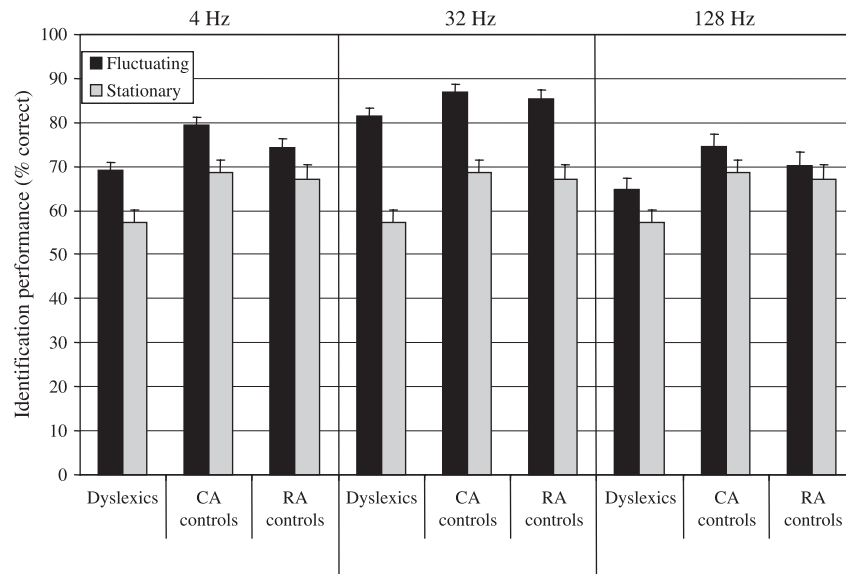


Figure 3 The effect of masking release (i.e. fluctuating versus stationary noise) across the three noise modulation conditions (4 Hz, 32 Hz, 128 Hz) for dyslexics, CA and RA controls. Error bars represent standard errors.

across all noise conditions. In both comparisons, the results were analyzed using $2 \times 3 \times 4$ ANOVAs with group (dyslexics vs. controls), phonetic feature (voicing vs. place vs. manner) and noise condition (stationary, 4 Hz AM noise, 32 Hz AM noise, 128 Hz AM noise) as factors. The results showed a main effect of group (CA-match: $F(1, 35) = 11.76, p < .01$; RA-match: $F(1, 36) = 3.77, p < .06$) and phonetic feature (CA-match: $F(2, 210) = 95.98, p < .0001$; RA-match: $F(2, 216) = 125.01, p < .0001$). The double interaction between group and phonetic feature as well as the triple interaction failed to reach significance in any of the comparisons (all $F_s < 1$). Because of a tendency in the data for a slightly larger deficit for place, we conducted multiple *t*-test comparisons for each feature in each noise condition. The results in the CA comparison showed a significant deficit for *place-of-articulation* in average noise, stationary noise and 4 Hz AM noise (all $p_s < .001$). The deficit for *manner* was significant in average and 4 Hz AM noise. Finally, the deficit for *voicing* was not significant in any of the noise conditions. In the RA comparison, only the *place-of-articulation* deficit was significant and this only in average noise ($p < .05$). Figure 4 presents the reception of speech features in average noise for the CA and RA comparison.

Step-wise regression analyses

An important issue is whether speech-perception-in-noise deficits predict reading impairment beyond variables that are known to affect reading and dyslexia. For this purpose, we conducted six stepwise regression analyses. In each regression, one potentially confounded factor was entered in step 1 of the regression while speech-in-noise performance was entered in step 2. This allowed

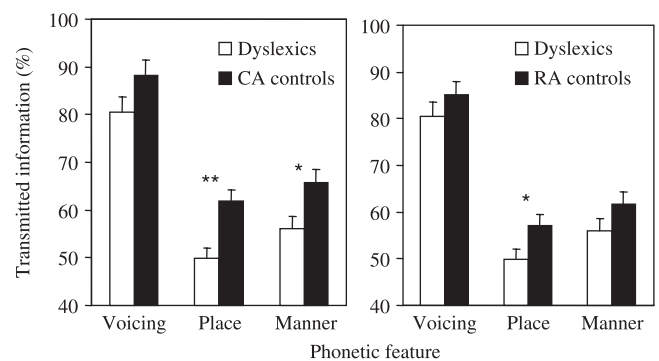


Figure 4 Percentage of transmitted information for different phonetic features (averaged across four noise conditions: stationary, 4 Hz AM noise, 32 Hz AM noise, 128 Hz AM noise). Significance level is given for average noise (*t*-tests). Error bars represent standard errors.

us to calculate the amount of unique variance (R^2 change) accounted for by speech perception after taking into account the following variables: general cognitive ability (IQ-P), sustained attention, low-level auditory perception, verbal memory, phonological output processes, and phonological awareness (see Table 3). Only dyslexics were taken into account in these analyses because (1) lumping together dyslexics and controls might create strong correlations simply because of the absolute differences between the groups (Rosen, 2003) and (2) one of the predictors might interact with group. For each regression, the predicted variables were a composite reading score (regular, irregular and nonword reading combined) and a measure of phonological decoding accuracy. The results are presented in Table 3.

It can be seen in Table 3 that speech-perception-in-noise accounted for a significant amount of unique variance in

Table 3 Stepwise regression analyses of phonological decoding and reading performance. Various control variables are entered in Step 1 while speech-perception-in-noise performance is entered in Step 2

Controlling for	Variables	Decoding			Reading		
		R ²	R ² change	F change	R ²	R ² change	F change
General cognitive ability	Step 1: IQ-P	.016		.27 <i>ns</i>	.015		.25
	Step 2: Speech-in-Noise	.410	.394	10.60**	.366	.351	8.86**
Sustained attention	Step 1: Cancellation	.001		.01 <i>ns</i>	.001		.014
	Step 2: Speech-in-Noise	.441	.441	12.62**	.382	.281	9.87**
Low-level auditory	Step 1: TOJ	.001		.015	.008		.137
	Step 2: Speech-in-Noise	.408	.407	10.99**	.381	.373	9.64**
Short-term memory	Step 1: Digit Span	.154		1.45	.251		2.68+
	Step 2: Speech-in-Noise	.450	.296	8.07**	.473	.222	6.30*
Speech output	Step 1: Word Repetition	.128		2.49	.208		4.46*
	Step 2: Speech-in-Noise	.405	.278	7.46**	.396	.188	4.99*
Phonological awareness	Step 1: Fusion, Deletion	.215		4.66*	.097		1.82
	Step 2: Speech-in-Noise	.625	.410	17.52***	.467	.370	9.64**

Note: TOJ = Temporal Order Judgments.

each regression analysis, that is, after partialling out general cognitive ability, sustained attention, low-level auditory perception, verbal memory, phonological output processes, and phonological awareness. The amount of unique variance accounted for varied between 28% and 44% in phonological decoding and between 18% and 37% in reading. The maximal amount of variance accounted for was obtained when phonological awareness and speech-perception-in-noise were taken together to predict phonological decoding (63% of the variance). It is interesting to note that speech-perception-in-noise explained unique variance even after entering phonological awareness in step 1 of the regression. This suggests that the link between speech perception and dyslexia is not only via impoverished phonological awareness skills.

Speech-perception-in-noise accounted for unique variance even after controlling for phonological output deficits, that is, after entering repetition performance of complex words in step 1 of the regression. This is an important result because our speech-perception-in-noise task contained a phonological output component (i.e. participants were asked to say aloud the perceived syllable). The fact that speech-perception-in-noise still accounted for unique variance rules out the possibility that deficits were due to phonological output processes. To further strengthen this claim, we ran a post-hoc analysis using a subset of our dyslexics who did not show any word repetition deficits ($n = 10$) and a group of matched CA and RA controls ($n = 10$, respectively). In this post-hoc analysis, there was no significant difference in word repetition between the three groups (95%, 97%, and 95% for dyslexics, CA controls and RA controls, respectively, $p_s > .20$). Yet, the speech-perception-in-noise deficit was still significant in both comparisons. For example, in the average noise condition, the subset of dyslexics achieved 69% correct, whereas the matched CA and RA controls achieved 80% and 77%, respectively. The deficit was significant in both comparisons ($t(1, 18) = 4.8$, $p < .0001$ and $t(1, 18) = 3.1$, $p < .001$, respectively).

General discussion

The present study found clear answers to the five questions raised in the Introduction. These will be discussed in the following sections along with a discussion of the link between speech-perception-in-noise deficits and reading.

Is there evidence for a speech-perception-in-noise deficit in dyslexia?

Our results show a very clear speech-perception-in noise deficit for children with DD. The present study therefore replicates the finding of Ziegler *et al.* (2005) who studied speech-perception-in-noise deficits in children with SLI. As in the previous study, no speech perception deficit was obtained in silence, whereas clear speech perception deficits occurred in noise. In our view, some of the previous studies might have found rather 'fragile' or 'weak' speech perception deficits (e.g. Adlard & Hazan, 1998; Blomert & Mitterer, 2004; Brandt & Rosen, 1980) because they presented speech in optimal quiet conditions. Such conditions allow the listener to rely on a great number of different and partially redundant speech cues. In contrast, noise makes many of these spectral and temporal speech cues less reliable (Assman & Summerfield, 2004). Thus, only the successful integration of a large number of different cues will allow the listener to recover from the noise. We argue that the simultaneous integration of various speech cues required for robust speech identification is deficient in children with dyslexia (see below for further discussion).

One important caveat is that our task required participants to verbalize the perceived syllables and therefore deficits might be due to phonological output processes rather than perception. This is a serious concern because our dyslexic children showed impaired performance in the repetition of complex words. Thus, it could be possible that their apparent speech perception deficits reflect phonological output deficits. However, there are

several elements that allow us to rule out the possibility that their deficits are due to phonological output rather than perception. First, if the deficit came from phonological output processes, we should see identical deficits across all conditions (including silence) because verbal output was strictly identical across all conditions. However, this was not the case because no deficit was obtained in silence. Second, if the deficit came from phonological output processes, speech-perception-in-noise should not explain any unique variance after partialling out repetition of complex words. However, speech-perception-in-noise explained about 20% of unique variance even after partialling out the repetition of complex words. Finally, in a post-hoc analysis on a subset of 10 dyslexics without repetition deficits, a significant speech-perception-in-noise deficit was found both in comparison with CA and RA controls.

Speech-perception-in-noise deficits – cause or consequence?

To what extent could speech-perception-in-noise deficits be a consequence rather than a cause of dyslexia? It is possible that in the course of phonological development learning to read and write has the effect of stabilizing possibly noisy phoneme representations (Ziegler & Muneaux, 2007). Indeed, we know that reading development is the major force driving phoneme awareness (Morais, Cary, Alegria & Bertelson, 1979). Thus it could be possible that the reading deficit causes the speech-perception-in-noise deficit rather than the other way around. However, in our study, speech-perception-in-noise deficits were obtained in some conditions even when the performance of dyslexics was compared to that of much younger children matched on reading age (for the importance of that comparison, see Goswami, 2003). This suggests that the speech-perception-in-noise deficit is not simply the consequence of the dyslexics' poor reading ability.

Are some phonetic features more affected than others?

An intriguing question is whether all phonetic features are affected similarly in DD or whether some are more affected than others. Most previous studies investigated only a very limited number of contrasts. For example, Lieberman *et al.* (1985) found deficits in the identification of place-of-articulation (/b-/d-/g/) but they did not measure any other contrasts (see also Godfrey *et al.*, 1981).

The advantage of the present paradigm is that speech perception was obtained for all consonants. Indeed, information transmission analyses (Miller & Nicely, 1955) allowed us to investigate the specific reception of three speech features (voicing, place, manner). Using the exact same procedure and stimuli, Ziegler *et al.* (2005) reported significantly stronger deficits for voicing than for place-of-articulation or manner in children with SLI.

In contrast, in our study, the reception of the three consonant features was impaired but voicing was no more impaired than the other features. If anything, place-of-articulation seemed to cause greater difficulties than the other features. This finding suggests fundamental differences in the nature of the phonological deficit between dyslexia and SLI. While dyslexics seem to have greater difficulties in the domain of place of articulation, children with SLI seem to have greater difficulties in the domain of voicing.

External noise: necessary or simply sufficient?

One intriguing hypothesis is that DD might result from a noise exclusion deficit (Sperling *et al.*, 2005, 2006). This hypothesis has been tested in the visual domain. More precisely, Sperling *et al.* (2005) noted that previous studies that found magnocellular deficits to be associated with DD tended to use noisy displays. Sperling *et al.* (2005) predicted that if noise exclusion deficits were at the origin of impaired processing of the magnocellular channel, then a similar deficit should be found in the parvocellular channel for noisy conditions and no deficits should be found in either channel in the absence of noise. The results indeed showed that noise was a necessary condition for the visual deficits to occur.

Our finding of a speech perception deficit that is present in noise but absent in silence is highly consistent with the idea of a general deficit in noise exclusion. We further investigated whether *external* noise was a necessary condition for speech perception deficits to occur. For this purpose, the speech signal was degraded not by the addition of background noise but by eliminating temporal fine-structure cues and smearing spectral cues while leaving the temporal envelope of the speech signal intact (Lorenzi, Gilbert *et al.*, 2006). Note that this manipulation degrades the speech signal without using external noise. The results showed clear deficits in this condition that were comparable to those obtained in noisy conditions. Although external noise might be the most frequent cause of stimulus degradation in real life, external noise does not seem to be a necessary condition for the occurrence of speech perception deficits. Note, however, from the point of view of noise exclusion theory (Doshier & Lu, 2000) it does not make a huge difference whether the source of the degradation is external or internal noise.

Beyond low-level auditory, memory or attention deficits

One important question is whether speech perception deficits are domain-specific or whether they result from general deficits in cognitive ability (nonverbal IQ), auditory perception, sustained attention, verbal production or verbal memory. To address this issue, we conducted step-wise regression analyses in which these different variables were entered in step 1 of the regression models.

The results showed that speech-perception-in-noise explained a significant amount of unique variance even after controlling for each of these potentially confounding factors. Thus, speech-perception-in-noise predicts reading skills beyond the contributions of low-level auditory, memory, production or attention skills.

More importantly, our dyslexics showed a perfectly normal (sometimes even slightly superior) masking release effect. Masking release reflects the ability of the auditory system to extract speech cues in the ‘dips’ or ‘valleys’ of fluctuating noise (Füllgrabe *et al.*, 2006) where signal-to-noise ratio peaks. A substantial and positive masking release effect indicates that temporal and spectral resolution are relatively spared – indeed patients with sensorineural hearing loss following cochlear damage show reduced or abolished masking release (Bacon, Opie & Montoya, 1998; Gustafsson & Arlinger, 1994; Peters *et al.*, 1998). Thus, normal masking release not only rules out a peripheral locus of the deficit but also suggests that the speech-perception-in-noise deficit is not due to poorer-than-normal spectral or temporal resolution (Tallal, 1980) or poorer-than-normal AM sensitivity (e.g. Lorenzi, Dumont & Füllgrabe, 2000; Rocheron, Lorenzi, Füllgrabe & Dumont, 2002). Note also that our dyslexics showed a normal masking release effect in rapidly fluctuating noise (128 Hz) as well as in slowly fluctuating noise (4 Hz). This finding seems again inconsistent with the rapid temporal processing deficit hypothesis. Altogether then, our finding of a normal masking release effect suggests that the auditory system of children with dyslexia is able to encode and use acoustic information quite well (i.e. temporal envelope, periodicity, temporal fine structure and spectral cues). The problem seems to arise in noise when a substantial portion of the speech cues is no longer available. This clearly points to a lack of speech robustness in noisy conditions.

Nature of speech perception deficits and their link to reading

Two key questions need to be addressed. What is the origin of the speech perception deficit and what is its link with reading and dyslexia? On the one hand, we can be fairly confident that the speech-perception-in-noise deficit is not due to poor spectral and/or temporal resolution because masking release was fully intact and because speech-perception-in-noise explained significant unique variance in reading even after partialling out low-level auditory perception. On the other hand, intact masking release also suggests that the core problem does not lie in impoverished or underspecified phonological representations. If phonological representations were impoverished or underspecified, phoneme restoration should be deficient, which would certainly reduce the size of the masking release effect. A similar conclusion has been put forward by Blomert, Mitterer and Paffen (2004) who found normal context compensation processes

suggesting that phonological representations are intact. Similarly, Serniclaes and colleagues (2004) reported that phonological representations of dyslexic children are, if anything, over-specified (allophonic) rather than under-specified.

Thus, if the deficits are neither due to poor temporal or spectral resolution nor to impoverished phonological representations, then the deficits must arise somewhere in between these two levels, most likely in the mapping of acoustic features onto phonological categories. As Blomert *et al.* (2004) pointed out, ‘this [mapping] process implies more than a simple decision rule (e.g. if VOT greater than –20 ms, then /b/) but rather the application of a multidimensional nonlinear function to a multidimensional stimulus pattern’ (p. 1031). The complexity of this process is amplified in noise. Speech-perception-in-noise can be compared to a process of ‘hyper-triangulation’ in an n -dimensional space through time, where n is likely to exceed 50 (Greenberg, 1997). Thus speech-perception-in-noise requires the simultaneous integration of various speech cues across different time scales. When noise distorts partially redundant cues, the integration process is pushed to its limit. Thus, we argue that the core deficit of the dyslexics seems to reside in their poor ability to simultaneously integrate various speech cues across different time scales. This leads to poor access of phonological representations and a lack of speech robustness in noisy conditions. This overall conclusion is strikingly similar to that of Nittrouer (1999) who argued that children with dyslexia do not integrate speech cues properly to achieve phonological categorizations.

The second question that needs to be addressed is why and how these speech-perception-in-noise deficits lead to dyslexia. The first possibility is rather straightforward. Learning to read is based on mapping orthography onto phonology, a process which is also referred to as phonological recoding (Share, 1995). In particular, the child needs to learn grapheme–phoneme correspondences (Hutzler, Ziegler, Perry, Wimmer & Zorzi, 2004). In the learning situation, phonemes are typically provided by the teacher in real classroom conditions. If children cannot perceive stable phoneme categories under noisy conditions, whether this is due to a general noise exclusion deficit or a specifically phonological problem, the learning of grapheme–phoneme correspondences will be extremely difficult, thus hampering the development of an orthographic lexicon. Note that noise in the classroom is not an exception but the rule. Indeed, the average noise in a primary school classroom is about 72 dB(A), which is enough to reduce speech intelligibility by over 50% (see Jamieson, Kranjc, Yu & Hodgetts, 2004).

A second possibility for the link between speech perception and dyslexia is based on the idea that speech-perception-in-noise and reading require similar fundamental cognitive and neural processes. One such process is focused attention because focused attention is

needed to filter out noise from the signal as well as to map graphemes onto phonemes (Facoetti, Zorzi, Cestnick, Lorusso, Moltenia, Paganoni, Umiltà & Mascetti, 2006; Perry, Ziegler & Zorzi, 2007). Another fundamental mechanism would be the integration of information across various time scales. Indeed, speech-perception-in-noise requires simultaneous integration of different acoustic cues across various time scales. Similarly, fluent reading requires the simultaneous integration of letters and letter clusters (Hawelka & Wimmer, 2005) and the mapping of these units onto a phonological and semantic code (Harm & Seidenberg, 1999). The common denominator between speech-perception-in-noise and reading might be the necessity to simultaneously integrate different types of information across various grain sizes and time scales, a process that is pushed to its limit in the presence of noise.

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