Impaired processing of rapid stimulus sequences in dyslexia

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Apart from their reading difficulties, dyslexic subjects often suffer from a variety of subtle sensory and motor deficits. Whether these deficits have a causal relationship to the reading disorder, form additional risk factors, or are totally independent of the reading problem, is under vivid debate. In this article, we review the evidence and suggest that 'sluggish attentional shifting' (SAS) can account for the impaired processing of rapid stimulus sequences in dyslexia. Within this novel framework attention-related prolongation of input chunks is decisive for many small deficits found in dyslexic subjects.

> 'It might seem impossible for you to convey that time doesn't always march in one direction, bringing with it a perfectly ordered sequence of causes and effects, but it is not hard for me to imagine it, because I'm dyslexic. When I hear a phone number spoken quickly, I hear all the numbers but don't have any impression in what order they were spoken. It's as if they came in all at once.'
> Scott Adams in *The Dilbert Future* (2000), Boxtree, p. 233.

This description from the famous cartoonist fits nicely with findings on dyslexic adults who seem to have a prolonged 'cognitive window' (or 'time or input chunk'[1]) within which the temporal order of successive items is easily confused[2–4]. In this article, we review the support for our hypothesis that chunk duration is prolonged because of sluggish attentional shifting (SAS) and that the prolongation may distort proper development of cortical representations that are essential for reading acquisition.

We start with a brief introduction to dyslexia, then review a series of behavioral experiments and current thinking on rapid sequence processing, attentional dwell time and visual mineneglect in dyslexia. Finally, we formulate the SAS hypothesis and discuss how it fits with other data in the field. We also suggest some new experiments to test the SAS theory.

Developmental dyslexia

Developmental dyslexia is a common neurocognitive disorder with genetic predisposition: approximately 4–10% of the population have difficulties in learning to read, despite adequate training, opportunity, and normal intelligence[5]. Dyslexic subjects fail to acquire adequate phonological skills that are needed when written letters are translated to sounds and words[6].

This interpretation of processing in dyslexia was first presented by Riitta Hari in her Curt von Euler Honorary Lecture in Cognitive and Neurobiological Aspects of Reading and Language Development (Rodin Academy Dyslexia Meeting, Riken, Japan, April 1999).

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However, they often also suffer from various sensory problems beyond the skills directly needed for reading[7–9]. The significance of these sensory-related problems for the genesis of dyslexia has remained unclear: they could have a causal role, form additional risk factors, or be totally unrelated to the reading impairment.

Tallal and Piercy[10,11] were the first to observe that language-learning impaired (LLI) children are inferior to control subjects in discriminating the sequence order of two tones of different frequencies presented at interstimulus intervals of less than 400 ms. Similar observations have been made in reading-disabled children[12,13], and the processing deficit has been shown to extend to adults and to stimuli of other sensory modalities[3,4,14,15]. We refer to these findings as deficits of processing rapid stimulus sequences to emphasize their relationship to distinct stimuli or distinct changes within a stimulus sequence; thus rapid stimulus sequence (RSS) processing does not include direct sensory deficits such as perception of auditory spectral pitch.

Dyslexic subjects can also have trouble in balance, motor control and muscle tone, probably because of cerebellar affision[16]. Moreover, dyslexics often display various visual abnormalities that are not directly related to RSS processing[7,17]. It has been suggested that the variable sensory symptoms derive from a disorder in the magnocellular neural pathways[7,18,19]. This 'M-deficit' is proposed to arise in the early life of genetically predisposed subjects[20], possibly as a result of immunoreactive damage of cell-surface substances common to magnocells[19,21]. Such a pathophysiological mechanism could lead to a variety of symptoms, depending on the most affected neuronal system.

Despite extensive studies of large subject groups, the relationship between dyslexia and the observed neuronal, behavioral and psychophysiological deficits has remained poorly understood and heavily debated. The most contentious issue is the possible causal link from the auditory low-level disorders to reading impairment, suggested to occur via secondary phonological or speech perception deficits[7,9,22–28].

Problems with rapid stimulus sequence processing

Accurate timing up to tens or hundreds of milliseconds is important for brain functions underlying percepts, movements, speech and learning. Adding to the extensive evidence on RSS processing deficits in LLI

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Box 1. Auditory saltation

The auditory saltation illusion[a] was designed as the auditory analogue of the tactile illusion 'cutaneous rabbit'[b]. Figure Ia shows the stimuli schematically. In a train of eight binaural 1-ms clicks, four left-ear leading clicks are followed by four right-ear leading ones; in all stimuli the interaural time differences are 0.8 ms. When presented in isolation, the binaural clicks are perceived as lateralized either to the left or to the right side. Thus at interstimulus intervals (ISIs) of 500 ms, the subject perceives four left-sided clicks followed by four right-sided clicks (Fig. Ib, right). However, when the ISI is shortened to 30 ms (Fig. Ib, left), a saltatory percept emerges, with the sounds appearing to jump from left to right at equidistant steps. At the intermediate ISIs, saltation is perceived but with larger jumps across the midline than between the other sounds of the train. It is enough to add just a single right-ear leading click after the four left-ear leading clicks to make the whole stimulus sequence appear to jump[a].

The strength of the illusion at different ISIs, quantified by measuring the size of the perceived jump from the fourth to the fifth click of the train, indicates (Fig. II) that dyslexic adults perceive the saltation at significantly longer ISIs than normal readers[c].

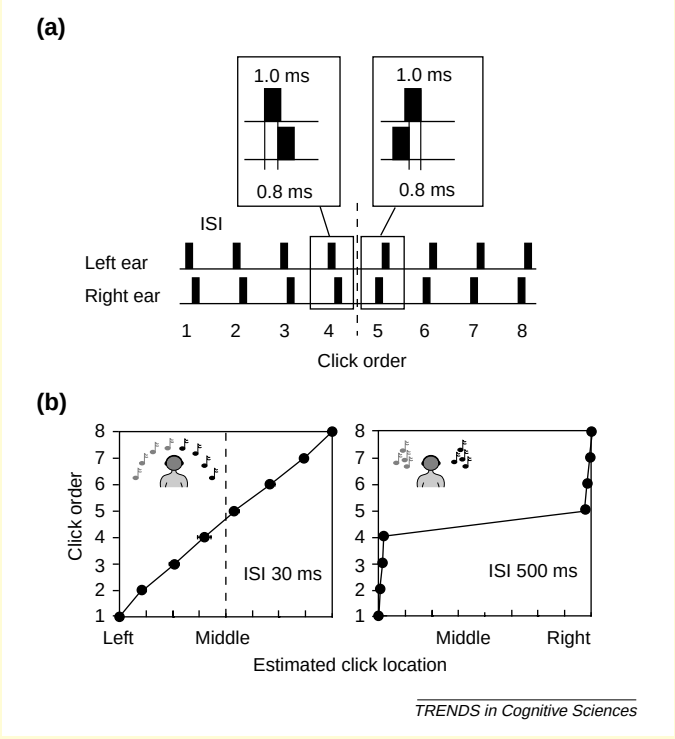

Fig. I. (a) Schematic presentation of the sounds in the auditory illusion experiment. The time interval between the successive clicks varied in different trains. (b) Estimates (mean ± SEM; 14 normal-reading subjects) of the click positions at interstimulus intervals (ISI) of 30 ms (left) and 500 ms (right). The order of the clicks, from 1 to 8, is given on the y-axis, and the perceived position of the click on a normalized left–right axis on the x-axis.

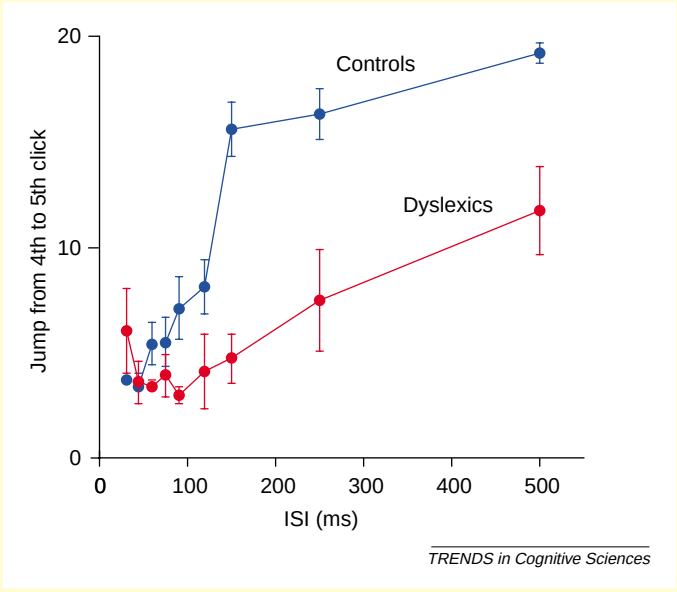

Fig. II. Mean (±SEM) size of the 'spatial jump' from the fourth to the fifth click as a function of the interstimulus interval (ISI) in the train. Data are given for 20 normal reading control subjects (blue) and 10 dyslexic individuals (red). The total left–right distance was coded as 20, and for a totally linear saltation each jump would be 2.5.

References

a Hari, R. (1995) Illusory directional hearing in humans. *Neurosci. Lett.* 189, 29–30

b Geldard, F. and Sherrick, C. (1972) The cutaneous 'rabbit': a perceptual illusion. *Science* 178, 178–179

c Hari, R. and Kiesilä, P. (1996) Deficit of temporal auditory processing in dyslexic adults. *Neurosci. Lett.* 205, 138–140

and dyslexic individuals, we have observed that adults dyslexic in Finnish, a language with transparent orthography, are significantly impaired in auditory RSS processing. For example, dyslexic subjects perceived an illusory auditory saltation[29] at significantly longer intersound intervals than normal readers[3] (Box 1), suggesting a prolonged 'cognitive integration window' within which percepts of successive sounds can interfere. It is important to note that the auditory illusion task does not require any verbal labeling of the stimuli, a frequent criticism of studies of temporal order judgment[28].

Similarly, the dyslexic adults needed significantly longer intersound intervals than normal readers in a pitch streaming task[4] to be able to follow each successive sound in a continuous fashion, without stream segregation (Box 2). These results agree with sluggish RSS processing and prolonged input chunks. Auditory masking studies support prolonged auditory input chunks in LLI children[30].

RSS processing disorders are also evident in other senses. For example, Laasonen *et al.*[14,15], using trains of brief auditory, visual and tactile stimuli, observed decreased segregation acuity in all modalities, both in dyslexic children and adults, and crossmodal segregation times were especially clearly prolonged.

Dyslexics are slow – but at what level?

The above data emphasize the pan-sensory nature of RSS processing impairments faced by dyslexic

Box 2. Pitch streaming

In an auditory pitch streaming sequence[a], high and low tones are presented alternately. When the sounds are temporally far apart, a continuous sequence of high-low-high-low… tones can be heard (Fig. Ia). However, when the interval between the sounds is shortened, the streams segregate and the listener perceives two separate streams: high-high-high… and low-low-low… (Fig. Ib). A similar segregation can also be obtained by increasing the pitch difference.

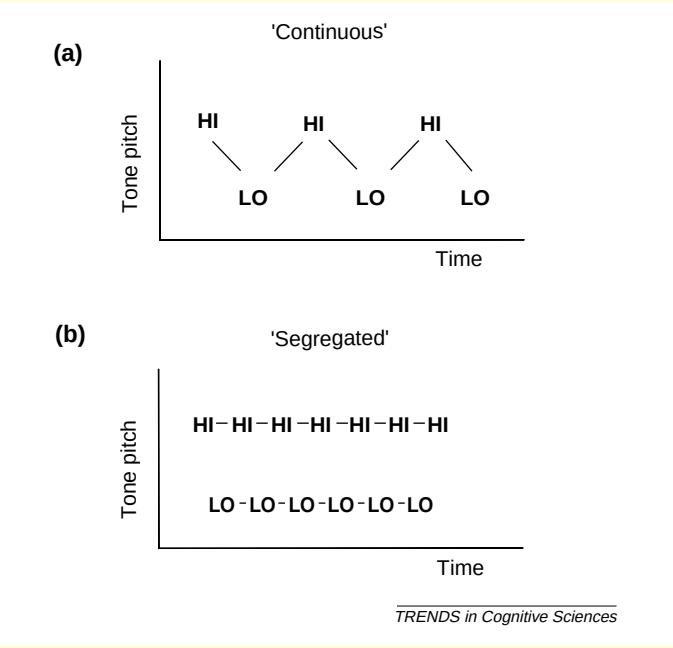

Fig. I. Principle of auditory pitch streaming (see text for explanation.)

The segregation threshold for the time interval between tones in dyslexic adults was almost double that of control subjects[b] (Fig. II). The segregation threshold of the dyslexic individuals correlated significantly with the speed of naming of colors, digits and letters.

References

a Bregman, A. (1990) *Auditory Scene Analysis*, MIT Press

b Helenius, P. *et al.* (1999) Auditory stream segregation in dyslexic adults. *Brain* 122, 907–913

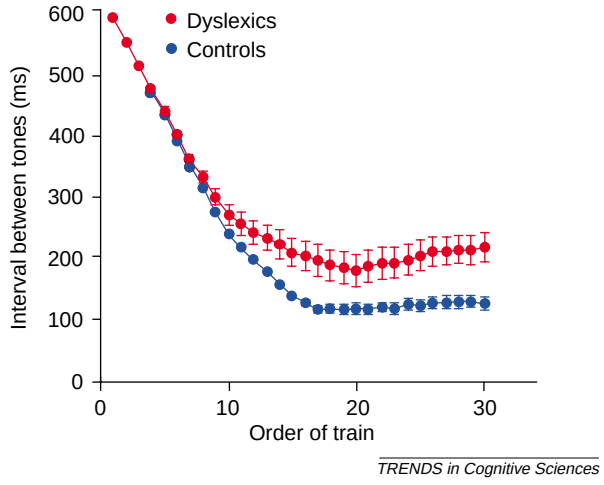

Fig. II. Performance of 18 normal-reading control subjects (blue) and 13 dyslexic adults (red) in the auditory stream segregation task. The horizontal axis refers to the number of trials in which the time interval between the sounds was modified in order to determine the segregation threshold.

individuals. One is thus tempted to search for the possible core deficit at a very basic neuronal level[31]. Rather surprisingly, however, dyslexics perceive interaural time differences of less than 1 ms in a normal manner, as is illustrated by their accurate detection of interaural phase modulations[32] and of interaural temporal cues[33]. Moreover, although dyslexic adults discriminate both spectral and periodicity pitch stimuli less accurately than control subjects, their performance is not relatively worse in the periodicity pitch task[33], which relies on temporal stimulus features only. Thus the neuronal circuitries of dyslexic individuals seem to react to sounds with normal phase locking, and the mechanism for impaired RSS processing has to be searched for at some other level, which we propose to be related to triggering of automatic attention.

Attentional dwell time and dyslexia

The auditory pitch streaming segregation thresholds can be related to the speed at which the 'mind's ear' is able to follow successive stimuli between frequency channels, so that the results of dyslexic subjects could reflect sluggish shifting of auditory attention. Such an interpretation is consistent with the prolonged visual attentional blink in dyslexic adults[34] (Box 3). Prolongations of attentional dwell time could naturally slow down the speed of RSS processing, and the attentional capacity limitations could, in part, be modality-specific[35].

Interestingly, hemineglect patients suffering from right parietal lobe damage display prominent prolongations of attentional blink, of up to 400% of that in healthy subjects[36]. Therefore our recent studies have tested whether dyslexics with a relatively minor (30%) prolongation of the attentional blink could suffer from a 'minineglect'; a mild form of parietal-lobe hypofunction.

Visual minineglect

Two visual tasks (Box 4) were applied to test the minineglect hypothesis[37]. The temporal order judgment between visual hemifields was adopted because it has previously demonstrated abnormal right-hemifield preference in neglect patients[38]. The line motion illusion[39] was applied to test whether dyslexics have difficulties in their automatic attentional capture[40]. Neither task required motor responses, and so possible motor clumsiness did not affect the results.

Box 3. Attentional blink

An 'attentional blink' can be measured with a dual task procedure consisting of two targets – the first one to be identified and the second to be detected[a]. For 400–600 ms after the first target the subject is transiently 'blind' to other targets; this 'attentional dwell time' is probably caused by capacity limitation[a].

Figure Ia shows an attentional blink setup[b]. Letters were presented on a screen once every 107 ms and the subject had to: (i) identify the white letter, and (ii) detect and report whether letter X followed it or not (X was present in two thirds of the trials).

In both groups the percentage of detected X letters was very low immediately after the first target (Fig. Ib). In the control group the 75% performance level was obtained at 540 ms, and this was taken as the duration of the attentional blink. In dyslexics, the attentional blink was prolonged up to 700 ms. Both groups correctly reported 'no X' during more than 90% of the 'non-X' trials, indicating equal ability to sustain a general level of attention during the task. Although dyslexic readers might have longer letter recognition times, the attentional blink reflects the difference between two recognition times and thus should not depend on the recognition time of a single letter.

References

a Duncan, J. *et al.* (1994) Direct measurement of attentional dwell time in human vision. *Nature* 369, 313–315

b Hari, R. *et al.* (1999) Prolonged attentional dwell time in dyslexic adults. *Neurosci. Lett.* 271, 202–204

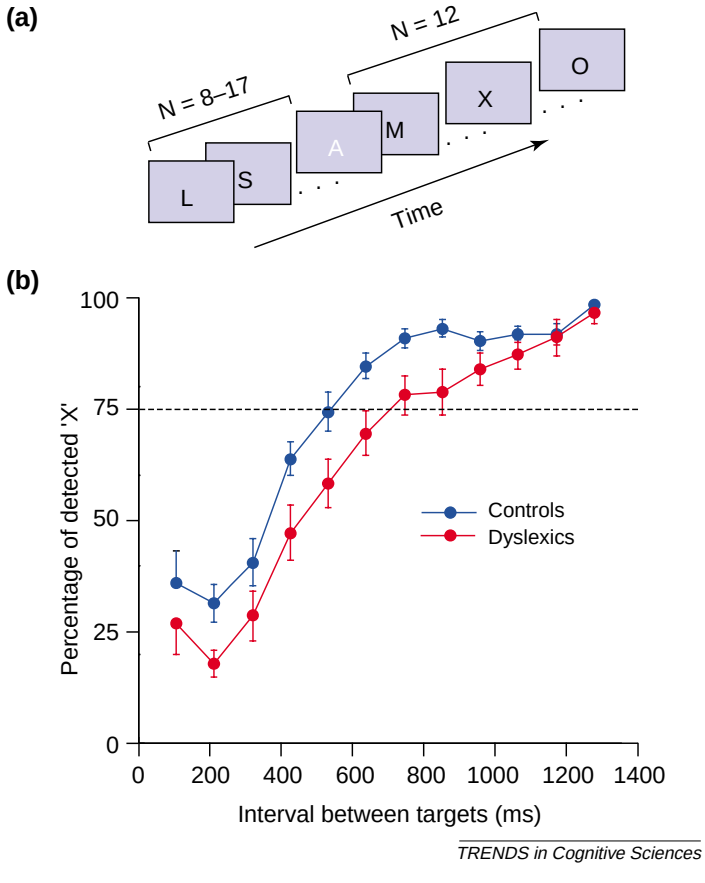

Fig. I. (a) An example of stimuli in a single trial during the attentional blink task. (b)The performance (mean ± SEM) of 22 control subjects (blue) and 18 dyslexic individuals (red) in detecting the target 'X' after correct identification of the white letter at different intervals between the targets. Dyslexics required a longer interval in order to reach a 75% performance criterion.

The dyslexic subjects differed consistently in both tasks from normal readers (Box 4), showing slower processing of stimuli in the left than the right visual hemifield, in agreement with the left minineglect hypothesis. In addition, attentional capture was sluggish in both hemifields. Figure 1 illustrates schematically how such differences might have arisen. Bilaterally sluggish processing would spread the response curve at both ends, and a left minineglect would shift the whole curve towards the left (Fig. 1a). The measured data resemble the simultaneous action of both effects (Fig. 1b).

The observed minineglect is so mild that it is unlikely to cause any significant direct effects on reading, but rather serves as an indicator of an underlying attentional problem. These results are in line with reports that children with reading difficulties and unstable vergence control make more errors in locating targets in the left than the right visual hemifield[41,42]. Furthermore, dyslexic children display left inattention in visual flanker and cue-target reaction time tasks, associated with over-distractability in the right visual field[43,44]. They also seem to distribute their attentional resources more diffusely than normal readers, probably because of difficulties in narrowing the attentional focus[45].

M-deficit, parietal lobe, and reading

Because right parietal-lobe lesions may result in left neglect, a minor deficit of the right parietal lobe could underlie the left minineglect in dyslexia[37]. One possible causal link is the decreased magnocellular input to the dorsal visual stream[17,46]. Even a balanced weakening of both parietal lobes could lead to right-hemisphere symptoms, because of the importance of the right intraparietal sulcus region for capacity-limited attentional processing, evident from brain imaging during an attentional blink task[47].

In general, data from robust right parietal dysfunctions are concordant with the presence of minineglect in dyslexic subjects. For example, lesions of the posterior parietal lobe can produce 'acquired dyslexia' with, for example, letter omissions and letter naming errors during reading[48], and right hemisphere injections of sodium amobarbital result in reading errors resembling those in dyslexia[49]. Interestingly, children with attention deficit hyperactivity disorder (ADHD) have been suggested to suffer from hypoarousal of the right parietal lobe[50]; significant co-morbidity exists between dyslexia and ADHD.

When the subjects are learning to read, they also have to learn rapid attentional shifts along the lines of text, to allow accurate targeting of rapid eye movements[51]. Covert attention and saccade control involve activation of common areas in the parietal, frontal and temporal lobes[52], and these two functions are closely interrelated during perception: one first has to shift attention to the target location before a saccade can be made towards it[53]. Thus

Box 4. Temporal processing between visual hemifields

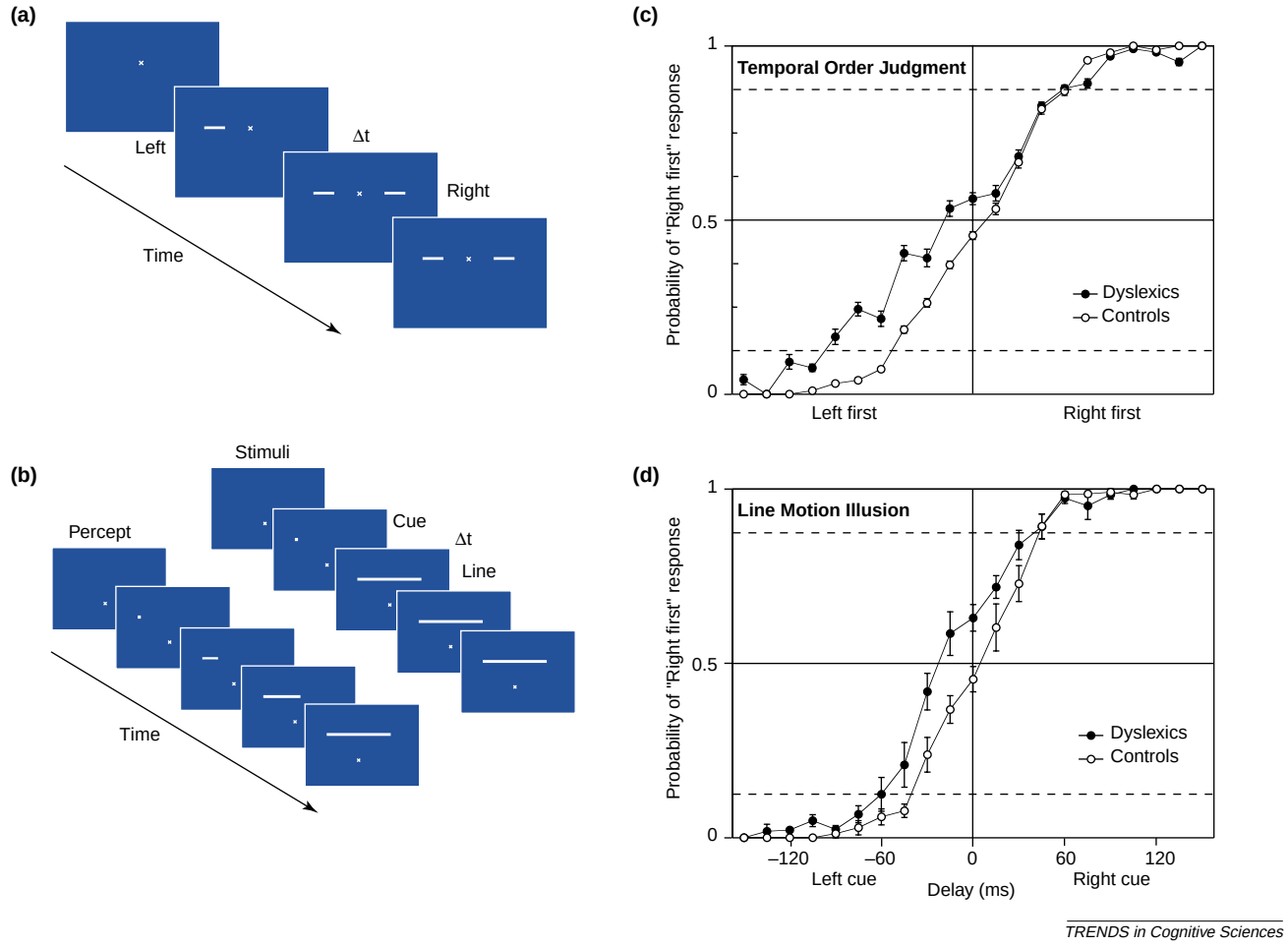

Fig. I. (a) Set-up for the visual temporal-order judgment task. (b) Set up for the line motion illusion task. Rather low luminances were applied to sensitize the tests to possible deficits of magnocellular visual pathways. (c)The mean (± SEM) responses of nine dyslexic and 14 normal-reading subjects in the temporal-order judgment task. The perceptual judgments, given as the probability of answering 'right first', presented as a function of the time delay between the left- and right-sided bars; the 0.5 probability level of the perceptual judgment axis refers to equal numbers of 'left first' and 'right first' responses.The negative and positive time delays refer to left- and right-sided stimulus precedence, respectively. (d) The corresponding data from nine dyslexic and 14 normal reading subjects in the line motion illusion task. In both tasks, the response distributions of dyslexic subjects were biased towards 'right first' responses.

The following two tasks were used to find out whether dyslexic subjects differ from normal readers in their preference for the left or right visual field. In the visual temporal-order judgment task[a] (Fig. Ia) the subjects indicated verbally whether a visual bar in the left (or right) hemifield preceded a similar bar on the right (or left); in this task the delay between the bars varied randomly from 0 to 210 ms, and either the left or the right bar appeared first in a random fashion.

In the line motion illusion task[b] (Fig. Ib) the subject illusorily perceives a line growing from a site where a cue stimulus has been presented slightly earlier.The illusion is interpreted to reflect faster processing of stimuli falling into the cued, and therefore attended, locations[b]. It is assumed that especially stimuli relying on the M-pathways capture transient automatic attention[c] and speed up sensory processing at the site of attentional focus.

Results from both tasks are illustrated in Fig. 1c and d.The control subjects performed symmetrically for both stimulus orders. In both tasks, the response distributions of dyslexic subjects were biased towards 'right first' responses so that the response curve centered significantly left from zero, indicating advantage for the right visual field[d]. Furthermore, in the temporal-order judgment task the 'simultaneity window', derived from the 75%–25% width of the distribution, was statistically significantly prolonged in dyslexic individuals compared with that of the controls, indicating increased sluggishness of temporal processing; a similar but statistically non-significant trend was seen for the line motion illusion.Thus dyslexic and normal readers differed consistently both in judging the temporal order of visual stimuli presented to the two hemifields and in perceiving a line growing from left to right or vice versa in the line motion illusion task.

References

a Robertson, I. *et al.* (1998) Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. *Nature* 395, 169–172
b Hikosaka, O. *et al.* (1993) Visual attention revealed by an illusion of motion. *Neurosci. Res.* 18, 11–18
c Steinman, B.A. *et al.* (1997) Transient visual attention is dominated by the magnocellular stream. *Vis. Res.* 37, 17–23
d Hari, R. *et al.* (2001) Left mineneglect in dyslexic adults. *Brain* 124, 1373–1380

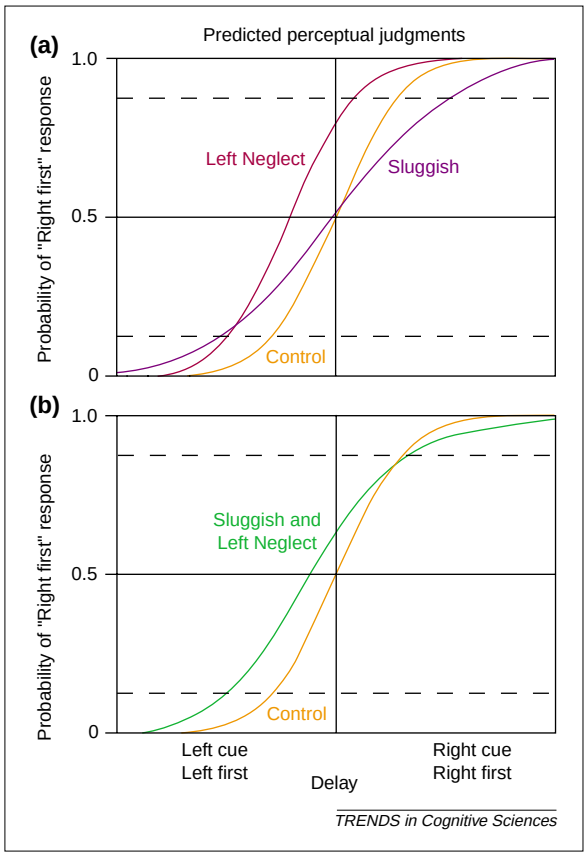

Fig. 1. A schematic illustration of predicted results in the temporal order judgment and temporal motion illusion tasks. (a) Compared with the control subjects' symmetric response curve (orange), sluggish attentional capture (purple) would spread the curve at both ends so that the 75% performance level (within the dashed lines) would be achieved at longer time intervals than in the control subjects. Left-sided minineglect (red) would shift the curve towards the left because left-sided stimuli would need longer processing time; otherwise the shape of the curve would remain the same as that for the control group. (b) Combination of both sluggish attentional capture and minineglect (green) would give results similar to those obtained for dyslexic individuals (see Fig. Id in Box 4).

attention shift can be the prerequisite for proper sensory sampling.

SAS theory of dyslexia

General

We now propose SAS as the pathophysiological link between the magnocellular deficit and the impaired RSS processing in dyslexic subjects. The attention of dyslexic subjects, once engaged, cannot easily disengage, and vice versa; this all takes time. The sluggish attentional capture and prolonged attentional dwell time would thus impair processing of RSSs. In agreement with experimental data, the SAS theory predicts that RSS processing deficits should be seen in all sensory modalities. SAS would also prolong sensory input chunks, thereby degrading cortical representations essential for fluent reading. These steps will be discussed in more detail below.

Within the SAS framework the same core deficit in the M-system could have independent effects at various processing levels, and all these effects – direct sensory impairments, attention-related RSS processing deficits, and prolonged input chunks – could contribute to the reading impairment, thereby forming a cascade of causal factors.

Time chunks and cortical representations

The concept of input (or time) chunks is an essential part of our SAS theory. We suggest that the input chunks are prolonged in dyslexic subjects because of sluggish attentional shifts. The chunking could be a result of modality-specific attentional capacity limits[35], but additional global limitations are likely to exist. For example, lesions of the parietal cortex, known to have crucial effects on visuospatial attention, can produce visual, tactile and auditory neglect[54].

Phonological awareness, essential for reading acquisition[6], needs stable and invariant phonological representations in the brain. Tallal *et al.*[22] suggested that language- and reading-impaired children, because of their basic auditory temporal processing deficit, are unable to establish stable and invariant phonemic representations, and that the auditory processing deficits are thereby sufficient to disrupt reading development.

If we consider phonemic maps to be formed according to self-organizing principles of neural networks[55,56], the general conclusion drawn from the modeling studies is that shorter and less complex input chunks are always organized better than the longer ones, which would need higher-dimensional representations (Teuvo Kohonen, pers. commun.). Therefore, distortion of the auditory (e.g. tonotopic) maps or poor signal-to-noise ratio are not the only reasons for decreased accuracy of the phonological representations. Rather, any deficit prolonging the chunks could impair speech perception and reading skills via distorted phonological representations, despite relatively normal maps in the auditory cortices. According to the motor theory of speech perception[57], the phonological representations could also be directly affected by faulty articulation mechanisms[58].

New interpretations and predictions within the SAS framework

Parietal lobe dysfunction and RSS processing deficits

Within the SAS framework we assume that the RSS processing deficits are secondary to weakened parietal-lobe-supported attentional capture, which affects all sensory modalities. Therefore any parietal lobe lesion could impair RSS processing. In fact, Carlyon *et al.*[59] recently observed that neglect patients suffering from right parietal lobe damage were impaired in stream segregation of sounds presented to their left ear. Also in line with the SAS theory, Cusack *et al.*[60] demonstrated that subjects with right-parietal-lobe lesions show a neglect between, but not within, auditory objects.

As the next step, binaural pitch streaming, similar to that used in our study, could be tested in neglect patients. Another relevant approach would be to test whether a transient neglect, produced by a transcranial magnetic stimulation applied to the parietal lobe[61], would impair RSS processing.

SAS and formant transitions

LLI children, many of whom (but not all) become dyslexics later[62], have difficulties in identifying rapid

formant transitions[63]. Extensive remediation procedures have been developed to improve such discrimination and thereby to help language and reading skills in general. Tallal, Merzenich and co-workers used acoustically processed speech in which the formant transitions were doubled in duration and intensified in loudness, producing metallic-sound speech[24,64,65]; very encouraging improvement was reported following a few weeks' intensive training. Such results appear to strongly support the tight link between the linguistic performance and the accurate perception of auditory frequency transitions[66].

Within the SAS framework, the remediation with acoustically processed speech could also work via training the automatic attentional shifts to become more brisk. Voluntary attention can compensate deficits of the automatic attention, for example by top-down influences[67].

To better understand the underlying mechanisms, future remediation procedures could try to distinguish training of attentional briskness versus detection of frequency transitions, for example by comparing training with speech that contains either amplitude enhancements or transition prolongations[68]. The SAS theory would predict better results with the previous remediation, although the combined training might still give the best results[24,64,65]. A proper control group with only attentional training would be desirable.

SAS and cortical reactivity

Recently, Nagarajan *et al.*[69] demonstrated that the 100-ms response (N100m) of the human auditory cortex to the second stimulus of a sound pair is, at short stimulus onset asynchronies, suppressed in poor readers who were also inferior to the controls in judging the temporal order of two tones. The authors interpreted the result in terms of modified auditory cortical representations. Rather similar suppression of responses to the second tone of a pair have been observed in mice with induced cortical ectopias[70]; this finding is relevant to the present discussion because ectopias have been found in the brains of dyslexic humans[71].

The auditory N100m is elicited by any abrupt sound, and it increases with the behavioral saliency of the stimulus[72]. Therefore the suppressed N100m responses[69], similarly to the suppressed mismatch responses of dyslexic individuals to infrequent sound deviances[73], could be interpreted as supporting weakened capture of auditory attention.

Intermittent background 'masking' sounds, such as speech, music and noise bursts, which effectively dampen the N100m responses to probe sounds[74], should have a more distracting effect on the responses of dyslexic than normal-reading subjects; this could be studied in future experiments.

SAS and experimental differences

Besides the subject selection and possible co-morbidity, the experimental situation significantly affects the obtained results. For example, warning sounds or other alerting methods to keep subjects vigilant during the tasks might cause drastic differences in the results. The spatial imbalance of performance in the temporal order judgment between visual hemifields can be transiently improved in neglect patients by a warning sound presented just before the visual stimuli[38].

For the dyslexic subjects, whom we assume to have weakened attention capture, the stimulus parameters are of the utmost importance. For example, sound intensities and rise times affect the orienting value of the stimuli, as does the stimulus history (e.g. presentation order, interstimulus interval, etc). Reconsideration of the tasks and stimulus parameters within the SAS framework might clarify some of the variability between previous studies of RSS processing.

Conclusion

We have suggested that SAS serves as the pathophysiological link between neuronal-level disorders and behavioral deficits in dyslexic subjects. SAS can impair processing of RSSs in all sensory modalities, affect motor sequencing, and can distort cortical feature representations. Experimental conditions and stimulus features affect the attentional capture value of the stimuli and may have drastic effects on results obtained in RSS studies of dyslexic subjects. The SAS theory allows testable predictions to be made for further experiments.

The multiple disorders observed in dyslexic subjects need not be mutually exclusive or form a single causal chain, and some of them may even be unrelated to the reading problem. Most probably we are not dealing with isolated sensory problems or with an isolated language disorder. Rather, the causes and effects of dyslexia are multifactorial.

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References

1 Merzenich, M. *et al.* (1993) Neural mechanisms underlying temporal integration, segmentation, and input sequence representation: some implications for the origin of learning disabilities. *Ann. New York Acad. Sci.* 682, 1–22
2 Cutting, J. and Pisoni, D. (1978) An information-processing approach to speech perception. In *Speech and Language in the Laboratory, School and Clinic* (Kavanagh, J. and Strange, W., eds), pp. 38–72, MIT Press
3 Hari, R. and Kiesilä, P. (1996) Deficit of temporal auditory processing in dyslexic adults. *Neurosci. Lett.* 205, 138–140
4 Helenius, P. *et al.* (1999) Auditory stream segregation in dyslexic adults. *Brain* 122, 907–913
5 British Dyslexia Association (1995) *The Dyslexia Handbook*
6 Bradley, L. and Bryant, P. (1983) Categorizing sound and learning to read – a causal connection. *Nature* 301, 419–421
7 Stein, J. and Walsh, V. (1997) To see but not to read; the magnocellular theory of dyslexia. *Trends Neurosci.* 20, 147–152
8 Eden, G.F. and Zeffiro, T.A. (1998) Neural systems affected in developmental dyslexia revealed by functional neuroimaging. *Neuron* 21, 279–282

9 Habib, M. (2000) The neurological basis of development dyslexia. An overview and working hypothesis. *Brain* 123, 2373–2399

10 Tallal, P. and Piercy, M. (1973) Developmental aphasia: impaired rate of non-verbal processing as a function of sensory modality. *Neuropsychologia* 11, 389–398

11 Tallal, P. and Piercy, M. (1973) Defects of non-verbal auditory perception in children with developmental aphasia. *Nature* 241, 468–469

12 Reed, M. (1989) Speech perception and the discrimination of brief auditory cues in reading disabled children. *J. Exp. Child Psychol.* 48, 270–292

13 Tallal, P. (1980) Auditory temporal perception, phonetic, and reading disabilities in children. *Brain Lang.* 9, 182–198

14 Laasonen, M. *et al.* (2000) Rate of information segregation in developmentally dyslexic children. *Brain Lang.* 75, 66–81

15 Laasonen, M. *et al.* Temporal order and processing acuity of visual, auditory, and tactile perception in developmentally dyslexic young adults. *Cogn. Affect. Behav. Neurosci.* (in press)

16 Nicolson, R.I. *et al.* (2001) Developmental dyslexia: the cerebellar deficit hypothesis. *Trends Neurosci.* 24, 508–511

17 Demb, J. *et al.* (1998) Functional magnetic resonance imaging of early visual pathways in dyslexia. *J. Neurosci.* 18, 6939–6951

18 Lovegrove, W. *et al.* (1980) Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. *Science* 210, 439–440

19 Galaburda, A. and Livingstone, M. (1993) Evidence for a magnocellular defect in developmental dyslexia. *Ann. New York Acad. Sci.* 682, 70–82

20 Gilger, J. *et al.* (1991) Risk of reading disability as a function of parental history in three family studies. *Reading Writing: Interdisciplinary J.* 3, 205–217

21 Stein, J. and Richardson, A. (1999) Cognitive disorders: a question of misattribution. *Curr. Biol.* 9, R374–R376

22 Tallal, P. *et al.* (1993) Neurobiological basis of speech: a case for the preeminence of temporal processing. *Ann. New York Acad. Sci.* 682, 27–47

23 Studdert-Kennedy, M. and Mody, M. (1995) Auditory temporal perception deficits in the reading-impaired: a critical review of the evidence. *Psychonomic Bull. Rev.* 2, 508–514

24 Tallal, P. *et al.* (1998) Language learning impairments: integrating basic science, technology, and remediation. *Exp. Brain Res.* 123, 210–219

25 von Euler, C. *et al.*, eds (1998) *Basic Mechanisms in Cognition and Language*, Elsevier

26 Stein, J. *et al.* (2000) Controversy about the visual magnocellular deficit in developmental dyslexics. *Trends Cognit. Sci.* 4, 209–211

27 Skottun, B.C. (2000) On the conflicting support for the magnocellular-deficit theory of dyslexia. Response to Stein, Talcott and Walsh (2000) *Trends Cognit. Sci.* 4, 211–212

28 Marshall, C. *et al.* (2001) Rapid auditory processing and phonological ability in normal readers and readers with dyslexia. *J. Speech Lang. Hear. Res.* 44, 925–940

29 Hari, R. (1995) Illusory directional hearing in humans. *Neurosci. Lett.* 189, 29–30

30 Wright, B. *et al.* (1997) Deficits in auditory temporal and spectral resolution in language-impaired children. *Nature* 387, 176–178

31 Llinas, R. (1993) Is dyslexia a dyschronia? *Ann. New York Acad. Sci.* 682, 48–56

32 Witton, C. *et al.* (1997) Temporal pattern analysis in dyslexia. *Br. J. Audiol.* 31, 100–101

33 Hari, R. *et al.* (1999) Non-impaired auditory phase-locking in dyslexic adults. *NeuroReport* 10, 2347–2348

34 Hari, R. *et al.* (1999) Prolonged attentional dwell time in dyslexic adults. *Neurosci. Lett.* 271, 202–204

35 Duncan, J. *et al.* (1997) Restricted attentional capacity within but not between sensory modalities. *Nature* 387, 808–810

36 Husain, M. *et al.* (1997) Abnormal temporal dynamics of visual attention in spatial neglect patients. *Nature* 385, 154–156

37 Hari, R. *et al.* (2001) Left mineneglect in dyslexic adults. *Brain* 124, 1373–1380

38 Robertson, I. *et al.* (1998) Phasic alerting of neglect patients overcomes their spatial deficit in visual awareness. *Nature* 395, 169–172

39 Hikosaka, O. *et al.* (1993) Visual attention revealed by an illusion of motion. *Neurosci. Res.* 18, 11–18

40 Steinman, B.A. *et al.* (1997) Transient visual attention is dominated by the magnocellular stream. *Vis. Res.* 37, 17–23

41 Stein, J. *et al.* (1989) Disordered right hemisphere function in developmental dyslexia. In *Brain and Reading* (von Euler, C. *et al.*, eds), pp. 139–157, Macmillan

42 Riddell, P. *et al.* (1990) Spatial discrimination in children with poor vergence control. *Percept. Mot. Skills* 70, 707–718

43 Facoetti, A. and Turatto, M. (2000) Asymmetrical visual fields distribution of attention in dyslexic children: a neuropsychological study. *Neurosci. Lett.* 290, 216–218

44 Facoetti, A. and Molteni, M. (2001) The gradient of visual attention in developmental dyslexia. *Neuropsychologia* 39, 352–357

45 Facoetti, A. *et al.* (2000) The spatial distribution of visual attention in developmental dyslexia. *Exp. Brain Res.* 132, 531–538

46 Eden, G. *et al.* (1996) Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature* 382, 66–69

47 Marois, R. *et al.* (2000) Neural correlates of attentional blink. *Neuron* 28, 299–308

48 Brunn, J. and Farah, M. (1991) The relation between spatial attention and reading: evidence from the neglect syndrome. *Cognit. Neuropsychol.* 8, 59–75

49 Schwartz, T. *et al.* (1997) Reading errors following right hemisphere injection of sodium amobarbital. *Brain Lang.* 58, 70–91

50 Sheppard, D.M. *et al.* (1999) Effects of stimulant medication on the lateralisation of line bisection judgements of children with attention deficit hyperactivity disorder. *J. Neurol. Neurosurg. Psychiatry* 66, 57–63

51 Vidyasagar, T.R. (1999) A neuronal model of attentional spotlight: parietal guiding the temporal. *Brain Res. Rev.* 30, 66–76

52 Corbetta, M. *et al.* (1998) A common network of functional areas for attention and eye movements. *Neuron* 21, 761–773

53 McPeek, R.M. *et al.* (1999) Saccades require focal attention and are facilitated by a short-term memory system. *Vis. Res.* 39, 1555–1566

54 Marshall, J.C. (2001) Auditory neglect and right parietal cortex. *Brain* 124, 645–646

55 Kohonen, T. (1988) *Self-organization and Associative Memory*, Springer-Verlag

56 Kohonen, T. and Hari, R. (1999) Where the abstract feature maps of the brain might come from. *Trends Neurosci.* 22, 135–139

57 Liberman, A.M. and Whalen, D.H. (2000) On the relation of speech to language. *Trends Cognit. Sci.* 4, 187–196

58 Heilman, K.M. *et al.* (1996) Developmental dyslexia: a motor-articulatory feedback hypothesis. *Ann. Neurol.* 39, 407–412

59 Carlyon, R.P. *et al.* (2001) Effects of attention and unilateral neglect on auditory stream segregation. *J. Exp. Psychol. Hum. Percept. Perform.* 27, 115–127

60 Cusack, R. *et al.* (2000) Neglect between but not within auditory objects. *J. Cogn. Neurosci.* 12, 1056–1065

61 Pascual-Leone, A. *et al.* (1994) Induction of visual extinction by rapid-rate transcranial magnetic stimulation of parietal lobe. *Neurology* 44, 494–498

62 Snowling, M. *et al.* (2000) Is preschool language impairment a risk factor for dyslexia in adolescence? *J. Child Psychol. Psychiatry* 41, 587–600

63 Tallal, P. and Piercy, M. (1974) Developmental aphasia: rate of auditory processing and selective impairment of consonant perception. *Neuropsychologia* 12, 83–93

64 Merzenich, M. *et al.* (1996) Temporal processing deficits of language-learning impaired children ameliorated by training. *Science* 271, 77–81

65 Tallal, P. *et al.* (1996) Language comprehension in language-learning impaired children improved with acoustically modified speech. *Science* 271, 81–84

66 Wright, B.A. *et al.* (2000) Nonlinguistic perceptual deficits associated with reading and language disorders. *Curr. Opin. Neurobiol.* 10, 482–486

67 Driver, J. and Mattingley, J.B. (1995) Selective attention in humans: normality and pathology. *Curr. Opin. Neurobiol.* 5, 191–197

68 Lacerda, F. and Lindblom, B. (1998) Some remarks on Tallal's transform in the light of emergent phonology. In *Basic Mechanisms in Cognition and Language* (von Euler, C. *et al.*, eds), pp. 197–222, Elsevier

69 Nagarajan, S. *et al.* (1999) Cortical auditory signal processing in poor readers. *Proc. Natl. Acad. Sci. U. S. A.* 96, 6483–6488

70 Frenkel, M. *et al.* (2000) Neocortical ectopias are associated with attenuated neurophysiological responses to rapidly changing auditory stimuli. *NeuroReport* 11, 575–579

71 Galaburda, A. *et al.* (1985) Developmental dyslexia: four consecutive patients with cortical anomalies. *Ann. Neurol.* 18, 222–233

72 Hari, R. (1990) The neuromagnetic method in the study of the human auditory cortex. In *Auditory Evoked Magnetic Fields and Potentials. Advances in Audiology* (Vol. 6) (Grandori, F. *et al.*, eds), pp. 222–282, Karger

73 Baldeweg, T. *et al.* (1999) Impaired auditory frequency discrimination in dyslexia detected with mismatch evoked potentials. *Ann. Neurol.* 45, 495–503

74 Hari, R. and Mäkelä, J.P. (1988) Modification of neuromagnetic responses of the human auditory cortex by masking sounds. *Exp. Brain Res.* 71, 87–92