nature neuroscience

© 2005 Nature Publishing Group http://www.nature.com/natureneuroscience

Deficits in perceptual noise exclusion in developmental dyslexia

Anne J Sperling[1], Zhong-Lin Lu[2], Franklin R Manis[2] & Mark S Seidenberg[3]

We evaluated signal-noise discrimination in children with and without dyslexia, using magnocellular and parvocellular visual stimuli presented either with or without high noise. Dyslexic children had elevated contrast thresholds when stimuli of either type were presented in high noise, but performed as well as non-dyslexic children when either type was displayed without noise. Our findings suggest that deficits in noise exclusion, not magnocellular processing, contribute to the etiology of dyslexia.

Behavioral, neuroimaging, computational modeling and intervention studies indicate that mental representations of phonemes are usually impaired in developmental dyslexia[1–3], although the bases of the impairment are less clear. Reading researchers have attempted to identify perceptual, learning or memory impairments that could affect phonological processing[4–8]. One prominent hypothesis involves the magnocellular (M) visual pathway[4,5,7]. In some studies, individuals with dyslexia performed more poorly than normal readers on tasks that primarily involve the M channel. However, the evidence for a deficit in the M channel and its linkage to deficits in reading is weak at best, as is true of the temporal processing deficit to which it is thought to be related[8]. Moreover, many studies that have found magnocellular deficits in dyslexia have used noisy displays[4,5,7]. We investigated an alternative hypothesis based on theories of signal-noise discrimination[9]: that dyslexia is associated with deficits in noise exclusion. This deficit could explain the impaired M channel processing in studies using noisy displays; however, it also predicts that a similar effect would be found in the parvocellular (P) channel in noisy conditions, and that no deficits would be found in either channel in the absence of noise. We tested this hypothesis by presenting dyslexic and non-dyslexic children with stimuli that were designed to preferentially activate either M or P processes, shown either with or without noise.

The M stimulus consisted of wide bars that rapidly alternated between light and dark. The P stimulus consisted of thinner light and dark bars that did not alternate. In previous research, such stimuli have been regarded as differentially engaging the M and P pathways[4,5,7]. In the no-noise conditions, the pattern appeared to one side of a central fixation mark, while the other side was blank. In the high-noise conditions, noise patches (random bright and dark spots, similar to television static) appeared on both sides of the fixation mark, with the signal pattern embedded in one patch (**Fig. 1**; **Supplementary Methods** and **Supplementary Videos 1–4** online). The child's task was to indicate the side that had the pattern. Threshold contrast was determined for each child.

We identified 28 dyslexic and 27 non-dyslexic children (**Supplementary Methods** and **Supplementary Table 1**) and obtained informed written parental consent and child assent. Ten dyslexic children also showed broader language impairments. In the no-noise conditions (**Fig. 2a**), contrast thresholds for dyslexic and non-dyslexic children did not reliably differ for either M (12.3% versus 11.3%, respectively) or P stimuli (16.4% versus 16.4%). In the high-noise conditions, however, dyslexic children's contrast thresholds were significantly ($P < 0.05$) higher than non-dyslexic children's, in both M (28.7% versus 23.9%) and P (38.3% versus 29.7%) tasks. The high-noise conditions yielded graded effects for both M and P tasks (**Fig. 2b**), with the language-impaired dyslexic children having the highest thresholds, the non-language-impaired dyslexic children intermediate thresholds, and the non-dyslexic children the lowest thresholds (**Supplementary Methods**).

Contrast thresholds in both high-noise conditions were correlated with all language measures. In addition, there were moderate correlations with performance on vocabulary, non-word reading and reading comprehension (**Supplementary Table 2**), and high-noise M threshold accounted for 7.8% of the variance in Exception Word Reading independent of Verbal IQ. When Verbal IQ was co-varied out of the regressions, all correlations (with the exception of high-noise M threshold and Exception Word Reading) became non-significant, suggesting a general link to language, literacy and vocabulary skills, rather than a narrow connection to specific reading measures. Verbal IQ itself is probably strongly affected by noise exclusion deficits, accounting for its relationship to the development of phonological and orthographic skills.

These data parallel findings concerning speech perception, which indicate weaker phoneme boundaries and/or non-optimal perceptual templates in dyslexic children with concomitant specific language impairment[10]. Both vision and speech data suggest that dyslexia

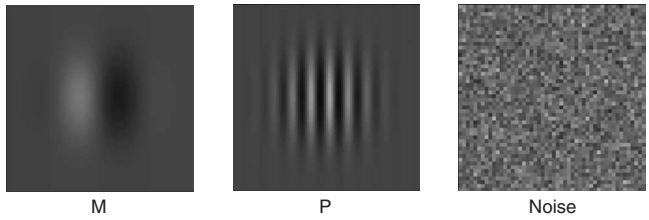

Figure 1 Examples of M and P stimuli and noise patch.

[1]Georgetown University Medical Center, 4000 Reservoir Road NW, Building D, Room 207E, Washington, DC 20057, USA. [2]University of Southern California, Seeley G. Mudd Building, Los Angeles, California 90089, USA. [3]University of Wisconsin, Madison, 1202 West Johnson St., Madison, Wisconsin 53706, USA. Correspondence should be addressed to A.J.S. (ajs76@georgetown.edu).

Published online 29 May 2005; doi:10.1038/nn1474

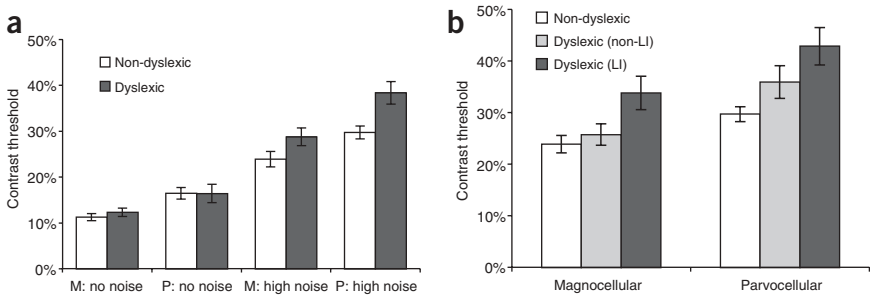

Figure 2 Contrast thresholds. (**a**) Contrast thresholds in the M and P conditions for the non-dyslexic and dyslexic groups. (**b**) Contrast thresholds in the high-noise condition, for non-dyslexic, dyslexic without language impairment and dyslexic with language impairment (LI) groups. Error bars represent s.e.m.

with specific language impairment involves a more severe impairment in noise exclusion than dyslexia alone.

The results are also consistent with findings that poor readers exhibit motion perception deficits only under visually noisy conditions (A.J.S., Z.L.L. & F.R.M., *Cog. Neurosci. Soc.* **10**, D154, 2004), again indicating a potential deficit in noise exclusion, rather than in motion perception (or M channel processing) *per se*, as previously suggested[4,5].

How might a deficit in noise exclusion affect reading acquisition? One possibility is that the visual impairment does not directly affect reading, but rather is part of a broader problem with noise exclusion that affects other modalities as well (such as speech). The visual deficit may be a marker of a more basic underlying perceptual problem that interferes with the formation of perceptual categories in a variety of domains[11], phonological categories being the most relevant to reading acquisition. This hypothesis is consistent with evidence that speech perception is abnormal in infants at risk for dyslexia[12].

A second possibility is that the deficit directly affects reading through the visual modality. Letter recognition requires abstracting away from variations in size, font and style; this may be more difficult if visual processing is hampered by deficits in noise exclusion. Failures to form representations that allow efficient recognition of letters and letter sequences would also interfere with identification of word shape and boundaries between words as well as the representation of sequential orthographic structure. Finally, deficits at this level could have detrimental effects on the development of phonological representations, because experience with an alphabet shapes development of phonemic segments[13]. These effects may not be mutually exclusive; the noise exclusion deficit may be debilitating because it affects reading through several causal pathways.

Our results implicate a perceptual processing deficit in reading and language disorders, one related to noise exclusion rather than anomalies in the M channel or temporal processing. The noise exclusion hypothesis provides a causal theory of the distal neurological basis of dyslexia, via its effects on language development and the acquisition of orthographic and phonological skills.

Note: Supplementary information is available on the Nature Neuroscience website.

ACKNOWLEDGMENTS

This research was supported by National Institute of Child Health and Human Development grant HD29891 (F.R.M.), and by National Institute of Mental Health grants KO2-MH01188 and P50-MH64445 (M.S.S.).

COMPETING INTERESTS STATEMENT

The authors declare that they have no competing financial interests.

Received 24 January; accepted 4 May 2005
Published online at http://www.nature.com/natureneuroscience/

1. Olson, R. *et al. J. Learn. Disabil.* **22**, 339–348 (1989).
2. Harm, M.W. & Seidenberg, M.S. *Psychol. Rev.* **106**, 491–528 (1999).
3. Gottardo, A., Stanovich, K.E. & Siegel, L.S. *J. Exp. Child Psychol.* **63**, 563–582 (1996).
4. Cornelissen, P.L. *et al. Vision Res.* **38**, 471–482 (1998).
5. Demb, J.B., Boynton, G.M. & Heeger, D.J. *J. Neurosci.* **18**, 6939–6951 (1998).
6. Willcutt, E.G. & Pennington, B.F. *J. Child Psychol. Psychiatry* **41**, 1039–1048 (2000).
7. Lovegrove, W.J. *et al. Science* **210**, 439–440 (1980).
8. Ramus, F. *et al. Brain* **126**, 841–865 (2003).
9. Lu, Z.L. & Dosher, B.A. *Vision Res.* **38**, 1183–1198 (1998).
10. Manis, F.R. & Keating, P. in *The Connections Between Language and Reading Disabilities* (Erlbaum, Mahwah, New Jersey, 2004).
11. Sperling, A.J., Lu, Z.L. & Manis, F.R. *Ann. Dyslexia* **54**, 281–303 (2004).
12. Molfese, D.L. *Brain Lang.* **72**, 238–245 (2000).
13. Read, C. *et al. Cognition* **24**, 31–44 (1986).

© 2005 Nature Publishing Group http://www.nature.com/natureneuroscience