children at familial risk for dyslexia: birth to early school age. *Annals of Dyslexia*, **54** (2), 184–220.
Nicolson, R.I., Daum, I., Schugens, M.M., Fawcett, A.J., & Schulz, A. (2002). Eyeblink conditioning indicates cerebellar abnormality in dyslexia. *Experimental Brain Research*, **143** (1), 42–50.
Nicolson, R.I., & Fawcett, A.J. (1994). Reaction times and dyslexia. *Quarterly Journal of Experimental Psychology*, **47A**, 29–48.
Nicolson, R.I., & Fawcett, A.J. (2000). Long-term learning in dyslexic children. *European Journal of Cognitive Psychology*, **12**, 357–393.
Nicolson, R.I., Fawcett, A.J., Berry, E.L., Jenkins, I.H., Dean, P., & Brooks, D.J. (1999). Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet*, **353**, 1662–1667.
Nicolson, R.I., Fawcett, A.J., & Dean, P. (2001). Developmental dyslexia: the cerebellar deficit hypothesis. *Trends in Neurosciences*, **24** (9), 508–511.
White, S., Milne, E., Rosen, S., Hansen, P., Swettenham, J., Frith, U., & Ramus, F. (2006). The role of sensorimotor impairments in dyslexia: a multiple case study of dyslexic children. *Developmental Science*, **9** (3), 237–255.
Wolf, M., & Bowers, P.G. (1999). The double-deficit hypothesis for the developmental dyslexias. *Journal of Educational Psychology*, **91**, 415–438.

What happens when 'dyslexic' subjects do not meet the criteria for dyslexia and sensorimotor tasks are too difficult even for the controls?

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This is a commentary on White *et al.* (2006).

This study claims to have evaluated many of the prominent theories of dyslexia. However, the data below (from Tables 1 and 2) show that the majority of children in the 'dyslexic' group scored well within the normal range on standardized reading and phonological awareness tests:

Reading – WRAT3 mean = 85.78 (SD = 11.86)
Non-word reading – PhAB mean = 93.39 (SD = 6.74)
Rhyme mean = 96.26 (SD = 14.09)
Spoonerisms mean = 98.00 (SD = 9.19)
Alliteration fluency mean = 99.61 (SD = 10.66)
Rhyme fluency mean = 101.70 (SD = 11.93)

How do these standardized scores translate into the data in Figure 1a showing all of the 'dyslexics' scoring below the 5th percentile in literacy?

Although the control group was intended to be representative of normal readers, they were, in fact, scoring well above average on the standardized tests:

Reading – WRAT 3 mean = 112.64 (SD = 10.57)
Non-word reading – PhAB mean = 114.95 (SD = 12.68)

Nonetheless, the authors argue that, as the groups were matched on age and non-verbal IQ, the 'dyslexics' should be compared to this small ($N = 22$) group of above-average readers, rather than on population norms. Furthermore, any control subjects with low performance on any task were identified as 'outliers'.

> To detect the outliers on each task, any control outliers more than 1.65 standard deviations (SDs) below the control mean were removed in order to obtain a better estimate of normal performance, regardless of controls who might have performed abnormally on any one task. The control mean and SD were then recalculated and outliers were defined as those lying more than 1.65 new SDs below this new control mean. (pp. 243–244)

This new control mean was set to a z-score of 0.00 (SD = 1.00). It must be emphasized that all analyses reported, for all literacy and sensorimotor tasks, were based on these recalculated new control mean z-scores. The effect of eliminating low scoring control data in this small group of already above-average readers had a highly differential effect, based on how easy or difficult each task was for the controls. For example, eliminating low scoring control data for the WRAT3 reading

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measure on which controls scored above average further elevated the recalculated mean z-score for literacy. Based on this recalculated control mean score, the dyslexic children (who actually scored less than −1 SD below the standardized mean) now scored a whopping −3.19 (0.85) below the new control mean z-score. Thus, it is only by using this unusual method of recalculation of the data that the authors demonstrate in Figure 1a how children (whose standardized reading scores place them within the low average range) can be converted into 'severely dyslexic' subjects all scoring below the 5th percentile in literacy.

Next, let us look at what happens when the controls score very poorly on a task or group of tasks. This occurred most often on the very difficult psychoacoustic tests as well as several of the other sensorimotor tasks. Specifically, data show that almost one-third of the control sample (7/22) performed at chance on the auditory tasks designed for this study.

The authors emphatically state that,

> The sensorimotor tests were chosen to reflect those currently in use by the proponents of each theory and on which they have found significant group differences. This allowed direct comparison to be made with previous studies and, therefore, any differences between this and previous results could not be attributed to the use of different experimental measures. (p. 240)

This is not, in fact, true, especially for the auditory tasks.

Few of the tasks used in this study are the same as those used in previous studies that have demonstrated deficits in auditory processing (specifically temporospectral auditory processing) in children in this age range (8–12 years). Both critical stimulus features (i.e. the number of formant transitions used to synthesize stop consonant syllables, the type of non-verbal stimuli used) as well as task design features (i.e. the introduction of significant attention, cognitive and memory load into what are intended to be 'pure' perceptual tasks) differ significantly from the studies these authors claim to be attempting to replicate.

The dismal performance of the control children provides compelling evidence that the auditory tasks developed initially for adults were inappropriate for 8–12 year olds. Despite being of normal intelligence and performing above average on literacy and phonological tests, an unacceptably high percentage of controls (7 out of 22) performed at chance on the auditory tasks. To exacerbate this matter, we are told that,

> If the function obtained for a test result was not significantly different from chance performance ($p < .1$), it was replaced with the worst result above chance taken from all the children, on the assumption that the threshold was meaningless. (p. 242)

To clarify, control children who performed at chance were assigned the worst score above chance of any child in the 'dyslexic' or control group. As a third of the control subjects scored at chance on the auditory tests, control subjects' data represent a 'floor effect' below which the 'dyslexics' could not fall. Thus, no significant differences were found. Similarly, having a third of each group's chance scores replaced with the same low score makes it highly unlikely that significant correlations between the auditory measures and other variables with more normal distributions will be observed.

This is a very unfortunate paper for many reasons. Perhaps most critically, the subjects selected do not represent the populations intended. This study is a comparison between average and above average readers, not between dyslexic and normal readers.

The spurious method used in this study for recalculating 'new control means' distorted every single analysis and conclusion reached in this study. The major effect of this manipulation of the data was to change the 'bar' for determining impaired performance based on how easy or hard a task was for the controls. As the controls performed so poorly on so many of the sensorimotor tasks, the results reported are merely a comparison between two groups of children on tasks that are not age appropriate.

Even for studies in which subjects do represent the intended populations, and tasks are age appropriate, behavioral psychophysical measures may not be sensitive enough in older children to accurately detect the presence of current sensorimotor deficits, or the potential influence such deficits may have had earlier in development. Two recent research approaches speak directly to this point:

1. In a prospective, longitudinal study comparing infants with or without a family history of language learning impairments (including dyslexia), Benasich and Tallal (2002) demonstrated a highly significant group difference in rapid auditory processing (RAP) thresholds. These RAP thresholds obtained at 6 months, together with male gender, accurately classified 91.4% of 3-year-old children who scored in the 'impaired' range in verbal intelligence. Longitudinal studies also have demonstrated the relationship between early language learning impairments (LLI) and dyslexia (Bishop & Adams, 1990; Scarborough, 1990). These longitudinal studies provide the best perspective from which to discuss the true 'role of sensorimotor impairments in dyslexia'.

2. Bishop and McArthur (2004) reviewed several previous studies that (like the current study) failed to find deficits on psychoacoustic tasks. Consistent with these negative studies no significant difference was found between LLI and control children, based on behavioral responses to rapidly presented tone pairs. However, a highly significant group difference was found for the same tone pairs based on electrophysiological (ERP) data, with virtually all of the subjects with LLI showing aberrant ERP responses to these rapid acoustic stimuli.

There are hundreds of studies demonstrating sensorimotor deficits in children with significant language and reading impairments (for review see: Habib, 2000; Tallal, 2004). I, therefore, fully agree with these authors' final conclusion that,

> . . . there is an undeniable association between phonological dyslexia and a sensorimotor syndrome including auditory, visual and motor disorders, which certainly points at some common underlying biological factor. (p. 253)

But, I do not agree that, this 'does not directly explain the reading disability' (p. 253).

In interpreting seemingly negative data, such as those reported in this study, it is essential to remember that patterns of deficit that may be seen in infants or very young children may fail to replicate in school age children, college students or adults. Thus, issues pertaining to the cause of developmental disabilities must be addressed from a developmental perspective. Even well after early patterns of deficit/difference/maturation of sensorimotor processing may have resolved, or become recalcitrant to behavioral assessment, they are likely to leave a lasting legacy on the way the brain has organized itself for phonological processing, language and reading throughout life.

References

Benasich, A.A., & Tallal, P. (2002). Infant discrimination of rapid auditory cues predicts later language impairment. *Behavioral Brain Research*, **136**, 31–49.

Bishop, D.V.M., & Adams, C. (1990). A prospective study of the relationship between specific language impairment, phonological disorders and reading retardation. *Journal of Child Psychology and Psychiatry*, **31**, 1027–1050.

Bishop, D.V.M., & McArthur, M. (2004). Immature cortical responses to auditory stimuli in specific language impairment: evidence from ERPs to rapid tone sequences. *Developmental Science*, **7** (3), F11–F18.

Habib, M. (2000). The neurological basis of developmental dyslexia: an overview and working hypothesis. *Brain*, **123** (12), 2373–2399.

Scarborough, H.S. (1990). Very early language deficits in dyslexic children. *Child Development*, **61**, 1728–1743.

Tallal, P. (2004). Improving language and literacy is a matter of time. *Nature Reviews Neuroscience*, **5**, 721–728.

White, S., Milne, E., Rosen, S., Hansen, P., Swettenham, J., Frith, U., & Ramus, F. (2006). The role of sensorimotor impairments in dyslexia: a multiple case study of dyslexic children. *Developmental Science*, **9** (3), 237–255.