RESPONSE

Weighing the evidence between competing theories of dyslexia

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This is a response to commentaries on White *et al.* (2006) by Bishop (2006), Goswami (2006), Nicolson and Fawcett (2006) and Tallal (2006).

We are pleased that our paper 'The role of sensorimotor impairments in dyslexia: a multiple case study of dyslexic children' has attracted comments by some of the prominent researchers in the field. Overall, our lack of support for a causal role of sensorimotor processes in the reading difficulties of dyslexics meets with strong resistance. However, we believe that this resistance owes more to theoretical preconceptions than to real methodological problems. We address in turn methodological, interpretation and theoretical issues raised by Bishop, Goswami, Nicolson and Fawcett and by Tallal, as this allows us to examine again the evidence and counterevidence for the claims we have made.

Methodological issues

Were our dyslexics dyslexic?

On the basis of participants' standard scores on reading and phonological tests, Tallal claims that our dyslexics were not dyslexic and that our controls were 'super-controls'. However, as already explained in the paper, there are very good reasons to believe that population norms for the WRAT3 and for the PhAB are largely outdated following the introduction of the 'literacy hour' strategy in UK classrooms in 1998. Additionally, WRAT3 norms are for US children and it has been shown that these tend to overestimate British children's reading age (Turner, 2000). Therefore it is more appropriate to compare dyslexics directly to the carefully matched control population than to read standard scores literally. Following this logic, Figure 2 clearly shows that the dyslexic participants in this study meet the regression definition of dyslexia. This also answers Nicolson and Fawcett's worries about inclusion: all these dyslexic children meet a discrepancy

criterion, therefore they are not a mix of dyslexic and non-dyslexic poor readers. Furthermore, this was only the second step of selection. The first step involved, for most dyslexic children, an independent formal assessment by the Dyslexia Institute documenting a history of reading disability.

Making an attack on the quality of our sample from a different direction, Tallal suggests that we manipulated the results by using z-scores with respect to control group performance, after removing control outliers. In fact, as she correctly quotes, we used this procedure only 'to detect the outliers on each task'. All the z-scores reported in tables and figures and used in the statistical tests were unaltered, without any data point excluded. Hence the dyslexic mean literacy score of -3.19 SD *is the raw data* (after factoring out non-verbal IQ), simply converted into a z-score (yes, they really were dyslexic).

Our removal of control outliers only affected the *deviance threshold* for each variable, i.e. the dotted lines in Figure 1, and the number of outliers in each domain. This measure was necessary to prevent the occasionally inattentive control from spuriously dilating the normal range of performance, thereby reducing the possibility of detecting dyslexic outliers. This was a conservative step with respect to our conclusions. Not taking it would have led to even fewer dyslexic outliers on sensorimotor variables.

In sum, it seems to be clutching at straws to believe that our dyslexic sample does not represent the intended population.

Was our sample big enough?

All experimenters would love to have more subjects in their experiments, and we are no exception. However, we also realize that there is no point spending time and effort going far beyond adequate statistical power. As far

as statistical power is concerned, our numbers of 23 and 21 children per group are quite typical of dyslexia studies. Dozens of published studies with equal or fewer numbers have found significant group effects on sensori-motor tasks (for recent ones, see Facoetti, Lorusso, Cattaneo, Galli & Molteni, 2005; Hämäläinen, Leppänen, Torppa, Müller & Lyytinen, 2005; Iversen, Berg, Ellertsen & Tønnessen, 2005; Meyler & Breznitz, 2005; Petkov, O'Connor, Benmoshe, Baynes & Sutter, 2005; Stoodley, Harrison & Stein, in press). There is therefore no reason to believe that our study is particularly underpowered. Nevertheless, the point of this study was not whether there are significant group differences or not, since we believe that overall group differences do not address the question of cause. The main point of this multiple case study was to analyse patterns of deficits within each individual. Individual data are important because they assess the extent to which children who are diagnosed as dyslexic have similar sensorimotor and cognitive profiles, and whether a single deficit could underlie the reading problems. Testing 44 cases in depth across a whole range of tasks should be sufficient to do this. Multiple case studies are rare, because most studies are done piecemeal, in such a way that different subjects are tested on different groups of tests and thus individual relationships of performance across tests cannot be examined.

The commentators also worried that the wide age range (8–12) might mask effects on unstandardized sensorimotor tests. Indeed we found that most of these scores correlated with age, and for this reason age (and non-verbal IQ) were partialled out from all the scores entered into statistical analyses and graphs.

Were our tests sensitive enough?

Tallal and Nicolson and Fawcett complain about floor and ceiling effects that might mask group differences. Indeed such effects were obtained, but only for the heel-to-toe and stork balance tasks, where most control children performed at ceiling, while the dyslexics did not. In any case, these variables did yield significant group differences.

Importantly, no floor effect was obtained on any of the auditory tasks. It is not the case that 'seven controls out of 22 performed at chance on the auditory tasks' (Tallal). In fact six of these seven controls performed at chance on just one task and one control on two tasks out of five auditory tasks (and not on the same ones). On average, controls performed normally on 4.6 tasks out of 5. To put it another way, in each auditory task, all controls but one or two performed normally. Unfortunately, our use of the phrase 'chance performance' seems to have been slightly misleading. As can be understood from the text, we meant that these subjects' performance did not yield

a significant fit. This means that thresholds were not meaningful, *not* that they represented chance performance. Typically, this happens when the child does not do the task or does not pay attention and responds at random. It is only in those few cases of non-significant fit that we assigned the child with the worst threshold obtained with a significant fit. This is in fact a conservative step, since in many cases the child might have achieved a better threshold if he/she had concentrated on the task.

In sum, the auditory tasks were of adequate difficulty, were performed normally by almost all the control children, and the lack of group differences cannot be accounted for by a floor effect.

Did our study have 'developmental' power?

Developmental power is not a scientific concept. Rather, the word 'developmental' is used by Goswami as an implicit quality label that can only be attributed to studies which either are longitudinal or use a reading-age control group. We believe that neither longitudinal studies nor matching by reading age are necessarily the most appropriate techniques when investigating the causes of reading difficulty. The notion that there is a gold standard that can answer every question in the field is flawed. Methods must be evaluated with respect to the question that is being asked. Here the question is: Can sensory or motor impairments explain most cases of dyslexia? There is little reason to believe that asking this question separately in 8-, 10- or 12-year-olds would bring any benefit, given that the existing literature suggests that sensori-motor deficits are no more frequent in 8-year-old than in adult dyslexics. Furthermore, as already explained, a reading-age (therefore younger) control group could only have poorer sensorimotor performance, hence defining a poorer normal range from which dyslexics would be even less likely to be outliers.

Could our results be explained in a different way?

Nicolson and Fawcett reject as '*ad hoc*' our definition of the phonology factor, i.e. aggregating phonological awareness, verbal short-term memory and rapid naming measures (we *did not* include semantic fluency in the phonology factor). Yet the partitioning of dyslexics' phonological deficit into these three cognitive components has been well established for a long time (Wagner & Torgesen, 1987). We agree that each of these tasks taps more than just phonological representations (who knows a pure task tapping a single cognitive component?). Admittedly, rapid naming might reflect general speed

factors rather than specifically the speed of retrieval of lexical phonological representations (Bishop). But in the absence of evidence that dyslexics have a generalized speed deficit, the default assumption is that it is restricted to the phonological domain.

Regarding basic speech categorization deficits, the published literature (and the present paper) show that they are much less widespread in dyslexics than the phonological deficit (as defined above), and for this reason they are not currently seen as part of the core phonological deficit (Rosen, 2003; Vellutino, Fletcher, Snowling, & Scanlon, 2004). There is therefore no reason to lump them into the phonological factor.

The potential circularity between reading and phonology measures (Bishop; Nicolson & Fawcett) is already acknowledged and addressed in the paper (footnotes 4, 5 and 6).

Tallal is concerned that the features of our auditory tasks do not correctly address the theories under discussion. We agree that one task, FM detection at 2 Hz, involves variations much slower than implicated in Tallal's 'rapid temporal processing' theory. That task was meant to address the more general 'dynamic processing' construct espoused by Talcott and colleagues (Talcott & Witton, 2002; Talcott, Witton, McLean, Hansen, Rees, Green & Stein, 2000), as it consistently shows auditory deficits in dyslexics, hence its inclusion here. On the other hand, all the speech and non-speech contrasts that we used (*coat-goat*, /ba/-/da/ categorization and discrimination in speech and non-speech versions) differ precisely in the short and rapid 'temporospectral' transitions (<40 ms) that are invoked in Tallal's theory (Tallal, Miller & Fitch, 1993). These four tasks therefore quite accurately address the theory.

Nicolson and Fawcett presume that 40–50% of dyslexics in our sample show balance problems. In fact the exact figure is 26% (six out of 23). These authors also remark that if a cerebellar deficit underlies the phonological deficit, then the cerebellar factor is not expected to account for reading variance after the phonology factor is entered. This is correct (to the extent that these authors give up on the automaticity hypothesis), so indeed our multiple regression is not entirely fair to the cerebellar (or to the auditory) theories. However, these theories predict before anything else that the cerebellar and auditory factors predict some variance in the *phonology factor*, which is not the case (after removing two outliers) (see also Ramus, Pidgeon & Frith, 2003). Additional evidence that sensorimotor impairments do not particularly predict phonological or reading deficits comes from our recent study of autistic children (matched and compared to the present sample), some of whom show massive sensorimotor impairments, yet perfect reading ability (White, Frith, Milne, Rosen, Swettenham & Ramus, in press).

Bishop proposes an alternative explanation of our results: rather than playing no causal role, sensorimotor impairments might exacerbate the impact of the phonological deficit. We were open to this potentially conciliatory hypothesis. However, our data speak against it: no significant difference was found in literacy or phonological performance between dyslexics with and without sensorimotor impairments.

How different is the phonological theory from other theories?

One general matter of debate is whether the phonological theory of dyslexia should be seen as incompatible with other auditory, visual or cerebellar theories of dyslexia (Goswami; Nicolson & Fawcett). This question is best answered by considering separately proximal and distal levels of causation.

At the proximal level, almost everybody agrees that a phonological deficit is the direct underlying cause of most cases of dyslexia. The only non-phonological alternatives are the automaticity hypothesis (one aspect of the cerebellar theory, Nicolson, Fawcett & Dean, 2001) and visual theories (Hari, Renvall & Tanskanen, 2001; Stein & Fowler, 1993; Valdois, Bosse & Tainturier, 2004). Although we have not evaluated all of these alternative theories, our results are consistent with the hypothesis that a minority of cases of dyslexia might be explained by non-phonological deficits. Indeed, we have found that some cases of dyslexia might be explained by visual stress, to the exclusion of any phonological deficit (although visual stress is not a theory of dyslexia *per se*; Wilkins, Huang & Cao, 2004).

At the distal level, the question is whether the phonological deficit is primary, or whether it is secondary to other cognitive, sensory or motor deficits. We argue that our results and the literature are consistent with the former, i.e. with the theory of a primary, specific phonological deficit. Indeed we find that sensorimotor impairments fail to explain the phonological deficit and dyslexia in general. This particularly strong version of the phonological theory is indeed incompatible with sensorimotor theories.

Note that the claim that the phonological deficit is primary does not imply that it is 'detached from any neural underpinnings' (Goswami). Of course, there must be a brain basis for a primary phonological deficit, and indeed there is plenty of compatible neurobiological data implicating left perisylvian areas involved in phonology (Eckert, 2004; Galaburda, Sherman, Rosen, Aboitiz & Geschwind, 1985; Ramus, 2004). But these are neural underpinnings of phonology, not of perception. Goswami's claim that no cognitive deficit is detached from sensory

or perceptual problems is an article of faith, not a scientific result.

Finally, Nicolson and Fawcett have introduced a new and interesting twist to the cerebellar theory, which has in fact transformed it into a hypothesis about the brain basis of the phonological deficit. Until recently, they postulated causal links between the cerebellum and dyslexia via poor automaticity, and via poor motor skill, poor articulation, thus poor phonological skills (Nicolson *et al.*, 2001). Now they propose that language-related regions of the cerebellum might be directly implicated in the phonological deficit. To the extent that it can be shown that phonology is supported in part by cerebellar areas, and that these areas are indeed dysfunctional, we find this hypothesis compatible with that of a primary, specific phonological deficit.

Conclusions

We are surprised that the target article has met with some fierce opposition, and sorry that this has led to insinuations that our methodology is flawed. Indeed, our main finding that sensory and motor deficits are not universal but restricted to a subgroup of dyslexics is consistent with every single published study showing reliable individual data (Ramus, 2003), including studies by our critics (e.g. Fawcett & Nicolson, 1999; Muneaux, Ziegler, Truc, Thomson & Goswami, 2004; Tallal, 1980). Furthermore, the results of the present multiple case study closely parallel those obtained in an earlier one on dyslexic adults (Ramus, Rosen, Dakin, Day, Castellote, White & Frith, 2003), and in similar studies on dyslexic children conducted by independent groups, one with (presumably) different working hypotheses (Kronbichler, Hutzler & Wimmer, 2002; Stoodley & Stein, 2004).

Obviously, the present study does not claim to have closed the debate on theories of developmental dyslexia. It simply shows that when one considers and tests a very wide range of possible sensory and motor explanations of dyslexia, even taken all together, these sensorimotor deficits fail to explain much more than half of dyslexic cases. Then, one must conclude either that some dyslexics have a specific phonological deficit (and one must explain the association between phonological deficit and sensorimotor syndrome, e.g. Ramus, 2004), or that the current methods are inadequate to reveal the real extent of sensorimotor deficits. In the latter case, the burden is on proponents of sensorimotor theories to improve their methods.

Finally, we strongly believe that it is no use to keep inundating the literature with study after study showing group differences on sensory or motor tasks: this will

not do. As explained by Tallal and Goswami, the best hope for sensorimotor theories to prove their case is in (1) longitudinal studies starting at birth, measuring early patterns of sensory deficits before they putatively 'resolve';[1] (2) potentially more sensitive measures that might reveal sensory deficits in all dyslexics even at school age.[2] In both cases, such studies will be convincing only to the extent that they provide reliable individual data and demonstrate a much greater prevalence and predictive power of sensorimotor deficits in the dyslexic population than has been observed so far.

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[1] Tallal's claim that sensorimotor impairments may resolve despite continued reading problems is at odds with her notion of an auditory treatment of dyslexia (Tallal, 2004), as such cases would show that resolution of the auditory impairment does not entail the resolution of the phonological deficit and reading disability.

[2] Goswami, Tallal and many others place great hopes in ERPs. However, using current paradigms, ERPs typically do not provide reliable individual data. If the idea is to use ERPs to demonstrate more group differences, this will be no improvement on the current situation.

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