

References

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Why dyslexics are different

Ellis, McDougall and Monk claim, on the basis of negative results on selected tests, that dyslexics and younger normal readers cannot be distinguished. We point out that even between their groups, there are significant differences, consistent with the previous literature. These differences are in line with the theory that there exists a biological abnormality underlying a cognitive deficit in the phonological system which manifests itself in a variety of ways depending on age, intelligence and education of the individual. Mere behaviourism will no longer serve as a basis for diagnosis or treatment.

Dyslexia is a developmental disorder with a biological basis, currently diagnosed in terms of behaviour criteria. As a result, classification of individuals as dyslexics or as ordinary poor readers tends to be arbitrary. It can also be anomalous. For example, a young dyslexic child cannot have a discrepancy between reading age and chronological age; an older individual may no longer show a discrepancy, even though this person still finds reading slow and effortful or breaks down under stress. In addition, schooling, general level of ability, and motivation will all influence performance on psychological tests, and thus may confound and confuse the detection of impairments.

A classification based on a coherent theory where behavioural signs are seen as manifestations of an underlying cognitive cause can overcome these problems (Morton and Frith, 1995). There is increasing evidence that at least one form of dyslexia is a phonological disorder discernible as persistent throughout life despite changes in reading performance. For instance, Nicolson and Fawcett (1995) report persistent phonological processing problems in dyslexics at age 13, 15 and 17; Pennington *et al.* (1990), and Bruck (1992), amongst others have shown that adult dyslexics still show problems on phonological tasks. This form of dyslexia has a genetic basis (Cardon *et al.*, 1994), with first signs already appearing at preschool age in spoken language (Scarborough, 1990). There is anatomical evidence of brain abnormalities (Galaburda, 1989) and there is evidence from functional neuroimaging studies that adult dyslexics show abnormal brain activation in precisely those regions known to subserve phonological processing (Rumsey *et al.*, 1992).

Against this background of research, the paper by Ellis, McDougall and Monk (EMM) is somewhat anachronistic. The question that EMM ask is whether children of different ages and abilities, but obtaining the same score on a word recognition test, process letters, words and texts in the same way. All they succeed in doing is showing that their four groups perform similarly on particular tests. They ignore the differences.

Most evidently, the groups can certainly be distinguished from each other on the basis of the selection criteria which include IQ and age. One feature of the group of dyslexics (IQ at least 105) that passes without comment, is that the verbal component is 18 points worse than the performance component. This difference, characteristic of dyslexics (Nelson and Warrington, 1980) is highly significant. Their group of so-called poor readers is a rag bag of children with performance IQs ranging from 46 to 104 and a discrepancy of RA to CA ranging up to 3 or 4 years. At least some of these children would have the same specific cognitive deficit—over and above any other deficits—as those here classified as dyslexic. Thus we would expect these two groups to show more similarities than differences on phonological tasks. This was indeed the case.

What EMM were looking for was performance of older bright dyslexics that was significantly worse than that of younger less bright readers (the RA controls). This is a tall order. Nevertheless, they got one such result which is consistent with the majority of previous studies (as reviewed by Rack, Snowling and Olson, 1992): Dyslexics achieved a lower nonword reading score (on our calculation $t=1.86$, $df\ 24$, significant at $p<0.05$ one-tailed). Now, if the difference between reading words and nonwords indicates something about the quality of reading, then the dyslexic group is reading in a different way—just as predicted from the phonological deficit hypothesis.

Other tests of phonological processes that were used in this study did not show this predicted difference of dyslexics compared to RA controls. Unfortunately this result is not informative because it cannot be ruled out that the advantage of age and ability that the dyslexics enjoyed over the RA controls cancelled out any disadvantage they had due to compromised phonological processing. EMM are inclined to dismiss earlier results that discriminate dyslexics from younger RA matched readers on phonological processing tasks in favour of their own results which failed to discriminate the groups. This confidence may not be warranted since null results generally are easier to obtain than not, be it due to procedural inadequacies or range effects on the measures used. One has to bear in mind that even sensitive tests are sensitive only at particular ages and levels of ability, usually for a rather narrow window of performance levels. In EMM's study a sizeable number of measures proved insensitive to differences in age and ability, quite apart from reading difficulty.

The fact that the bright dyslexic children in EMM's study did not have better phonological skills on the tasks used than a group of average children over 2 years their junior seems to us not without interest. Since EMM believe that phonological skills advance with reading ability, they would have to explain all this shortfall in terms of reversed cause: the dyslexic group are reading retarded and therefore their phonological skills are retarded. The scientifically interesting question is: what caused their difficulties in learning to read, and why do few people have such difficulties while many do not? EMM do not tackle this question, but instead appeal to some notion of delay. We feel that it is important to note that the dyslexic children were not generally developmentally delayed: they had superior performance on visual tasks and comprehension as well as the Wechsler performance tests. Thus there is a specific delay. This must have a specific cause.

In one experiment we have studied well compensated adult dyslexics and found differences from their peers on phonological processing tasks that were able to challenge the phonological system (Paulesu *et al.*, 1996). One such task is

Spoonerisms, where these compensated dyslexics took 3 to 4 times as long per item than their peers. On simple, non-challenging, rhyme judgment and short term memory tasks, they showed no behavioural difference from their peers. However, when they performed these tasks while being PET-scanned, the associated brain activity was highly abnormal. Negative behavioural evidence is not sufficient to disprove a cognitive hypothesis that is amenable to testing by brain imaging techniques.

It is no longer appropriate to ignore that dyslexia has a biological component (genetic factors, anatomical evidence of brain abnormalities, functional imaging evidence). Dyslexics are different, and the difference does not rely on a particular size of discrepancy of RA from CA, for a given IQ. What we need to work towards is a definition that overcomes the vicissitudes of behavioural criteria. We believe that a good way forward is to connect biological and behaviour facts via the cognitive level of explanation. We suggest it is the cognitive level that provides a psychologically relevant definition of dyslexia. The hypothesis of a phonological processing deficit has the power to explain the distinctive pattern of behaviour throughout development.

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