WHEN the dyslexia storm broke in September, I was not surprised that one tactic employed by detractors was to put words in my mouth and then attack them. I didn’t expect to find such an approach employed in The Psychologist.

The programme (entitled ‘The Dyslexia Myth’, not ‘The Myth of Dyslexia’) did not argue that dyslexia was a myth; it stated that ‘the common understanding of dyslexia is a myth which hides the scale and the scandal of true reading disability’. Indeed, as I (and others associated with the programme) have repeatedly said, the question as to whether dyslexia exists or not is essentially meaningless.

The fallacy of such a question becomes clear when one examines working definitions such as that of a BPS working party report which stated: ‘Dyslexia is evident when accurate and fluent word reading and/or spelling develops very incompletely or with very great difficulty.

This focuses on literacy learning at the “word” level and implies that the problem is severe and persistent despite appropriate learning opportunities’ (BPS, 1999, p.64).

On this basis, how could one question the existence of dyslexia? The more meaningful question is how can this position be reconciled with the many very different definitions (and symptoms) employed by others, and what relevance do these varied conceptions have for clinical/educational intervention? In this respect, I was pleased that Nicolson accepts the important point that diagnosing dyslexia is not the objective process that many are led to believe, neither does it point to appropriate forms of treatment.

Nicolson mentions the heritability findings as if this were something he was pointing out, but the heritability findings were highlighted in the programme itself. He says ‘the fact that 50 per cent of the variance in dyslexia is genetic means that dyslexia does have a clear and distinct basis, and hence cannot be a “myth”’. In fact, they show that poor reading has a clear and distinct basis, not that dyslexia as traditionally conceived (by reading/IQ discrepancy, visual reversals, etc.) has a clear and distinct basis. He sets up the ‘straw man’ by saying: ‘No one has ever suggested that children with generalised learning difficulties can’t learn to read.’ But we were actually questioning whether children with dyslexia (as traditionally defined) respond differently to intervention from those with generalised learning problems. In rejecting this, we highlighted the absence of clear evidence that there exists a particular teaching approach that is more suitable for a dyslexic subgroup than for other poor readers.

The programme did not sideswipe Nicolson’s cerebellar deficit hypothesis. It neither reported nor commented on any of the theories about the underlying pathology which might explain the phonological deficit (the immediate cause of reading problems) and the comorbidity often associated with this deficit but not thought to be its cause. The documentary did report criticism of the DDAT treatment for reading problems, which claims to be based on the cerebellar deficit hypothesis. It did so because the research Nicolson refers to as supporting this complementary approach (which has been widely publicised in the media) has been subjected

\[\text{LOOKING THROUGH COLOURED LENSES}\]

IN his introduction Professor Elliott was dismissive of certain ideas that highlight particular aspects of the subject. For instance he ridicules the idea of coloured lenses. This refers to a small fraction of individuals who suffer from scotopic sensitivity. They experience movement and distortion of black print on a white background, and successful treatment is offered by the use of tinted lenses or coloured overlays.

Normally this is only one aspect of an individual’s reading difficulties, but for some it can be a very significant feature. Scotopic sensitivity is a form of visual dyslexia, and it does exist. Specialists have highlighted its existence but no one claims that it is a complete solution. Does total agreement as to the nature or treatment of any human condition exist?

The same applies to coordination activities. Again they are not a complete answer to dyslexia, but they can help children who suffer from dyspraxia or coordination problems or dysgraphia (handwriting difficulties). Both these latter conditions are often associated with dyslexia and treating them appropriately can have a positive effect on the development of the child’s literacy skills.

If the general public, as Elliott claims, falls for single therapies as a complete answer to the very complex condition of dyslexia then that is surely their fault. However, specialists in the area have never claimed this. It may be true that certain sections of the population are over-influenced by new ideas. But that is no reason to consign to oblivion the term ‘dyslexia’, which has been a useful administrative and legal term for many years.

Peter Congdon
21 Hampton Lane
Solihull
to criticism by leading researchers on both sides of the Atlantic (e.g. Snowling & Hulme, 2003; Stein, 2003).

While his point about the Code of Practice is true up to a point, I am puzzled that Nicolson doesn’t recognise the more subtle ways that a dyslexic diagnosis can influence both teachers and gatekeepers to resources. Teachers are increasingly wary of litigation and may seek to protect themselves against legal challenge. It would be naive to underestimate the power of the label to access additional resources, a point recently noted by school SEN coordinators (SENCO-Forum, 2005).

Finally, I am rather surprised by the simplistic distinction between educational and ‘academic’ psychologists, finding this neither helpful nor meaningful. Is he, in actuality, differentiating between the diverse academic fields of cognitive and educational psychology? If so, it might be helpful if he didn’t offer imprecise and inaccurate accounts of the latter discipline. Certainly, there are areas in educational psychology where ascertaining the causes of a problem (even if this were possible) is not very helpful for guiding intervention.

Our knowledge of factors that underpin reading disability has massively increased in recent years and it seems likely that brain function and genetic studies offer much for the future. Hopefully, such work will ultimately provide valuable guidance in developing increasingly effective interventions. At the current time, however, splitting poor readers into two groups – dyslexic sheep and ordinary poor-reading goats – has little practical value for dealing with literacy problems. Rather than pouring resources into dyslexic assessments, we would, at the current time, be wiser to target all poor readers at an early age for intervention. This is the main point that the programme set out to make.

Julian Elliott
Durham University


HAVING worked in the field of specific learning disability for over 40 years, I am still greatly upset that my colleagues are trapped in their thinking by medical titles, such as ‘dyslexia’, ‘dyscalculia’, ‘dysgraphia’, etc., which are merely descriptions of symptoms.

We should be looking instead for the causes of these problems. Following research in Canada connecting such problems with metabolic problems, an article in Scientific American (from memory, in the 1980s) found the genetic link on the X chromosome. This explains the four-to-one ratio of such problems in boys vs. girls.

I have found over the years that, when there is evidence of SLD in WISC or WAIS subtest scatter, perceptual problems in visual or auditory modalities, fine motor problems and of course reading and spelling or calculation below the level expected from the intelligence level, comprehensive tests of allergies and of vitamin and mineral trace deficiencies almost invariably find the metabolic connection. The results are so often spectacular, that teachers and parents are amazed that the client is so greatly improved in concentration and ability to learn, often in a few days.

Terms like ADD and ADHD are again merely descriptions of symptoms, which are treated by the medical profession with drugs. This does not get at the cause of the problem, and often leads to dependency.

John H. Jenkins
University College London

THERE are a number of issues here that highlight what appears to be serious conceptual confusion in the field. These carve out an important agenda both for research and practice.
In order to consider what is at stake, it is helpful first to refer to the important theoretical framework proposed by Morton and Frith (1995; see also Morton, 2004). According to this framework, it is important when considering developmental disorders to separate the biological, the cognitive and the behavioural levels of explanation. Importantly, it is necessary to acknowledge that developmental disorders are dynamic and there are environmental interactions at all levels. So the behavioural manifestations of disorders, such as dyslexia, change with time, and also in different contexts—for example we would see different behaviours in a child taught to read in Italian or in one who received early intervention.

The phonological deficit theory of dyslexia, featured in the documentary, is a theory at the cognitive level. It explains a constellation of behaviours that are normally associated with dyslexia (short-term memory problems, word-finding difficulties, etc.). The phonological deficit theory is a well-specified, falsifiable theory that so far has not been refuted. What many respondents are upset about is that certain behaviours often associated with dyslexia are not explained by the theory—e.g. visual problems, problems of organisation and of motor control. Of course, it is correct that these behaviours often co-occur with dyslexia; they signal important co-morbidities. Why they do is poorly understood. Next steps must involve seeking both biological and cognitive explanations of these associated disorders so that ultimately we can begin to unpick what is dyslexia (the construct under threat), what is not dyslexia and why these behaviours co-occur so frequently. But, to gather everything under the umbrella of ‘dyslexia’ helps neither theory nor practice. As for the call for ‘cut-off points’ for ‘dyslexia’, we can as a profession agree criteria for extra time or a laptop computer, but it is meaningless to imagine quantitative criteria defining a dynamic developmental disorder.

Maggie Snowling
University of York

FOR some years I have been asking my university colleagues how they determine that a student has dyslexia, and thereby grant them extra time in examinations. None of the answers I have received are based on specific criteria and cut-off points drawn from an epidemiologically defined population, say a national sample of 18-year-olds. It would be good to update an old reference (Yule et al., 1974). Obviously, if anyone has such data it would help clarify if dyslexia exists.

James Thompson
Centre for Behavioural and Social Sciences in Medicine, University College London