~250 ug/l (*1*). This safe level plus a five-fold safety margin make up the present drinking water standard of 50 ug/l.

Within a factor of 2, the safe level of arsenic remains the same if good and reproducible science of the intervening 100 years is used (*2*). That requires weeding out controversial studies such as the one from northwestern Taiwan (*3*), which is highlighted in the news article.

Results presented in Table 4 of that study show 3, 3, 2, and 7 cases of urinary cancer and 1, 1, 2, and 6 cases of transitional cell carcinoma at arsenic levels below 10, 10 to 50, 50 to 100, and above 100 ug/l, respectively. Numbers of cases at the three levels below 100 ug/l are so small that no positive interpretation of increased cancer risk is possible. The claim that "cancer risk rose with arsenic levels even at these low exposures" is incorrect. There are hundreds of arsenical skin cancers on record and thousands of cases of the typical arsenicism, fully reproduced at levels above 200 ug/l. These cases and the complete absence of arsenical skin disease in the United States should be used to identify the safe level and to set a drinking water standard.

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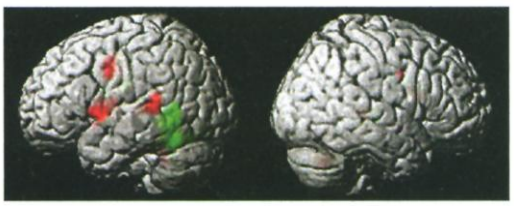
Defining Dyslexia

DYSLEXIA IS CALLED "THE LANGUAGE DISORDER that makes reading and writing a struggle" by Laura Helmuth in her News of the Week article "Dyslexia: same brains, different languages," (16 Mar., p. 2064). Although she is in the good company of many cognitive neuroscientists and educational psychologists, her terminology is in error. Evolution prepared us for language, but not for reading or writing. Indeed, Western cultures have demanded that all their normal children acquire script only within about the past 100 years. It is surprising and satisfying that most children do develop a reasonable reading skill—but many children don't. Most of them would never have become diagnosed as "language disordered" in an oral culture; they have speech and language skills that are entirely in the normal range. Calling dyslexics "language disordered" shows a lack of evolutionary and historical awareness and it risks being considered discriminatory.



Green areas of the brain are significantly less active in dyslexics compared to normal individuals when reading simple words.

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The wonderful report by E. Paulesu *et al.* does not make this error ("Dyslexia: cultural diversity and biological unity," p. 2165).

Still, in the Paulesu *et al.* report, developmental dyslexia is called a "disorder of genetic origin," and the authors discuss "brain abnormalities" that are apparently involved. The implicit assumption is that our brains should normally allow for the acquisition of reading. If they don't, then there must be an abnormality. The question is whether this "abnormality" is still within the normal evolutionary range.

In other words, would our ancestors with such brains have become normally speaking and normally functioning hunter-gatherers? If so, it is a misnomer to denote dyslexics as neurologically abnormal. It

should not be the arbitrary prevalent culture that defines what is neurologically normal or deviant.

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Response

SINCE READING REPRESENTS HIGHLY ARTIFICIAL behavior, and is of historically recent origin, Levelt concludes that a dyslexic would be well off in a nonreading world, and he therefore assumes that the dyslexic's altered pattern of brain response does not represent an underlying neurological abnormality.

However, reading difficulties can no longer be considered a necessary or a sufficient sign of dyslexia (*1*). The primary and enduring cognitive consequences of dyslexia are subtle deficits in speech/language processing. Affected individuals, from early childhood onwards, have problems in tasks that tap phonological skills (e.g., word repetition, verbal short-term memory) and tasks that require the rapid retrieval of words (e.g., object naming, digit naming). Phonological competence is part of linguistic competence and has a basis in the brain (*2*), plausibly with a heritable component (*3*). It is manifest with the ease by which we learn new words in our mother tongue and when we learn a foreign language (*4*). These are skills that even hunter-gatherers may have found useful for communication with their neighbors. The spectacular rise of writing systems in the last 5000 years testifies to the existence of a strong human instinct for communication.

Given that reading problems by themselves do not distinguish dyslexics from those who are merely at the tail end of the normal distribution, we suggest that the combination of cognitive neuropsychology and neuroimaging may provide a better criterion. Our results show that the brain activation pattern in dyslexics, identified as possessing impaired phonological skills, is different while they are reading simple words (*5*).

Why is reading affected at all in these people? In adult normal readers, the neural systems for reading largely overlap with those for object naming (*6*, table 2e); learning to read may imply a systematic moulding of that part of the neural system that allows the brain to name objects. We propose that dyslexic brains are not able to mould connections between the sight, sound, and meaning of a word as efficiently as other brains (*7*). In a preliterate world, this disorder would not lead to the same kind of social exclusion, but it could have subtle consequences for an individual's status in societies that value verbal ability.

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