The interface between genetics and psychology: lessons from developmental dyslexia

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Developmental dyslexia runs in families, and twin studies have confirmed that there is a substantial genetic contribution to poor reading. The way in which discoveries in molecular genetics are reported can be misleading, encouraging us to think that there are specific genes that might be used to screen for disorder. However, dyslexia is not a classic Mendelian disorder that is caused by a mutation in a single gene. Rather, like many other common disorders, it appears to involve combined effects of many genes and environmental factors, each of which has a small influence, possibly supplemented by rare variants that have larger effects but apply to only a minority of cases. Furthermore, to see clearer relationships between genotype and phenotype, we may need to move beyond the clinical category of dyslexia to look at underlying cognitive deficits that may be implicated in other neurodevelopmental disorders.

1. Developmental dyslexia: a familial disorder

Difficulty learning to read is an all-too-common problem in children who are otherwise developing normally. Some highly intelligent people struggle to learn to read, and even if they master basic literacy, they continue as adults to find reading a slow and ponderous process. Such unexplained reading failure is often referred to as ‘developmental dyslexia’. The popular view of dyslexia is that it is a clear-cut disorder, distinct from normal development, and with a specific organic basis. As it has been known for many years that dyslexia runs in families[1], this view has been extended to incorporate the idea that there are specific genetic mutations that cause dyslexia, with the expectation that we might develop a genetic test to screen children for dyslexia and give them special assistance before reading problems become apparent[2]. As we shall see, however, media reports suggesting a genetic test for dyslexia is just around the corner[3] are far from reality. There are two related issues to grapple with: first, the notion of dyslexia as a syndrome with clear-cut boundaries is unrealistic, and second, attempting to account for dyslexia in terms of single mutations is misguided.

Let us consider first how dyslexia is defined. Diagnostic manuals such as the International Classification of Diseases (ICD-10) [4] or Diagnostic and Statistical Manual of the American Psychiatric Association (DSM5) [5] aim to specify objective criteria for differential diagnosis of disorders. DSM categories are, however, a consensus view of a committee and lack independent validation. The traditional textbook definitions of dyslexia emphasize that there is a mismatch between reading achievement and measured intelligence, and they focus on problems with written language. In practice, however, research does not support the IQ-discrepancy criterion [6], and it is widely recognized that the problems experienced by poor readers typically involve processing of spoken as well as written language[7]. Another popular notion is that dyslexia involves distinctive symptoms such as reversal of letters when reading, or an unusual perceptual or cognitive profile. However, here again evidence is lacking and now many experts reject the term ‘dyslexia’ as misleading in implying a syndrome that is distinct from ‘poor reading’ [8]. This does not, however, mean that people deny the existence of children who have significant reading difficulties or that biological factors are unimportant.
Findings from family studies of dyslexia [9] led to initial optimism that genes for dyslexia would soon be discovered. However, once it became feasible to look for genes using molecular methods, it became clear that things were not so simple. There were occasional findings of rare mutations associated with dyslexia where there was a clear-cut pattern of inheritance [10]. However, these seemed specific to particular families, and it is the exception rather than the rule to find mutations associated with dyslexia.

2. Common genetic disorders are seldom Mendelian

When asked about genetics, many people will think back to schooldays where they learned about Mendel’s peas, where skin colour and wrinkliness could be explained in terms of variants in one or two genes. In medicine, introductory courses often illustrate genetics using a specific condition such as Huntington’s disease, where a rare mutation in a single gene causes neurological degeneration. However, the genetic basis for many disorders is quite different to this. Indeed, some have gone so far as to suggest that schoolchildren should stop being taught about Mendel’s peas because it provides too deterministic a view of contemporary genetics [11].

Common medical conditions such as heart disease, allergies, asthma and diabetes all run in families, but family pedigrees do not usually show a Mendelian pattern of inheritance. Furthermore, genetic association studies do not find common mutations of large effect that cause these illnesses. Instead, there appear to be many genetic variants that individually have a small influence on likelihood of developing the condition, but which in combination can create a high risk for disorder. The term ‘complex multifactorial disorder’ was coined to describe the situation when risk for disorder depends on the combined influence of many genetic and environmental influences [12]. This is typically modelled using ideas introduced by Falconer [13] in the 1960s; he proposed that there is an underlying normally distributed continuum of ‘liability’ for disorder, which is influenced by multiple risk factors, and disorder becomes evident when liability exceeds a given threshold (figure 1).

![Diagram of liability threshold model of disorder](http://rspb.royalsocietypublishing.org/)

This method was first developed in the context of reading disability. As noted by Plomin et al. [15], this view of neurodevelopmental aetiology: rates of impairment are elevated in relatives of cases. The key question is whether identical (monozygotic, or MZ) twins are more similar to one another than fraternal ( dizygotic, or DZ) twins, who are created when two ova are fertilized at the same time, and so are genetically as similar as regular siblings.

Over 99% of the DNA sequence of the genome is the same in everyone. When we discuss genetic similarity of related individuals in the context of a family study, we refer to polymorphic DNA, i.e. the one in every 300 or so DNA base pairs that can take different forms in different people. When we say that genetic relatedness in DZ twins is 50%, whereas it is 100% in MZ twins, we are referring to this polymorphic DNA, which is the only genetic material that can explain differences between people.

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Heritability of a trait is defined as the proportion of variance in that trait that can be attributed to genetic variation. For quantitative traits, a rough estimate of heritability can be obtained by measuring the twin–co-twin correlation for MZ and DZ twins, and doubling the difference, i.e. 2 \times (MZr – DZr). A better approach uses structural equation models which take into account sample size and provide confidence intervals around the estimates of heritability. These involve modelling underlying latent factors that determine covariance between twin phenotypes for MZ and DZ pairs [16].

This approach can be extended to the case of a categorical disorder, by converting the concordance for disorder in MZ and DZ twins to tetrachoric correlations [17]. If, however, the disorder is defined in terms of a quantitative dimension, another method, DeFries–Fulker analysis [18], can be used. This method was first developed in the context of reading
disability, which lends itself readily to the approach, because reading can be quantified in terms of a score on a reading test. We can define as ‘proband’s’ (affected cases) individuals whose scores fall below some cut-off on the reading distribution. The interest then is in how far we can predict the co-twins’ scores from the proband information. The logic of the method is easy to demonstrate with a thought experiment (figure 2). If the cause of poor reading were completely random, e.g. being struck on the head by a falling object, then you would not expect two members of a twin pair to resemble one another; instead, the expectation would be that the co-twins’ scores would average out at the population mean. If, on the other hand, the disorder were completely explicable by something in the environment shared by the twins, for instance if both children were taught by the same inadequate teacher, then the proband and co-twin should both score poorly. Finally, if genes were the sole cause of disorder, then the scores of MZ twins should be identical, whereas the mean for DZ twins would regress halfway to the population mean—because on average they share 50% of their polymorphic genes. In practice, we do not ask whether it is genes, shared environment or random factors that cause disorder; rather, we estimate the relative contribution of these three sources of variation. DeFries and Fulker demonstrated that if data were appropriately scaled, these estimates could be directly obtained from multiple regression analysis [18], in which the dependent variable of co-twin score is predicted from the proband score and the coefficient of relationship, which is 1 for MZ twins and 0.5 for DZ twins.

DeFries–Fulker analysis of reading difficulties has been used with three large samples: from the Colorado Learning Disabilities Research Center (CLDRC) [19], from the Twins Early Development Study [20] and from a large sample of twins and adoptees in Minnesota [17]. In all three studies, the heritability of reading disability was estimated to be at least 60%. The first two studies estimated the effect of shared environment at around 30%, with the remaining 10% of variance accounted for by random effects or environmental effects specific to one twin. The study that included adoptees found the effect of shared environment to be negligible. Such results confirm that genes are an important factor in influencing whether or not a child is a poor reader.

Heritability is sometimes assumed to be a fixed characteristic of a particular trait such as reading ability, but this is mistaken. Consider, for instance, reading ability in Brazil, where many children do not receive much schooling. A twin study done using the whole population of São Paulo would show low heritability, because much individual variation in reading would be related to educational experience. By contrast, in Helsinki, where nearly all children receive high-quality reading instruction, heritability will be higher, because relevant environmental influences are less variable. This example illustrates an important point about heritability: it is defined as the proportion of variance in a trait attributable to genetic factors and will vary with the range of genetic and environmental variation in the population. It follows that if we want to find genetic influences on reading, it will make sense to focus on children who have a fairly uniform environmental experience, i.e. where all have access to high-quality educational instruction. Such children fit well with the traditional definition of developmental dyslexia, as ‘poor reading ability despite adequate opportunity to learn’. As noted by Asbury & Plomin [21], the higher the educational standards in a population, the more likely it is that individual differences in ability are attributable to genetic differences between children.

4. Molecular approaches

A complex multifactorial disorder such as dyslexia (or reading disability) poses problems for those attempting to identify the genes that affect the condition, if we assume there are many variants that are common in the population, each of which contributes only a small effect. The genome-wide association (GWA) method was developed to address this problem; large samples (typically several thousand cases) of affected and unaffected individuals are compared to find genetic variants...
that influence specific traits [22,23]. The strategy is to look for associations between a set of known DNA variants (usually in the form of single nucleotide polymorphisms or SNPs) and the trait, either by categorizing people, e.g. as dyslexic or not, and comparing the proportions with different SNPs, or by seeing whether the specific allele at a given locus affects a person’s average score on a trait such as reading ability. These studies have the challenge of having huge numbers of SNPs to sift through, with a high probability that some of these will show an association with the phenotype by chance. A GWA study uses very large samples and considers whether the distribution of probabilities for association with all SNPs under investigation deviates from that expected by chance. It capitalizes on the fact that SNPs close together on a chromosome are inherited together, and so one need not evaluate the status of every SNP in the genome. Instead, a set of marker SNPs (typically between 100,000 and 1,000,000 SNPs) is used to give good coverage of all regions of the genome. When a SNP is found to be associated with a trait, it does not necessarily mean it is functional, but it can help identify a region of DNA likely to harbour a functionally significant gene. Although much research has focused on coding regions of DNA (exons), there is growing interest in a possible role of ‘silent’ regions of the genome (introns) in determining phenotypes [24].

Although such studies have found some replicated associations, the effect sizes are typically very small [25]. Reports of molecular genetic studies usually emphasize the p-value, a measure of how probable it is that an association could have arisen by chance. A low p-value indicates that a result is unusual, but it does not mean the effect is large. Molecular genetic studies typically use very large samples precisely because this allows them to detect even small effects. Consider one of the more reliable associations between genes and behaviour: several SNPs in proximity of a gene known as KIAA0319 have been found to relate to reading ability in several different samples [25]. In one study, entitled ‘Strong evidence that KIAA0319 on chromosome 6p is a susceptibility gene for developmental dyslexia’, an association with a two-SNP haplotype was reported with \( p = 0.00003 \) [26]. This is indeed strong evidence. However, in a large sample, a low p-value does not entail a large effect size, a point that is often misunderstood. In this case, one variant was found in 39% of normal readers versus 25% of dyslexics. And a different variant was seen in 30% of controls and 35% of dyslexics \( (p = 0.02) \). Thus, prediction of dyslexia on the basis of this haplotype would be very inaccurate. In a screening context, where the proportion of the population with dyslexia is around 10%, it would follow that the majority of individuals with the ‘dyslexia risk’ haplotype would not be dyslexic, and the majority of dyslexic individuals would not have that haplotype. This does not mean that these effects are uninteresting; findings like this can pave the way for functional studies into the effects of gene expression on brain development [23]. But it does mean that talk of a ‘gene for dyslexia’ is misguided.

5. Twin and molecular studies give different estimates of heritability

Comparison of results of twin and molecular genetic studies reveals a puzzle: the genetic findings from molecular studies do not come close to explaining the high levels of heritability found in twin studies.

This problem of ‘missing heritability’ is not restricted to reading difficulties, but is seen even for physical phenotypes such as height [27], where measurement is straightforward and very large sample sizes have been used. There are essentially two ways of accounting for missing heritability: either the GWA method does not account for all aspects of heritability or twin studies overestimate heritability.

Recent advances in statistical methods have confirmed that traditional GWA does indeed underestimate overall genetic effects on a trait, and it has been argued that we should talk of ‘hidden’ rather than ‘missing’ heritability [28]. A GWA study involves looking at each DNA variant separately to see whether the degree of association surpasses a stringent threshold. This means that weak but genuine associations with the phenotype may get missed. To address this issue, methods have been developed for comparing similarity between individuals on an entire collection of SNPs and then relating this similarity metric to phenotypic similarity. This approach, genome-wide complex trait analysis (GCTA), which does not identify specific genes associated with disorder, has been shown to account for substantially more phenotypic variance than conventional GWA methods [29]. To date, there has been one GCTA study focused on reading ability, and, as with studies of other phenotypes, it found substantially more evidence for genotype-phenotype association than a conventional GWA study (variance accounted for by SNPs = 0.28), but less than was observed in a twin analysis based on the same sample (heritability = 0.75) [30]. While bearing in mind that the sample sizes available for studies of reading disability have been relatively small, and so may miss genuine but small effects, it would appear that here, as for other traits, some ‘missing heritability’ remains to be explained.

(a) Rare variants and copy number variants

As Gibson [31] noted, people have tended to contrast two models of inheritance. The ‘infinitesimal’ model, which is often assumed in dyslexia, is poetically described by Kirkpatrick et al. [32] as involving common variants that are ‘each Lilliputian in effect size, but together, are legion in number’ and add together to create risk. By contrast, in the ‘rare allele’ model, the effects of risk variants are large, but the individual variants each account for only a tiny proportion of cases. Of course, these are not mutually exclusive and both models may well apply to dyslexia. Both are difficult to verify in a GWA study, which will not detect very small effects of common variants, or large effects of very rare variants.

Great excitement was caused in 2006 when it was shown that copy number variations (CNVs) are remarkably common in the general population [33]. Although people mostly have two copies of each strand of DNA—one from each parent—everyone has segments of the genome where chunks of DNA are deleted or duplicated—CNVs. Those affecting non-coding stretches of DNA may have little or no effect, but if the duplications or deletions include genes, then the CNV is likely to have functional consequences. CNVs could potentially account for individual differences, but on the other hand, the fact they are common in the general population means that it would be dangerous to assume that a particular CNV found in an individual necessarily plays a role in their disorder [34].
To date, few studies have assessed the role of CNVs in the aetiology of common neurodevelopmental disorders. The frequency of large CNVs is increased in cases of intellectual disability or autism, but in dyslexia the rate is closely similar to that found in unaffected controls [35]. This does not mean that CNVs are never implicated in dyslexia, but they do not seem to be a common cause.

(b) Gene–gene interaction
Two genetic variants that individually cause only mild risk for disorder may exert a much greater effect in combination—if, for instance, they are involved in the same neural pathway. To date, this idea of a ‘double hit’ on a neural circuit has been developed in the context of individuals with relatively large structural genetic changes and severe phenotypes [36]; however, the same logic could apply to combinations of common variants leading to milder phenotypes. Two common variants that independently exert only a small effect might together be more detrimental in combination. Because MZ twins share the same DNA sequence, they will be identical for such gene–gene combinations, whereas in DZ twins, if genes are inherited separately, then the odds of the detrimental combination is lower than the odds of inheriting just one detrimental variant. Thus, in the presence of gene–gene interactions (epistasis), twin studies will overestimate heritability if an additive model is assumed.

(c) Gene–environment interaction
Gene–environment interaction refers to the situation where the impact of genetic variation depends on the environmental context [37]. One way of testing for such interaction is to consider whether different levels of a measured environmental variable are associated with different levels of heritability. This was done in a study using DeFries–Fulker analysis with the CLDRC sample by Friend et al. [19]. As well as reporting overall heritability, these authors subdivided twins according to parental educational level. Heritability of poor reading was higher for children with highly educated parents ($h^2_0 = 0.71$, 95% confidence interval: 0.55–0.88) than for those with less well-educated parents ($h^2_0 = 0.49$, 95% confidence interval: 0.32–0.66). The authors concluded that the effect of genes will be particularly evident in children who fail to learn to read despite good environmental support. This makes intuitive sense [21], but it is noteworthy that the finding was not replicated in another study that looked at the same question using slightly different methods [17].

The possibility that genetic effects may vary with the environment has implications for ‘missing heritability’. Most twin studies focus on twins who are growing up together. Consider the situation depicted in figure 3: in panel (a), we have a trait affected by both genes and shared environment, with no interaction between the two. Panel (b) shows a gene × shared environment interaction (G × C). In a twin study, overall estimates of heritability will be similar for both these situations. Because the twin study compares similarity of MZ and DZ twins, it effectively controls for G × C effects, because C is by definition the same for the two members of the twin pair. However, a GWA study, which focuses on the regression of phenotype on genotype, may have weaker power to detect association in the gene × environment interaction case, because much of the variability in the phenotype is caused by environmental variation.

6. The importance of the phenotype: clinical categories do not necessarily work in genetic analyses
In dyslexia, problems with written language typically are accompanied by subtle difficulties with processing of spoken language. For instance, children with dyslexia often have difficulty identifying the individual speech sounds in words (phonological awareness) even when no written language is involved [6]. Furthermore, if one follows up young children with specific language impairment (SLI), who have unexplained problems in talking or understanding, many of them have problems learning to read [38].

Dyslexia and SLI have traditionally been regarded as quite different conditions, one coming under the domain of educators and the other under the domain of speech–language therapists, yet there appears to be substantial overlap between them. Such observations have raised the question of whether dyslexia and SLI should be regarded as different manifestations of the same underlying problem [7], and whether there is genetic overlap in the risk factors for these conditions.

Rather than forcing genetic studies to use clinically approved phenotypes, we can use them to validate a diagnostic system or to suggest alternative ways of categorizing disorders. In family and twin studies, we can ask whether a phenotype ‘breeds true’, and in molecular studies, we can consider whether genes associated with risk for dyslexia are also associated with other disorders. We can also look at genetic influences on cognitive traits that appear to underlie the observed problems with reading or language.

An illustration of the latter approach comes from Bishop [39], who started with a group of children who were selected because they had oral language difficulties; a high proportion of them also had literacy problems. DeFries–Fulker analysis indicated high heritability of reading problems in this sample. The next question was whether the same genetic influence affected reading and non-word repetition, which
is used as a test of phonological short-term memory. In this task, the child hears a meaningless sequence of sounds (e.g. ‘konternaking’) and has to repeat it. Children with SLI have unusual difficulty with this task when there are three or more syllables in the non-word, and this is thought to reflect limitations of a short-term memory system that is specialized for remembering speech sounds [40]. To look at the genetic relationship between non-word repetition and reading ability, a modification of DeFries–Fulker analysis was used, in which one first selects as probands those children who did very poorly on non-word repetition, and then considers how well one can predict the co-twin’s score on the other measure, reading ability. This gives an index of bivariate group heritability, which indicates how far deficits in the two tasks have common genetic origins. In this case, the analysis supported the conclusion that impairments in reading and in non-word repetition were influenced by the same genes.

This conclusion was tempered, however, by data from a second sample of twins [39]. These were recruited as a general population sample. Surprisingly, and in contrast to other twin studies, heritability of reading ability was non-significant in this sample. It seemed possible that this might reflect the fact that the sample was biased towards lower socio-economic status, and the reading problems in the probands were relatively mild. The data, however, looked more coherent when non-word repetition ability was taken into account: essentially, the heritability of reading ability increased as level of non-word repetition skill declined. The conclusion was that reading ability is influenced by both genetic and environmental factors, but that where genes were strongly implicated, it was more likely that the reading problem would be accompanied by broader difficulties with oral language, and especially with poor non-word repetition.

A subsequent study with a new twin sample tested this idea further [41]. A sample of 6-year-old twins was divided into one subset where both twins had normal-range scores on non-word repetition, and another subset where one or both twins were impaired on non-word repetition. For twins where one or both children were poor at non-word repetition, the data pointed to a strong genetic influence on reading ability (i.e. MZ twins were much more similar than DZ twins). But for those where both twins did well on non-word repetition, then there was no evidence of any genetic effect—the amount of twin resemblance in reading skill was equivalent for MZ and DZ twins. It would be oversimplistic to suggest that there are two kinds of poor reader: one whose reading problems are environmentally determined and the other that is caused by genetic factors; in practice, both influences are likely to be present to different degrees. But we could draw two important conclusions from these findings. First, for some children, there is a biological risk that makes language and literacy learning especially challenging, and such children are characterized by a distinctive deficit on a simple test of non-word repetition. Second, quantitative measures of underlying processes may be better measures of phenotype than binary diagnostic categories when trying to uncover genetic origins of disorder [23].

7. The importance of the phenotype in molecular genetic studies

It can be tempting to think that molecular genetic studies are superior to twin studies, because they look directly at putative biological causes. However, a molecular study is only as good as the phenotypes that it uses; twin studies can be invaluable in identifying which phenotypes are heritable and hence which measures are worth including in large-scale molecular studies.

This point was demonstrated in a review of genetic influences on language and literacy impairment [42]. This concluded that deficient phonological short-term memory, as indexed by a non-word repetition task, was linked to specific genetic loci and appeared to play a role in reading impairment as well as in SLI. Subsequent work has converged on similar conclusions: non-word repetition deficits are seen in both dyslexia and SLI, and may be influenced by the same genetic mechanism [43]. It should be noted, however, that phonological short-term memory is just one cognitive correlate of dyslexia. An increasing body of work challenges the idea that we will find a single deficit that can explain reading problems in all children. It seems more likely that there are multiple cognitive deficits that are subject to genetic influence and that dyslexia is one outcome when a particular constellation of deficits occurs [44].

The same underlying cognitive deficits may lead to other neurodevelopmental disorders when they occur in a different combination. Finally, different languages may pose different challenges for children learning to read, depending on factors such as the regularity of letter–sound relationships [45].

8. Practical implications

Some researchers justify their search for genetic bases of disorder by proposing that they will find genetic variants that could be used in screening, but this is generally unrealistic. As noted above, even where robustly significant associations between genotype and phenotype are found, the effect size may be very small. This point can be illustrated with a recent study by Powers et al. [46,47]. In addition to identifying two sequences in a gene called DCDC2 associated with risk of reading or language problems, the authors noted an interaction with a risk haplotype of another gene, KIAA0319, such that children with risk haplotypes in both genes were particularly likely to have problems. The relevant data are shown in figure 4.

There are several points to note from this plot, bearing in mind that dyslexia would normally only be diagnosed if a child’s reading was at least 1.0 s.d. below age level.

(i) For children who have either KIAA0319 or DCDC2 risk haplotypes, but not both, the average score on reading measures is only slightly below average.

(ii) For those who have both risk haplotypes together, some of the reading/language measures give z-scores between −0.2 and −0.3 s.d. However, there is little evidence of deficit on non-word reading, which is often used as a diagnostic test for dyslexia.

(iii) The number of children with the two risk haplotypes together is small, around 2% of the population.

It follows that it would be ineffective to screen children for future dyslexia on the basis of these DNA variants. We may further note that there are many failures to replicate findings of association in this field, and it is possible that even those associations that seem solid may prove to be overestimated.
Figure 4. Mean scores on language, IQ and literacy-related measures by genotype. The sample is categorized according to whether they have a risk haplotype in DCDC2 and/or a risk haplotype in KIAA0319. Redrawn from figure 3a from corrected data provided by Powers et al. [47].

or to apply only to a specific population [48]. Meanwhile, we do have much simpler behavioural, familial and environmental measures that do a pretty good job of identifying young children at risk for reading difficulties [49,50]. It would only be worth developing a genetic screening test if we could demonstrate that it improved sensitivity and specificity when combined with behavioural measures.

9. A genetic aetiology does not mean a condition is untreatable

Could genetic findings be useful in intervention? All too often it is assumed that if genetic effects are found, the child will be untreatable. Yet, high heritability does not imply immutability: it implies that the range of environmental experiences that is usually encountered in everyday life does not have much impact on a trait, but says nothing about potential impact of novel environmental experiences. When, for instance, a child has the heritable myopia, we do not treat them as passive victims of their genetic destiny. Instead, they are given spectacles: an intervention that is outside the range of normal environmental experiences, but which is tailored to counteract the genetic effect. Similar logic can be applied in the case of dyslexia: if there are genetic variants that affect how children learn, we need to find out how they work to affect brain development and function. That will allow us to develop ways of intervening to overcome the problem—interventions that may need to be different from regular teaching experiences. We are still a long way from knowing how to do this, but genetic information points us towards the right path. It is not helpful to assume that all poor readers are the consequence of poor teaching and that additional or earlier reading instruction will fix the problem. We need studies that examine which kinds of reading instruction are most effective for children at high genetic risk, who often have disproportionate difficulties with aspects of speech sound analysis and associative learning that other children find easy. Genetic research does not lead us to write off children who are poor readers, but rather to recognize that they may need more individualized instruction tailored to their specific needs.

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