

Genes, Environment, and Reading Disabilities

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Introduction

First, what is the nature (cause or causes) of learning disabilities? The answer to this initial organizing question for the book will be addressed in the present chapter from a behavioral-genetic perspective on reading disability. It will be shown that reading disability often tends to run in families. Evidence from identical and fraternal twins will show that the familial pattern of transmission is due to both genetic factors and shared-family environment. Second, how are reading disabilities most effectively diagnosed? This question will be addressed from both a behavioral-genetic and medical-genetic perspective. It will be shown that some component skills in reading have stronger genetic influences than others, and that the degree of genetic influence may vary depending on characteristics such as disabled readers' IQ, phonological decoding, and age. Current evidence from analyses of disabled readers' DNA suggests the future use of genetic markers and ultimately specific genes for the early diagnosis of risk for reading disability. Third, how are reading disabilities most effectively remediated, and to what extent is remediation possible? It will be strongly argued that evidence for genetic influence, even very strong genetic influence in some cases, should not discourage our best efforts in remediation. However, the genetic evidence and results from

training studies will suggest that some extraordinary environmental intervention may be needed for many disabled readers.

Definition of “Reading Disability”

Large differences in literacy between countries and much of the differences within countries is due to cultural variation in instruction and reading practice. For example, one elementary school in an impoverished area of Denver recently showed average reading scores at the 13th percentile on a nationally normed test. Another school in a more affluent Denver neighborhood had average reading scores above the 80th percentile. It is likely that most if not all of this difference in reading performance is due to environmental factors. A major factor in this example may be the high number of children in the low-performing school whose first language was not English.

The behavioral-genetic studies with families and twins that will be discussed in this chapter have often limited the range of environmental influence on literacy by excluding children from known environments or language backgrounds that would obviously constrain reading development. These studies tend to select disabled readers with adequate schooling (for normal readers), average or above average levels of socio-economic status, and English as the first language. Individuals with some obvious environmental cause for a reading disability such as signs of brain damage (i.e., seizures) or poor school attendance

are excluded. Cases with extremely low IQ are also excluded. Thus, reading disability is unexpected from the individual's known environment and general intelligence. However, many relevant aspects of the family environment are not directly assessed in most studies. There may be considerable variation in factors such as parental expectations regarding literacy, television viewing habits, books in the home, children's pre-literacy activities, lead exposure, or some other environmental influence that is shared by the family but not always identified in behavioral-genetic studies. Analyses of data from identical and fraternal twins will show that differences in shared family environment do have a substantial influence on many cases of reading disability.

Because reading ability in the population is normally distributed (Rodgers, 1983; Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992), the severity criterion for reading disability is arbitrary and varies somewhat across studies. The studies reviewed below typically selected subjects who were more than about 1.5 standard deviations below the population mean (i.e., below the 10th percentile) in their sampling areas.

Rate of Familial Incidence and Prediction of Children's Reading Disabilities

Evidence from a number of family studies has shown that if a child is diagnosed with reading disability, there is a higher than normal probability that other family members will also be reading disabled (c.f., Finucci, Guthrie, Childs,

Abbey, & Childs, 1976; Hallgren, 1950; Gilger, Pennington, & DeFries, 1991; Vogler, DeFries, & Decker, 1985). The exact probability seems to depend on a variety of factors, including the severity of the child's reading disability and the type of assessment for other family members' reading skills. For example, when the parents' diagnosis for reading disability is based on self report, the familial incidence tends to be lower than when the diagnosis is based on the direct measurement of parents' reading skills (Gilger et al., 1991).

Most studies of familial incidence first diagnose a child with reading disability using a severity criterion that would identify 5-10% of children with normal intelligence and educational opportunity. Then the investigators attempt to use a similar severity criterion to diagnose reading disability in the parents. Evidence for the familial nature of reading disability is based on parental rates that are substantially above the 5-10% rate estimated for the population. Hollis Scarborough (personal communication, 1997) computed the average rate of reading disability among parents across eight family studies that included a total of 516 families. The rate across studies varied from 25% to 60%, with a median value of 37%. Thus, all studies found rates for reading disability among parents of reading disabled children that were significantly higher than expected in the normal population. The median proportion of reading disability among fathers (46%) was slightly higher than the median proportion among mothers (33%).

Results from the above family studies suggest that evidence for parents' reading disabilities could be used to predict a greater than normal risk for their

children. A few studies have attempted to directly estimate this prospective risk when parental disabilities are identified first. Finucci, Gottfredson, & Childs (1985) found that of 115 parents who had attended a special school for disabled readers when they were children, 36% reported that at least one of their children was reading disabled. Scarborough's (1990) prospective study of children with one or two reading-disabled parents found that 31% of the second-grade children were eventually identified as reading disabled by their schools. When the diagnosis of reading disability was based on Scarborough's extensive test results showing that reading was at least 1.5 standard deviations below the population mean, the rate was twice as high (62%). (A higher than school identification rate when children are actually tested is a common result in large epidemiological studies (Olson, Forsberg, & Wise, 1994; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990)). In contrast, Scarborough found that only 5% of children were reading disabled if both parents were normal readers.

In summary, the prospective studies clearly show that parents' reading disabilities predict a higher than normal rate of reading disabilities in their children (31%-62% vs. 5%-10%). While parental reading disabilities are not completely predictive of their children's reading disabilities, the substantially greater risk at least warrants very close monitoring of their children's progress in early language and literacy development.

Evidence for the partial familial nature of reading disability is necessary but not sufficient evidence for inferring genetic influence, because families also

share their environments. As discussed earlier, most family studies attempt to eliminate obvious environmental risk factors such as different native languages, low socio-economic status, and unusually poor schools. Nevertheless, there could be a variety of less obvious environmental risk factors associated with the reading disabilities of both parents and children. For example, parents with reading disability sometimes express the concern that they might environmentally transmit their negative attitudes about reading to their children. The next section reviews results from behavioral genetic studies with twins that attempt to separate the proportional influences due to genes and shared-family environment.

Twin Studies of Genetic and Environmental Influences on Reading Disabilities

The Twin Method

Twins who are raised together share their family environment. This is true for both monozygotic (MZ) “identical” twins and dizygotic (DZ) “fraternal” twins. However, the MZ and same-sex DZ twin pairs differ markedly in their genetic similarity. MZ twins develop from the same sperm and egg and are therefore genetically identical. DZ twins develop from two different egg-sperm combinations and share half their normally segregating genes, on average (Plomin, DeFries, & McClearn, 1990). Ordinary siblings also share half their segregating genes, but their different birth days may lead to less shared-environment influence compared to MZ and DZ twins. If MZ twins share their reading disabilities

significantly more often than same-sex DZ twins, it is assumed that the greater genetic similarity of MZ twins is responsible.

Of course, genes can only be expressed through their interaction with the environment. This interaction begins with the complex process of embryological brain development, and ultimately extends to genetic effects on the child's selection of their reading environment. For example, genetic effects on early brain development resulting in unusual difficulties in learning to read could ultimately cause a child's frustration and avoidance of reading practice.

In contrast, relative ease in learning to read, due to favorable brain development, could lead to much more reading practice and enjoyment of literature. Such child-selected environmental differences could have a strong impact on reading development. Because the cause of this environmental selection was some prior genetic effect, behavioral-genetic analyses include any effects of this or any other genotype-environment correlation in their estimation of total genetic influence. Implications for the remediation of genetically influenced reading disabilities will be considered in the final section of the chapter.

Twin Concordance Rates for Reading Disability

Several early twin studies of reading disability found that MZ twin pairs shared their reading disabilities significantly more often than DZ twin pairs (Bakwin, 1973; Halgren, 1950; Zerbin-Rudin, 1967). When an MZ or DZ twin pair shares a disorder, such as reading disability, diabetes, or schizophrenia, the

twin pair is referred to as “concordant” for the disorder. If only one twin of a pair has the disorder, the pair is referred to as “discordant”. In Halgren’s classic study, the concordance rate for MZ and DZ twins was 100% and 52% respectively. This extreme result suggested that nearly all cases of reading disability are due to genetic factors. Other studies have reported smaller differences in MZ - DZ concordance rates, but with the exception of Stevenson, Graham, Friedman, & McLoughlin (1987), all find a significantly higher concordance rate for MZ pairs (DeFries and Alarcon, 1996).

The ascertainment of disabled readers in the early concordance studies may have been biased by a higher tendency to report concordant than discordant pairs (Harris, 1986). The ongoing Colorado twin study of reading disability reduces this potential referral bias by ascertaining reading disabled twins in grades 3-12 from school performance records and then giving the twins an extensive battery of tests in the laboratory (DeFries, Filipek, Fulker, Olson, Pennington, Smith, & Wise, 1997). DeFries and Alarcon (1996) reported that the unbiased concordance rate for reading disability in this sample is currently 68% for 186 MZ pairs and 38% for 138 DZ pairs. This is a highly significant difference, but it is somewhat smaller than the extreme differences found in some earlier and potentially biased studies.

Differences between MZ and DZ concordance rates can provide evidence for significant genetic etiology, but they do not provide precise estimations of the relative magnitude of genetic and environmental influences. Also, concordance rates are insensitive to the degree of twins’ reading deficits below and above the

severity criterion for a categorical diagnosis of reading disability. When reading disability is defined as the lower 10% of the population in reading skills, there is still a very large range of reading deficit within this group. Also, there is a large range of reading ability between the criterion level and the normal mean. A regression analysis developed by DeFries and Fulker (1985) is sensitive to this variability above the criterion level. Their basic regression model yields direct estimates of the proportion of genetic influence on the disabled group's reading deficit.

Assessment of Genetic Influence from Cotwin Regression to the Population Mean

The DeFries and Fulker (1985) analysis defines reading disabled twins as those who fall below a selected severity criterion, usually the lower 10th percentile relative to a normal sample of twins. In this type of behavioral genetic analysis, twins that meet the selection criterion for reading disability are called "probands". In cases where a twin pair is discordant for reading disability (one member of the pair is disabled and the other is not), the twin that does not meet the criterion for reading disability is called the "cotwin".

The DeFries and Fulker (1985) analysis compares the degree to which reading performance of the MZ and DZ cotwins regresses toward the population mean. The population mean is defined by the average performance level in a comparison sample of normal twins. Cotwins' regression to the population mean is indicated by the amount that their scores fall above the criterion level for reading

disability, toward the population mean. To aid in understanding how the analysis works, three extreme examples of MZ and DZ cotwin regression are given below that would indicate exclusive influence from (1) genetic factors, (2) shared environment, or (3) non-shared environment.

If reading disability is completely due to genetic influence, both members of the genetically identical MZ pairs would be affected probands: There would be no cotwin regression to the population mean. However, that result alone would not prove a genetic influence, because MZ twins also share their family environment. A comparison must be made with DZ twins, who also share their family environment, but who share only half their segregating genes on average. If reading disability were completely due to genetic influence, many of the genetically dissimilar DZ pairs would have a cotwin that did not meet the selection criterion. The expectation for complete genetic influence is that MZ twins show no regression and DZ cotwins regress half way to the population mean, on average.

If reading disability were entirely due to shared-family environment, both MZ and DZ pairs would have two probands and no cotwin regression to the mean. This is because both types of twin pairs share their family environment.

The third extreme example is that if reading disability were due entirely to some non-shared environmental factor, both MZ and DZ cotwins would be expected to average nearly complete regression to the population mean, constrained only by the defined rate of reading disability in the population. Non-

shared environmental factors among twins could include such things as selective gestational or birth problems, non-shared illnesses, or head injury. Test error is also included in behavioral-genetic estimates of non-shared environment.

The actual patterns of cotwin regression to the mean for reading and related skills fall between the above extreme examples and yield proportional estimates of the balance of genetic (\underline{h}^2_g), shared environment (\underline{c}^2_g), and non-shared environment (\underline{e}^2_g) influences. The subscript “g” for each of the three estimates indicates their reference to the average group deficit. The estimates from these behavioral-genetic analyses do not specify proportional influences for any individual within the deviant group.

When the DeFries and Fulker (1985) regression analysis was recently applied to a composite measure of reading that combined the Peabody Individual Achievement Tests (PIAT, Dunn & Markwardt, 1970) for reading comprehension, word recognition, and spelling, the estimated proportion of genetic influence (i.e., heritability) on the group deficit was 56%, or $\underline{h}^2_g = .56$ with a standard error of .09 (DeFries & Alarcon, 1996). Thus, there is a 95% probability that the true group \underline{h}^2_g for the composite PIAT measure of reading disability is between .38 and .74.

It should be noted that the above estimate for genetic influence on the group deficit in reading is specific to the Colorado sampling constraints. If a broader range of reading environment (i.e., poor inner-city schools) had been included in the sample, it is likely that the proportional influence of shared-

environment would have been higher, and that from genetic factors would have been lower for the average group deficit. On the other hand, if the range of the twins' reading environment had been more constrained, it is likely that genetic influence would be higher. An average population estimate of genetic influence on individual differences in a behavior is jointly influenced by the range of relevant genetic variation and by the range of relevant environmental variation.

Genetic and Environmental Influences on Specific Skills in Reading and Language

Olson, Forsberg, and Wise (1994) extended the DeFries and Fulker (1985) genetic analysis to derive estimates of the proportion of influences from genetic factors (h^2_g), shared-family environment (c^2_g), and non-shared environment (e^2_g) for several separate reading and language skills. Probands (twins below the 10th percentile) were separately selected for each of the PIAT tests of word recognition, spelling, and reading comprehension that DeFries and Alarcon (1996) used in their composite measure. Proband membership in the three deviant groups largely overlapped because of the positive correlations between the PIAT tests. However, the correlations were far enough from 1 to allow for some differences in proband group membership and in average genetic influence across the tests. The results from the separate PIAT analyses are presented in Figure 1. Both word recognition and spelling had substantial and similarly high levels of genetic and shared-environment influence. However, for reading comprehension, shared-environment influence was high (52%) and genetic influence was low (27%). The

specific vocabulary and world knowledge needed to correctly answer the PIAT comprehension questions may have been strongly influenced by the twins' shared school and home environment. In contrast, the rate of growth in word recognition and spelling appears to have been more constrained by genetic factors. Which of these measures is most appropriate for the diagnosis of reading disability? If the interest is in reading disabilities with significant genetic influence, the word recognition and spelling measures would be most useful.

Insert Figure 1 about here

Olson, Wise, Conners, Rack, and Fulker (1989) examined genetic influences on group deficits in word recognition, phonological decoding (oral reading of nonwords), orthographic coding (selection of words from word-pseudohomophone pairs such as rain-rane), and a measure of phoneme awareness that was similar to "Pig Latin". In this small twin sample, there were significant genetic contributions to the group deficits in word recognition, phonological decoding and phoneme awareness, but not in orthographic coding. It was argued that deficits in the orthographic task may have been more influenced by shared-environment differences in print exposure (Stanovich and West, 1989). However, in larger twin samples analyzed by Olson et al (1994) and in more recent unpublished analyses, it is clear that there is also substantial genetic influence on the group deficit in orthographic coding.

The current estimated levels of genetic influence depend somewhat on the criteria for proband selection. The estimates in Figure 2 are based on the selection of probands on the individual variables regardless of their school history for reading disability. When proband selection is further constrained by school history as in DeFries and Alarcon (1996), Genetic influence is about 10-15 percentage points higher and shared-environment influence is about 10-15 percentage points lower. It is not clear which approach is most appropriate, so the most conservative approach for genetic influence is used here.

Insert Figure 2 about here

Current estimates of genetic influence are substantial and statistically significant for group deficits (-1.5 SD) in PIAT word recognition (204 MZ, 151 DZ pairs), phonological decoding (187 MZ, 124 DZ pairs), orthographic coding (162 MZ, 114 DZ pairs), and phoneme awareness (137 MZ, 101 DZ pairs). It appears that genetic influence might be somewhat higher for phonological decoding and phoneme awareness compared to word recognition and orthographic coding, but the contrasts are not statistically significant. The influence of shared environment is statistically significant for all variables except phoneme awareness. Shared-environment influence is higher for the group deficit in word recognition compared to the other variables. This may reflect a greater role for shared-environment differences in print exposure in the development of word recognition.

Phonological skills and the high precision of orthographic representations required for correct responses in the orthographic choice task appear to be less influenced by shared environment.

Gayán, Datta, Castles, and Olson (1997) noted that the heritability estimates for word recognition and phonological decoding seem to depend on whether fluency is included as a part of the assessment. An experimental measure of time-limited word recognition (correct responses had to be initiated within 2 seconds) yielded a higher heritability estimate for the group deficit ($h^2_g = .55$; $c^2_g = .40$) compared to the untimed PIAT test of word recognition ($h^2_g = .44$; $c^2_g = .47$). Similarly, the high heritability and low shared-environment estimates for phonological decoding in Figure 2 ($h^2_g = .58$; $c^2_g = .28$) are based on deficits in the combined z scores for percent correct and latency on correct trials. In comparison, heritability for the percent of nonwords read correctly was much lower and shared-environment was higher ($h^2_g = .41$; $c^2_g = .45$). It seems that shared environment plays a stronger role in limiting disabled readers' accuracy in word recognition and phonological decoding, while deficits in accuracy and fluency combined are relatively more influenced by genetic factors. If the diagnosis of genetically based disabilities in word recognition and phonological decoding is a goal, measures of speed should be included in the test battery. The implication of these results for the remediation of reading disabilities will be considered in the final section of the chapter.

Genetic Covariation and Independence across Measures

Are the same genes involved in disabled readers' correlated deficits in word recognition, phonological decoding, orthographic coding, and phoneme awareness? The answer is partly yes and partly no. Olson et al (1994) reported significant genetic covariance among the measures in bivariate extensions of the DeFries and Fulker (1985) twin regression model. Gayan and Olson (in press) performed a different type of genetic analysis to estimate specific and shared genetic influences for individual differences on the measures. The results showed a common genetic influence across all measures, consistent with the earlier results from bivariate analyses of group deficits. However, there were also specific, non-shared genetic influences for individual differences in each of the measures. This significant independent genetic variance for each of the measures indicates that differences in disabled readers' profiles of component reading and language skills may have a partly genetic basis.

Converging on Genetic Influence for Individual Disabled Readers

It is important to reiterate that the above behavioral-genetic twin analyses do not provide information about the balance of genetic and environmental influences on any individual with reading disability. These studies only estimate the average relative influences for the reading-disabled group. Within the group, some disabled readers' deficits may be primarily due to their genes, some may be primarily due to shared environment, and some may be due to a more equal influence from both factors. However, we can look for interactions between other

subject variables and the average degree of heritability for reading deficits. If significant interactions are found, this information could be used to make more accurate probability statements about the degree of genetic etiology for an individual's reading disability.

Gayán, Datta, Castles, and Olson (1997) found that when they assessed the heritability for the disabled group's deficit in PIAT word recognition, depending on the probands' level of phonological decoding, probands with relatively low phonological decoding had a significantly higher average genetic influence on their deficit in PIAT word recognition, compared to probands with relatively high phonological decoding. Gayán et al suggested that relatively severe deficits in phonological decoding are strongly influenced by genetic factors that place a genetically based constraint on the growth of word recognition. Extremely poor phonological decoding would lead to frequent decoding errors when children independently attempt to read unfamiliar printed words. In contrast, disabled readers with relatively good phonological decoding compared to their word recognition may have deficits in word recognition that are more due to shared-environment deficits in print exposure.

Olson, Forsberg, Gayán, and DeFries (in press) proposed a similar explanation for an apparent difference in genetic influence on deficits in word recognition, depending on subjects' full-scale IQ scores. In this analysis, subjects with relatively high IQ compared to their word recognition had a stronger average genetic influence. Shared environment was a relatively stronger influence for

subjects whose IQ scores were lower (see Figure 3). Olson et al. suggested that a poor home and educational environment could be jointly responsible for the concurrent expression of low IQ and low word recognition. In contrast, disabled readers with higher IQ scores were more likely to have a relatively good educational environment, and their failure in reading would be more likely due to genetic constraints.

Insert Figure 3 about here

The final example of individual variation in genetic influence is a fascinatingly complex interaction between genetic influence, age, and measures of reading and spelling reported by DeFries, Alarcon, and Olson (1997). They found that genetic influence on PIAT word-recognition deficits tends to decline across the 8 year to 20 year age range of the cross-sectional Colorado twin study, while genetic influence on PIAT spelling deficits tends to increase with age. It was suggested that correct reading of the high-level items in the PIAT word recognition test was more dependent on amount of print exposure, compared to the shorter, more phonologically regular, and orally familiar items at lower levels of the test. In contrast, the higher-level spelling items in the PIAT test may demand a level of precision in subjects' orthographic representations that is more constrained by genetic factors.

The three examples given above indicate that estimates of the likely proportional genetic influence on individual disabled readers' reading and spelling deficits can be improved through knowledge about other subject characteristics. However, a direct analysis of disabled readers' DNA may ultimately provide much more precise knowledge about likely genetic influences on an individual's reading disabilities. Some progress toward this goal has already been made. Cardon, Smith, Fulker, Kimberling, Pennington, & DeFries (1994) analyzed the DNA from same-sex DZ twins in the Colorado study and from an independent sample of extended families that included several disabled readers. Cardon et al. found that in both samples, there was significant evidence for the linkage of many cases of reading disability to the HLA (immune system) region of chromosome 6. This apparent linkage needs to be replicated in additional samples before we can have strong confidence in its validity and have a good estimate of the proportion of reading disabilities that may be influenced by the gene or genes in this area. Current linkage analyses of a new set of DZ twins from the Colorado study suggest that the results of the earlier study will be replicated, and that the strongest linkage to this area is for probands with the most severe deficits in orthographic coding, phonological decoding, and phoneme awareness.

At least one independent study has reported linkage to a similar region of chromosome 6 (Grigorenko, Wood, Meyer, Hart, Speed, Shuster, & Pauls, 1997). The strongest linkage in this region was for deficits in a reading-related measure of phoneme awareness. Deficits in word recognition were more strongly linked to a

region on the short arm of chromosome 15. The authors argued that deficits in component skills in reading and related language processes are linked to different regions of the genome. This is certainly possible in view of the partially independent genetic effects found through behavioral-genetic analyses for different component reading skills (Gayan & Olson, in press). However, it appears from the Grigorenko et al. report that differences in the strength of linkage for phoneme awareness and word recognition would not be significant at either locus. Cardon et al (1994) did not find significant linkage for reading disability on chromosome 15, but an earlier study by Smith, Kimberling, Pennington, and Lubs (1983) did find significant linkage here. It is not entirely surprising that different linkages emerge in different samples. It seems likely that there will be more than one important genetic contribution to reading disability across the population (Smith, Kimberling, & Pennington, 1991). Whole-genome scans made possible by the human genome project may help locate several additional regions of the genome that account for some significant proportion of genetically influenced reading disabilities.

Following the confirmation of linkage for reading disability to the HLA region of chromosome 6 and the short arm of chromosome 15, a search through the millions of base pairs in these regions must be undertaken to find the gene(s) responsible, identify the protein(s) that are coded by the gene(s), and ultimately understand the developmental pathway from gene(s) to brain structure and function that lead to genetically-based reading disabilities. Prior to our complete

understanding of the specific gene(s) and their developmental pathway, it may soon be possible to use reliable DNA markers that are close enough to the responsible genes to identify children who are at a high genetically-based risk for reading disability. This information could then be used to begin preventative measures before the child experiences reading failure and frustration in school.

Implications of Behavioral-Genetic and Linkage Results for Education

Educators are often wary of behavioral-genetic studies. They may be concerned that evidence for genetic influence will be mistakenly used to account for the poor average reading performance of disadvantaged racial or ethnic groups. They may also be concerned that evidence for genetic influence will be used as an excuse for educational policy makers to give up on disabled readers, assuming that their problems with reading are absolutely determined by their genes and without hope for remediation. It must be emphasized that these are clear mistakes in the interpretation of the evidence for genetic influences on reading disabilities.

First, the behavioral-genetic evidence has nothing to say about differences in reading performance between different racial or ethnic groups, because it is impossible to disentangle the effects of the substantial environmental differences between these groups (e.g., different dialect, different first language, SES, etc.). Second, even if the genetic influence on some individual reading disabilities proves to be very strong, this only implies that some extraordinary environmental

intervention may be required. Some day this intervention may be partly biological, perhaps through the manipulation of neural transmitters, as in the successful treatment of some attention-deficit disorders with Ritalin. At present, the behavioral genetic evidence has some important implications for educational policy.

One implication is that some children may be poor readers in spite of their parents' and schools' provision of an environment that is quite adequate for reading development in normal children. Many parents and their children with reading disability feel a high level of guilt about the problem. The parents may feel that they have failed to provide their children with a "good" environment for reading development. While this may be true in some cases of reading disability, it is clear that there are strong genetic influences in many cases. Many parents are often quite relieved when they learn that the problem may have originated in their genetic makeup rather than in their failure to provide a good environment for their children.

A "good" or "normal" environment for reading development may not be nearly enough for some children to reach a functional and enjoyable level of literacy. The direct manipulation of brain processes through medical intervention is a future possibility, but we must rely on our best educational efforts now. These efforts may include special types of pre-reading and early reading instruction and they may also need to include a significantly greater amount and intensity of accurate reading practice to compensate for some disabled readers'

biological liabilities. Some of this additional support might be efficiently provided through computer-based exercises and reading practice (Wise & Olson, 1995). The behavioral-genetic evidence can help parents and educators understand why this extra effort is needed for many children with reading disability.

I will close the chapter with some reflections on how results from recent training studies with disabled readers may be related to some of the genetic influences that have been described above. Recall that one likely pathway for genetic influence in many children is from early failure and frustration with reading to a low level of reading practice. Thus, some children may be initially slowed in their reading development because of prior biological and/or environmental constraints, and their rate of growth is slowed further by the avoidance of reading in favor of more enjoyable and less frustrating activities. Interventions that provide well-motivated, structured, and accurate reading practice may therefore show significant improvement for many disabled readers, including those who get off to a bad start because of negative genetic influence.

The benefits of structured and accurate reading practice for disabled readers in the schools were clearly shown in a study by Wise and Olson (1995). Children in the second to fifth grades who were in the lower 10 percent of their class in word recognition were trained for a half hour each day over a semester, for a total of 25 hours. This training occurred during times that the children would otherwise be in their regular reading class. A common core of two different training programs described below included the reading of interesting stories on the

computer. The stories were selected so that most of their words were not too far beyond the child's reading level. Accurate reading of difficult words was supported by synthetic speech when children targeted the words with a mouse. This structured reading experience resulted in an average gain over the semester of about ten standard-score points on several measures of word recognition. This was an impressive improvement for these children during a relatively brief training period, although most were still well below the mean for their class. Our observations suggested that the amount of accurate reading during the training periods was substantially greater than most of these disabled readers would have experienced in their regular reading class.

Unfortunately, follow-up testing one and two years after the end of training indicated that subjects' growth rate in word recognition returned to the slow rate experienced before training (Olson, Wise, Ring, & Johnson, in press). Their structured practice in accurate reading was clearly beneficial during training, but apparently it did not improve most children's poor reading habits after training. More needs to be done to ensure that disabled readers maintain a high level of reading practice over the long term and achieve a level of reading fluency that is high enough to support their continued enjoyment of reading. Olson et al. noted the need to provide much longer training in the schools to bring disabled readers closer to the level of their peers. It will also be important to work with the families of disabled readers to boost their accurate reading practice at home, possibly with the assistance of computer-based reading programs in the home.

Other results of the Wise and Olson (1995) study may be understood with reference to the strong genetic effects reported earlier for deficits in phonological decoding and phoneme awareness. One of the two training conditions explicitly targeted the subjects' deficits in these skills. The other group practiced comprehension strategies while reading both on and off the computer. Subjects in the phonologically trained group showed substantially greater improvement in their accuracy for reading nonwords and in several measures of phoneme awareness. Thus, it seemed that we had remediated much of their deficit in phonological skills. Unfortunately, this apparent improvement in phonological skills was not accompanied by a similar expected advantage in growth for disabled readers' word recognition during training. At the end of training, there was a trend favoring the phonological group on the untimed PIAT measure of word recognition, but the other group was significantly better on our measure of time-limited word recognition. Olson, Wise, Johnson, and Ring (in press) reviewed several other recent studies that found a similar dissociation between growth in disabled readers' phonological skills and word recognition when similar treatment comparisons were made at the end of training.

Follow-up tests for the Wise and Olson (1995) subjects, one and two years after training, found no significant differences in any of the word recognition measures, although the phonological group retained a significant superiority in nonword reading accuracy and phoneme awareness at least through the first year following training (Olson et al., in press). These results seem inconsistent with the

view that better phonological decoding skills should provide a “self-teaching” mechanism that would support more rapid growth in word recognition (Share, 1995).

What are we to make of these results from a genetic point of view?

We have seen that individual differences in phonological decoding and phoneme awareness are phenotypically and genetically correlated with reading and spelling in the population (Olson et al., 1994; Gayan & Olson, in press). In most normal readers, phonological skills emerge as a consequence of learning how to read, even if they are not given much explicit instruction. In disabled readers, phonological skills tend to lag significantly behind their development in word recognition, unless explicit instruction is given (Rack, Snowling, & Olson, 1992). However, raising disabled readers’ performance in nonword reading and phoneme awareness to a level consistent with or better than expected from their word recognition does not mean that we have created the same reading process seen in younger normal children at the same level of reading development. The normal children’s integration of their phonological skills in the reading process may be much more natural and automatic, requiring less explicit attention for their use during fluent reading. In contrast, the phonological awareness and decoding skills learned by disabled readers in many training programs may be less well integrated and automatized (Sternberg & Wagner, 1982).

The high shared genetic influence on deficits in fluent phonological decoding and reading may help explain why it is difficult for many training

methods to penetrate to the level of phonological processing that may be responsible for this genetic covariance. New and more intense training methods may be needed to improve disabled readers' automatic phonological processes, facilitate their transfer to fluent reading, and implement the automatic "self-teaching" function that seems to support the rapid growth of word recognition and spelling in normal development. The amount of print exposure required for the development of fluent reading may still be greater than in normal readers, but less than would be required without the strengthening of disabled readers' automatic phonological processing.

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Figure legends

Figure 1. Percent of genetic, shared environment, and non-shared environment influences on disabled readers' group deficits in PIAT word recognition, spelling, and reading comprehension.

Figure 2. Percent of genetic, shared environment, and non-shared environment influences on disabled readers' group deficits in PIAT word recognition, phonological decoding, orthographic coding, and phoneme awareness.

Figure 3. Percent of genetic, shared environment, and non-shared environment influences on disabled readers' group deficits in word recognition across three levels of WISC-R IQ.