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## Behavioral Genetic Approach to the Study of Dyslexia

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### Abstract

**Objective**—Dyslexia is a prominent focus of practitioners, educators, and researchers due to the myriad consequences of failing to read proficiently. The aim of the current study was to provide a brief overview of how twin studies can offer insight on the etiology of many human behaviors and disorders including dyslexia, discuss common misconceptions regarding findings from behavioral genetic studies, briefly review the evidence on the relationship between genes, environment, and dyslexia, and finally present some findings from a large-scale twin study on reading and dyslexia.

**Method**—Participants were twins from a large ethnically and socioeconomically diverse twin sample in an ongoing longitudinal study of reading and dyslexia. Heritabilities of reading ability and dyslexia were calculated for 1,024 first grade twins on a standardized reading measure. Children were identified as dyslexic if they scored at the fifteenth percentile or below on a reading measure.

**Results**—Relatively high heritabilities were observed for both reading ability and dyslexia indicating substantial genetic influences. Further, results indicated some overlap of genetic factors influencing reading ability and dyslexia.

**Conclusions**—Behavioral genetic studies offer a means of understanding the etiology of dyslexia. The current study extended research to a more diverse sample than extant studies and found lower heritability estimates of reading ability and dyslexia, but a similar pattern of results indicating genetic overlap. Twin studies provide perspective for discoveries of specific genes involved in dyslexia by quantifying the amount of variance waiting to be accounted for by genes while simultaneously providing an impetus to continue working on efforts for environmental intervention.

### Keywords

dyslexia; heritability; twins; genetic

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Dyslexia, or specific reading disability, is often defined as an unexpected problem with learning to recognize printed words (1) that is not due to general intellectual impairments, sensory impairments, or lack of opportunity to learn from competent instruction (2). Current definitions of dyslexia assume that the disorder is neurobiological in origin. Though once thought to be primarily due to deficits in the visual system, there appears to be a consensus that the signs and symptoms of dyslexia are caused by a deficit in a specific language skill that is responsible for the processing of phonological information (1). Most estimates of the prevalence rate of

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dyslexia in school-age children range between 7% to 15% (3,4,5), and the negative correlates of the disorder extend well beyond childhood. For instance, a 20-year follow-up of students diagnosed with dyslexia found that these individuals achieved a lower socioeconomic status (SES) than their parents had and an increased proportion of them reported a diagnosis of a mental illness compared to their non-disabled peers (6). A review of the literature on long term outcomes for students diagnosed with learning disabilities concluded that these students are less likely to receive or complete postsecondary education than their counterparts without disabilities and that employment rates are lower for persons with learning disabilities than for those without a learning disability (7).

There have been numerous studies of reading and dyslexia that have used many different methods to investigate both normal reading development and disorder. Brain imaging studies have used positron emission topography (PET), functional magnetic resonance imaging (fMRI), and magnetoencephalography (MEG) to investigate brain activation profiles of children with dyslexia (8,9,10). Eye tracking studies (11), cognitive neuroscience studies (12), individual differences studies (13) and sociocultural studies (14) have all provided a different framework for investigating reading and reading problems. An additional approach is behavioral genetics, the focus of this paper. Behavioral genetics is the field that examines the relative contributions that genes and environments make in explaining differences in behavior. One particularly powerful method in behavioral genetics is the study of twins (15). In this paper, we provide a brief overview of how the study of twins can shed light on the etiology of many human behaviors and disorders including dyslexia, discuss common misconceptions regarding findings from behavior genetics studies, briefly review the evidence on the relationship between genes, environment, and dyslexia, and finally present some findings from a large-scale twin study on reading and reading problems.

## Overview of Twin Methodology

Twins can be thought of as a naturally occurring experiment (16) that offers a unique and informative means by which to study phenomena such as reading and dyslexia. By taking advantage of the fact that monozygotic (MZ) twins share virtually all of their genes while dizygotic (DZ) twins share about half their segregating genes on average, the relative influences of genes and environment upon behavior can be estimated. By comparing the similarities within MZ and DZ twin pairs, the observed variability in a given behavior can be decomposed into additive genetic (A), shared or common environment (C), and non-shared or specific environment (E) components. An additional source of variance is non-additive genetic effects which reflect the interactions among genes, however, those are effects not typically found in studies of reading. Shared environmental factors are those which twin pairs have in common and can include variables such as books in the home, socioeconomic status, and school environment. Non-shared environmental factors are unique to individuals such as injuries, different friends, different classrooms within a school, or other experiences not shared with the cotwin. Twin studies offer a means of estimating the magnitude of environmental and genetic influences associated with individual differences in a behavior. The magnitude of the genetic influence, or heritability, is the proportion of total observed variance due to genetic factors. Estimates of heritability can be computed by inspecting the differences in MZ and DZ correlations. Similarities in MZ twins are due to shared genes and shared environment while similarities in DZ twins are due to only about half shared genes and shared environment. Hence, doubling the difference in similarities (correlations) between MZ and DZ twins results in an estimate of heritability. Estimates of shared environment and non-shared environment can also be obtained from these observed MZ and DZ correlations. Shared environment is calculated by doubling the DZ correlation and then subtracting the MZ correlation. Non-shared environment, which causes twins to be dissimilar, is the proportion of variability not accounted for by either genes or shared environment and also includes measurement error. While these

computations are illustrative and often quite accurate, more sophisticated structural equation model-fitting techniques offer opportunities for more complex modeling and increased precision of estimates. Each one of these estimates (A, C & E) can range from 0, meaning no influence, to 1.0, meaning 100% of the variation seen in the behavior is due to either genes or environmental influences, but estimates of 0 or 1.0 are relatively rare because both genes and environment contribute to complex behaviors like reading and dyslexia.

Behavioral genetics includes analytic techniques to assess the proportions of the genetic and environmental influences across the continuum of normally distributed traits such as reading ability. These sorts of analyses offer insights into the relative influences of genes and environment across the broad range of the trait. However, in contrast to continuously measured traits, disorders are typically viewed as either being present or absent rather than measured on a continuum. Concordance rates of affected twins and their cotwins can be calculated for dichotomous diagnoses to gauge the similarity of MZ and DZ twins and the extent to which genetic factors may affect the disorder. When a disorder is conceptualized as an extreme end of a continuous trait as is the case for dyslexia, group heritability can be calculated. Group heritability is estimated using DeFries-Fulker extremes analysis (DF; 17,18) which simultaneously considers dichotomously measured diagnoses and continuously measured traits to assess the extent to which genetic factors influence proband group membership.

While each of these heritability estimates is informative in and of themselves, taken together they can answer another question. It is possible that reading ability is influenced by a particular set of genes while dyslexia is affected by different genetic factors. Conversely, the same genetic factors may be at play across the spectrum of reading ability, including dyslexia. A group heritability estimate (from DF) comparable in magnitude to an individual differences heritability estimate can indicate substantial genetic overlap of reading and dyslexia (19).

Twin studies can go beyond answering the question of how much genetics plays a part in a particular disorder or skill such as reading. Genetically sensitive designs are able to explore how genetics and environments influence multiple skills concurrently and longitudinally. Multivariate extensions of the above described twin analyses allow for assessment of overlap in genetic and environmental factors on multiple skills. As an example, analyses of reading-related skills including phoneme awareness, word recognition, phonological decoding, and orthographic coding demonstrated bivariate overlap of genetic influences on these skills ranging from .55–.97 (20). In addition to genetic overlap across skills, genetic overlap within a trait can be assessed. Ability across the range of skill can be investigated to determine whether disabled individuals represent extremes of normal variation or whether disability is etiologically distinct. Researchers have begun to explore genetic overlap both between and within learning abilities, finding substantial overlap but independence as well (19).

Genetically sensitive longitudinal studies offer opportunities to investigate the shifts and stability in genetic and environmental influences as reading skills develop over time. A longitudinal study of 8–16 year old twins (mean age 10.3) at initial testing and follow-up five years later has found both genetic stability and common genetic influences for reading (21) and dyslexia (22). Currently, there is a large International Longitudinal Twin Study (ILTS) of preschoolers investigating prereading skills and continuing into early school years with the goal of better understanding the etiology of the development of prereading to reading skills as children learn to read (23). The Florida Twin Project on Reading (FTP-R) is a new study based on a large ethnically and socioeconomically diverse twin sample of kindergarten through fifth graders. Ongoing collection of data each academic year offers opportunities to analyze continuity and change in genetic and environmental influences.

Beyond parsing proportions of variance into genetic and environmental influences, advances in behavioral genetic methodology are allowing study of gene x environment interactions. It is clear that reading and reading difficulties are highly heritable (16), however it is less clear how environmental factors moderate genetic effects on reading. Identifying moderating environmental effects offers sources of variability that can potentially be manipulated and impact reading outcomes. For example, a recent study of the role of teachers found that higher quality teachers allowed students to more fully realize their genetic potential in early reading (Taylor JE, Roehrig AD, Soden Hensler B, et al. submitted manuscript, 2009).

Studies employing genetically sensitive designs can lead to a more complete understanding of the etiology of learning ability and disability as well as other disorders. An important implication of genetic research is to offer etiological evidence to support diagnostic classifications rather than reliance on presentation of symptoms alone. More sophisticated understanding of genetic vulnerability to learning difficulties can inform optimal treatments and ultimately maximally positive outcomes.

## Misconceptions about Behavioral Genetic Research on Reading and Dyslexia

Although behavioral genetic research can offer much insight into the etiology of dyslexia, this evidence has not been uniformly welcomed by the educational field. This is in large part due to a number of misconceptions regarding the sort of evidence offered by behavioral genetics research and the conclusions that can be drawn. It is interesting to note that although the nature versus nurture debate has long been considered settled – we are neither *tabula rasa* nor predetermined by our genes, rather the interplay between nature and nurture contributes to variability in nearly every aspect of life – there seems to be a delayed acceptance of the role of genetics in academic achievement and learning disabilities (24).

A potential reason for this delay might stem from the misunderstanding of behavioral genetic research findings. One common misconception is that heritability and environmental estimates apply to specific individuals. This is incorrect. These values are estimates from a sample that apply to the population from which they were obtained. For example, a heritability estimate of .50 for a particular reading assessment implies that on average approximately half of the variability seen among people in that population in that reading task is due to genetic factors. It does not mean we can attribute genes or environment as the reason why any one person in the sample is reading differently from another. Estimates of heritability and environmental influence are estimates for a population, not estimates for a given person.

Another related misconception is that a high heritability estimate for a given behavior means that the behavior is hardwired and unchangeable. Estimates of the relative influence of genes and environments provide a snapshot at any one particular time. These snapshots can differ across samples drawn from different populations or from samples drawn within a single population over very long periods of time. Because the estimates of genetic and environmental influences must sum to (and cannot exceed) 100%, changes in the environment can influence estimates of heritability. High estimates of heritability for a given behavior should not be taken to mean that the environment can have little influence on that behavior (16). For example, estimates of important prereading skills such as phonological awareness and phonological decoding have shown a relatively high level of heritability in recent studies (25,26) yet there have been many interventions that have been developed that successfully improve these skills (27,28). Estimates of heritability and environmental influence are dependent upon what is happening at that particular point in time for a specific population. If there is little relevant environmental variation influencing a given behavior (e.g., when the sample is drawn from a single city or province and the education for reading is highly similar for members of the sample), then an increasing portion of the variability will be attributed to genetic factors.

Conversely, if a sample is highly variable in relevant environmental factors (e.g., when the sample is drawn from multiple cities or locales that differ widely in the quality and/or type of education for reading), then the proportion of variability accounted for by genetic factors will be lower.

## Behavioral Genetic Studies of Reading and Dyslexia

Past behavioral genetic studies investigating reading and reading-related skills have shown that both reading and dyslexia are highly heritable. Twin studies have demonstrated high heritabilities of reading ability across developmental stages. A recent international study of preschoolers has shown that genetic influences on prereading skills such as phonological awareness are heritable ( $h^2 = .61$ ) (29). Follow up of the same children in kindergarten revealed that phonological awareness continued to show substantial genetic influence ( $h^2 = .63$ ) and further that early reading is highly heritable ( $h^2 = .70$ ). As these children continued on to second grade, word reading ( $h^2 = .84$ ) and phonological decoding ( $h^2 = .74$ ) were found to be highly heritable along with reading comprehension ( $h^2 = .67$ ) (30). Similarly, high heritabilities were found in a study of 7-year old twins in the UK on word reading ( $h^2 = .63-.74$ ) and teacher-assessed literacy ( $h^2 = .65-.66$ ) (31). Additionally, relatively comparable genetic estimates were reported for a similarly aged US sample for reading comprehension ( $h^2 = .76$ ) but a slightly lower estimate for phonological decoding ( $h^2 = .51$ ) and much lower estimate for phonological awareness ( $h^2 = .14$ ) (32). Reading continues to have substantial genetic influence in later school years as an older US sample (mean age 10.3) demonstrated on word reading ( $h^2 = .76$ ) and reading comprehension ( $h^2 = .67$ ) and again at a five-year follow-up on the same measures (word reading,  $h^2 = .68$ ; reading comprehension  $h^2 = .60$ ) (21). Lower estimates of heritability were found for a 13-year old UK sample on word reading ( $h^2 = .19$ ) and reading comprehension ( $h^2 = .44$ ) (32).

Estimates of genetic influences on dyslexia are high as well. Seven-year olds in the lowest 10% on a reading composite of word recognition and phonological decoding showed group heritabilities between .50-.67 (34) and heritabilities of liability on the lowest 13.4% on the same measures between .42-.56 (31). Group heritability of dyslexia for an older group of dyslexic children (mean age 12, ranging 8-20) ascertained for reading difficulties was comparable in magnitude on a reading composite ( $h^2_g = .51-.65$ ) (35,36). Much lower group heritabilities were reported for a different adolescent sample on word recognition ( $h^2_g = .03$ ), phonological coding ( $h^2_g = .41$ ), and a reading composite ( $h^2_g = .21$ ) (37).

As stated previously, heritability and environmental estimates are sample estimates of population parameters that are designed to draw inferences about the population from which the sample is drawn. One limitation of the existing behavioral genetic studies of reading and dyslexia is that the samples used in these studies are primarily from middle- and upper-SES families and are predominantly white (23,34,32,26). It is currently unknown whether the estimates of genetic influence on reading and dyslexia will hold in an ethnically and socioeconomically diverse sample. To illustrate the utility of twin methodology for understanding the etiology of dyslexia, data from first grade twins participating in the FTP-R study are presented.

## METHOD

### Participants

**Florida Twin Project on Reading**—The Florida Twin Project on Reading (FTP-R) is a recently established longitudinal twin study that is part of the Learning Disabilities Center at Florida State University and the Florida Center for Reading Research. A strength of the FTP-R sample is that it is ethnically and socioeconomically diverse. As such, it has the potential to

test and extend many behavioral genetic findings of academic achievement such as dyslexia that have previously only been tested on largely white and middle-class twins. The diversity in the FTP-R sample is a result of the means by which twins were recruited.

All FTP-R study members were initially identified as potential twin pairs/multiples based on a match of children with the same last name, birth date, and school from Florida's Progress Monitoring and Reporting Network (PMRN). Recruitment was carried out through a combination of direct mailings to parents and/or mailings sent to schools to be carried home by a randomly selected member of the potential twin pair. Along with a cover letter explaining the study, the parent mailing included a questionnaire consisting of five questions regarding the similarity of the twins that has been used in previous twin studies and shown to determine zygosity with an accuracy rate of over 95% when compared to DNA tests (38). Recruitment is ongoing and at the time of this report 5,716 twins/multiples have been identified as potential twins/multiples, of which 2,466 (43%) responded. Nearly all (95%) those who responded (4,345; 1,425 MZ and 2,920 DZ) have agreed to be included in the study.

Data collection occurs via the PMRN, a database containing both longitudinal progress monitoring and achievement data for reading collected by trained testers on nearly two million students attending schools in both rural and urban areas throughout the state of Florida in kindergarten through 12th grade. The PMRN was initially designed for use by schools participating in Reading First, a national program aimed at raising the reading achievement of at-risk and low-achieving students. However, the use of the PMRN was subsequently adopted by many non-Reading First schools and this has resulted in a diverse group of participating schools ranging from extremely low performing schools in economically disadvantaged areas of Florida to some of the best public schools in Florida in affluent areas of the state.

Consequently, the FTP-R study contains a racially/ethnically and socioeconomically diverse twin sample that is reflective of the larger PMRN sample. Less than half of the sample is self-identified by parent report as White (47.2%), approximately one quarter Hispanic (25.6%), and one fifth African American (20.1%), with the remaining (7.1%) self-identified as Asian, American Indian, or Mixed race. Socioeconomic status can be measured by qualification rates of free- or reduced-price lunch through the schools. Each year of data collection, at least 53% of twins in the sample qualified for the free- or reduced-price lunch program. The FTP-R may also represent a more diverse sample of twins than other studies since participation in the FTP-R required minimal investment of time or energy on the behalf of the twin families to be included. Since all achievement data were already collected as part of normal school attendance, there was no need for parents to have transportation or make time for participation in testing, a potential burden to many low-SES families which might prevent volunteering in studies.

Participants examined in this report were 1,024 (382 MZ and 642 DZ) students for whom SAT-10 Reading scores were available for both twins completing first grade at the same time. The mean age of the sample was 7.17 (SD = .50). Approximately half of the students were female (50.4%). Self-identified ethnicity representation was 36.2% African-American, 34.1% White, 22.0% Hispanic, 5.6% Mixed ethnicities, 0.6% Asian. Nearly three quarters (73.6%) of the children qualified for free or reduced-priced lunch during the year of data collection. Data were collected during the spring of first grade in 2003–2004 through 2008–2009 school years. For cases of grade retention, data were used the first year of first grade and only when first grade was completed at the same time.

## Measure

**Stanford Achievement Test-Reading subtest (10<sup>th</sup> edition; SAT-10)**—SAT-10 (39) is a standardized and nationally norm-referenced measure of reading comprehension.

Classroom teachers administered this untimed test in a group format. Students answered multiple choice items that assess initial understanding, interpretation, critical analysis, and awareness and usage of reading strategies after reading literary, informational, and functional text passages. The reported reliability coefficient for SAT-10 reading comprehension is .91. Considerable evidence of content, criterion-related, and construct validity is available for the test (39).

## RESULTS

Intraclass correlations on SAT-10 Reading were .79 for MZ and .53 for DZ twins. The heritability for SAT-10 Reading in this sample was .53 indicating that 53% of the variability in first grade reading was due to genetic factors. Shared environmental influence accounted for 25% of differences in SAT-10 Reading scores, while non-shared environment explained 21% of the variability in these scores.

Although understanding the relative proportion of variability due to genetics across the range of reading ability is interesting and informative, it does not necessarily generalize to the dyslexia population, a focus of this paper. Group heritability for dyslexia addresses that question. In order to estimate the role of genetics in dyslexia, dyslexic students were identified from the 512 twin pairs. Though there has been much debate about the best classification definition of dyslexia which has historically been defined a variety of ways (e.g., 40), this topic is beyond the scope of this paper and a straightforward cut-point definition of dyslexia will be used here. Dyslexia was defined as students performing at the 15<sup>th</sup> percentile or below on SAT-10 Reading, a cut-point within the range of other behavioral genetic studies (19) that provided an adequate sample of children who are clearly experiencing reading difficulties (see Table 1). Given this dyslexia definition, 159 of 1,024 individual twins (72 MZ and 87 DZ) were identified as dyslexic. Probandwise concordance rates, the most appropriate concordance rates for generalizing findings to the greater population (41), were .58 for MZ twins and .32 for DZ twins. The greater probandwise concordance rates of MZ versus DZ twins suggests genetic influences on dyslexia.

While the individual differences heritability demonstrates genetic influences on reading and probandwise concordance rates suggest genetic influences on dyslexia, modeling the two simultaneously via DF extremes analysis tests whether a particular set of genes is affecting reading ability while a different set of genes is impacting dyslexia. Though DF extremes analysis was designed to assess the heritable influences that explain mean differences of those with dyslexia and the unselected population, other researchers have used the technique as a means of assessing whether the same genetic factors are responsible for variability in continuously measured ability and dichotomously assessed dyslexia since both are used in estimating group heritability (19). In DF analysis cotwins' scores are predicted from proband twins' scores and the coefficient of relationship (1.0 for MZ and 0.5 for DZ). The extent to which DZ cotwins regress to the unselected population mean more so than MZ cotwins provides an index of heritable influences on proband (dyslexic) group membership, or group heritability. Group heritability was estimated at .59 indicating that genetic factors account for 59% of mean differences between those with dyslexia and the unselected population (i.e., normal variation). Group shared environment was .19 and group non-shared environment was .23.

Both heritability estimates were fairly comparable and provide unique information about the extent to which genetic influences are responsible for variability in both reading ability and dyslexia. Overall, these analyses suggest that genetic factors play a substantial role in early reading (see Figure 1). These data further suggest that at least to some extent that there is

overlap of genetic factors across the range of ability, including the low end of the continuum. Importantly, reading is also influenced to a large extent by environmental factors.

## DISCUSSION

The purpose of this article is to provide an overview and illustration of twin methodology as a tool in uncovering the etiological architecture of dyslexia. The current study investigated the etiology of reading ability and dyslexia in a diverse sample of first grade twins and found substantial estimates of heritability for both reading and dyslexia. Past studies of reading ability using similarly aged twin samples showed higher individual differences heritabilities ( $h^2 = .63-.84$ ) (30,31,32). The lower heritabilities found in the present study could be due to the increased variability in the environments of the current sample as compared to other twin samples. Another plausible explanation is the differences in measures used in the current study versus those of the other research groups. Either way, the discrepancy in results highlights the fact that estimates of heritability are sample statistics and will vary based on the level of environmental variability in the population under study.

Previous studies of reading ability and dyslexia investigating individual differences and group heritabilities on the same sample have found patterns of results indicating genetic overlap across the range of ability (34,19). To test the generalizability of this finding, the current study analyzed heritability of reading and dyslexia in a diverse twin sample. While a different observed pattern of results may have indicated that different genetic factors affect reading and dyslexia, the current study also found a pattern of results demonstrating evidence of genetic overlap of reading and dyslexia. However, it is interesting to note the lower magnitudes of the heritability estimates in the current study as compared to those of the previous studies. Lower heritability for dyslexia may be due to differences in severity of reading difficulties due to both overall study sample abilities and choices of cut-points identifying dyslexia. Alternatively, it may be the case that more variable environments in the FTP-R sample resulted in relatively higher proportions of variability due to environmental factors as discussed for reading ability above. Further study of the FTP-R sample will investigate whether this pattern of results holds across other measures and during different developmental stages.

A limitation of this twin study and, to our knowledge, all of the cited past studies on dyslexia and reading ability in this report relied on assumptions of the twin study method. As such, the estimates of genetic and environmental influences were calculated based on the assumption that MZ twins are genetic controls in order to estimate heritability and environmental influences. However, recent studies of epigenetic effects and other molecular mechanisms are challenging this convention by showing evidence that MZ twins can show phenotypic differences that are not accounted for by chromosomal genotype or environment (e.g., 42,43, 44,45). While these new findings challenge some core assumptions of the twin study method, even critics acknowledge that twin studies have allowed for the new discoveries and continue to be worthwhile to confirm the presence of genetic variation in guiding future research (42). There are a variety of methodological techniques to evaluate twin data and twin studies such as the current one are the starting point and a necessary foundation from which to build and inform more sophisticated and in-depth analyses such as gene x environment interactions to identify causal environmental effects and identification of specific genes (46,42).

### Future Directions in Behavioral Genetic Studies of Dyslexia

Learning disabilities in general, and dyslexia in particular, are a prominent focus of educators, physicians, and researchers alike. The use of genetically informative research designs such as twin studies provides a means of defining the underlying etiological architecture of dyslexia. Over the past decades behavioral genetic research has added much to our understanding of genetic influences on reading and dyslexia, yet there are still many questions left unanswered.



While past studies have contributed tremendously to the knowledge base, as in all research, generalization of findings are limited by the samples studied. There are a few notable gaps in the extant research that we feel are crucial next steps in understanding the etiology of reading and dyslexia: representativeness of samples, longitudinal studies, and gene x environment interactions.

We know of no twin studies of academic achievement data that include substantial representation of ethnic minorities or low socioeconomic status families. Yet, there is evidence to suggest that SES affects heritability estimates via gene x environment interactions on intelligence tests and standardized academic achievement measures (47,48). The FTP-R sample is ethnically and socioeconomically diverse as described earlier and emerging reports using the sample show promise in filling this gap and will add greatly to the existing literature.

Longitudinal studies offer much more explanatory power regarding the relative influences of genetic factors over time. In addition to demonstrating the relative proportions of genetic and environmental influences, the stability of particular genetic factors as well as whether new genetic influences come online can be tested. Multivariate studies can disentangle whether the same genetic factors that impacted particular prereading skills later impact skills such as reading comprehension. Studies of this design have the potential to identify optimal windows for intervention. The use of twin samples allows measurement of genetic influences at various developmental stages, of especial interest are the phases of reading acquisition and the transitional time between “learning to read” and “reading to learn.” Collapsing across age groups and variables of interest may be obscuring important distinctions. Several ongoing longitudinal twin studies are beginning to shed light on these sorts of questions including the ILTS mentioned previously, Western Reserve Reading Project (WRRP; e.g., 32), and Twins Early Development Study (TEDS; e.g., 34,49). The FTP-R study provides a critical complement to these other studies through the examination of a large and diverse twin sample that covers both the “learning to read” period (which is less well covered by other twin studies) and the “reading to learn” period. Longitudinal twin studies will provide a blueprint of the etiological architecture of reading ability and dyslexia and will reveal the extent to which genes and environment interact to produce the wide variability in children’s reading achievement. Twin studies will continue to provide perspective for discoveries of specific genes involved in dyslexia by quantifying the amount of variance waiting to be accounted for by genes while simultaneously providing an impetus to continue working on efforts for environmental intervention.

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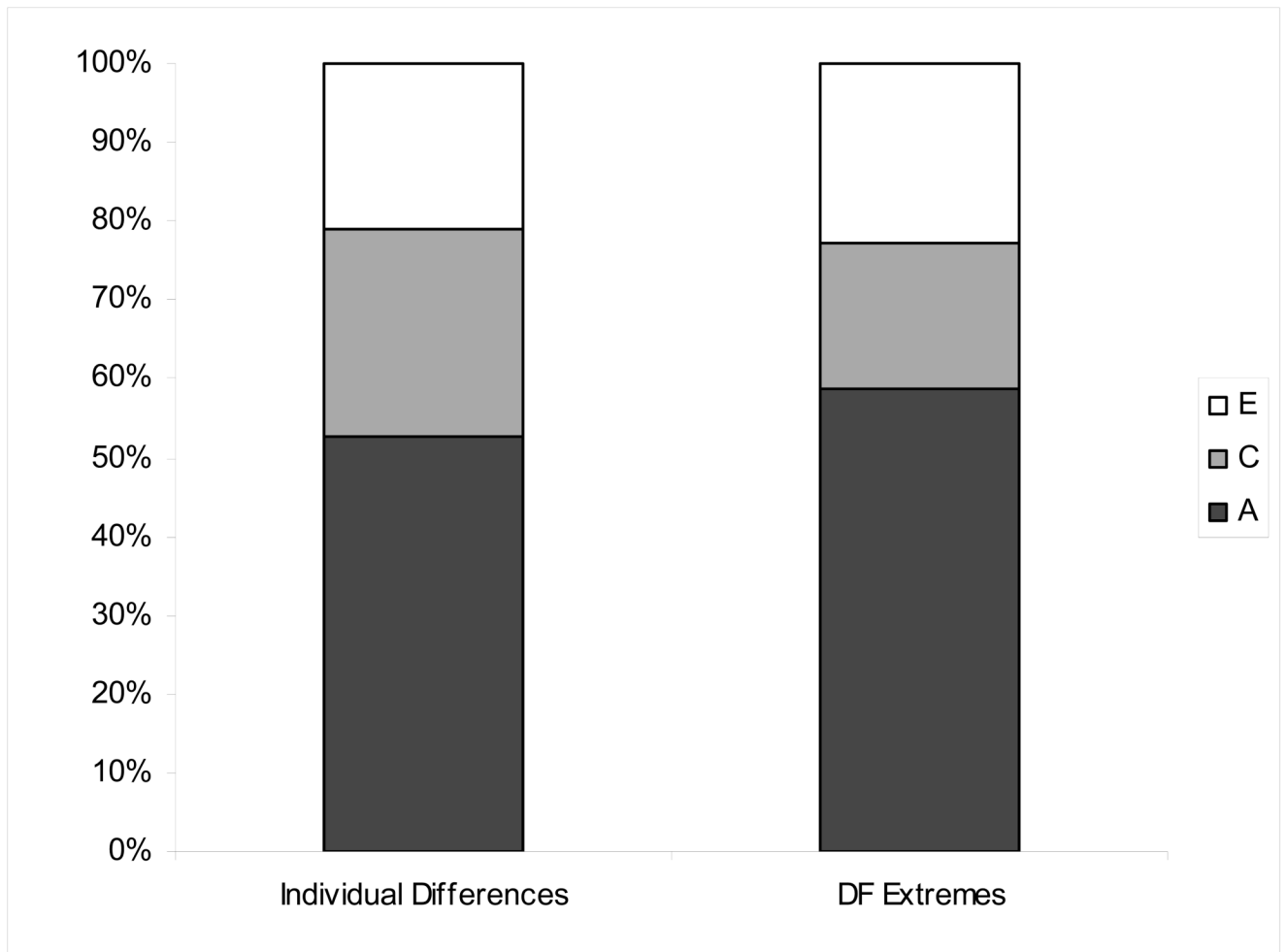
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## REFERENCES

1. Vellutino FR, Fletcher JM, Snowling MJ, et al. Specific reading disability (dyslexia): What have we learned in the past four decades? *J Child Psychol Psychiatry* 2004;45:2–40. [PubMed: 14959801]
2. Lyon GR, Shaywitz SE, Shaywitz BA. A definition of dyslexia. *Ann Dyslexia* 2003;53:1–14.
3. Lyon, GR.; Fletcher, JM.; Barnes, M. Learning disabilities. ch 12. In: Mash, EJ.; Barkley, RA., editors. *Child psychopathology*. 2nd ed.. New York: Guilford; 2002. p. 520-588.
4. Pennington, BF.; Peterson, RL.; McGrath, LM. Dyslexia. ch 6. In: Pennington, BF., editor. *Diagnosing learning disorders: A neuropsychological framework*. 2nd ed.. New York: Guilford; 2009. p. 45-82.
5. Shaywitz SE, Escobar MD, Shaywitz BA, et al. Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. *N Engl J Med* 1992;326:145–150. [PubMed: 1727544]

6. Raskind MH, Goldberg RJ, Higgins EL, et al. Patterns of change and predictors of success in individuals with learning disabilities: Results from a twenty-year longitudinal study. *Learn Disabil Res Pract* 1999;14:35–49.
7. Levine P, Nourse SW. What follow-up studies say about postschool life for young men and women with learning disabilities: A critical look at the literature. *J Learn Disabil* 1998;31:212–233. [PubMed: 9599955]
8. Simos PG, Breir JI, Fletcher JM, et al. Brain activation profiles in dyslexic children during non-word reading: a magnetic source imaging study. *Neurosci Lett* 2000;290:61–65. [PubMed: 10925175]
9. Dehaene S, Naccache L, Cohen L, et al. Cerebral mechanisms of word masking and unconscious repetition priming. *Nat Neurosci* 2001;4:752–758. [PubMed: 11426233]
10. Price, PJ.; McCrory, E. Functional brain imaging studies of skilled reading and developmental dyslexia. ch 25. In: Snowling, M.; Hulme, C., editors. *The science of reading: A handbook*. Oxford: Blackwell; 2005. p. 473-496.
11. Rayner, K.; Juhasz, BJ.; Pollatsek, A. Eye movements during reading. ch 5. In: Snowling, M.; Hulme, C., editors. *The science of reading: A handbook*. Oxford: Blackwell; 2005. p. 79-97.
12. Gabrielli JD. Dyslexia: A new synergy between education and cognitive neuroscience. *Science* 2009;325:280–283. [PubMed: 19608907]
13. Bowey, JA. Predicting individual differences in learning to read. ch 9. In: Snowling, M.; Hulme, C., editors. *The science of reading: A handbook*. Oxford: Blackwell; 2005. p. 155-172.
14. Gee, JP. Discourse and sociocultural studies in reading. ch 14. In: Kamil, ML.; Mosenthal, PB.; Pearson, PD., et al., editors. *Handbook of reading research*. Vol. Vol 3. Mahwah, NJ: Lawrence Erlbaum; 2000. p. 195-208.
15. Plomin, R.; DeFries, JC.; McLearn, GE., et al. *Behavioral genetics*. 4th ed.. New York: Worth; 2001.
16. Pennington, BF.; Olson, RK. Genetics of dyslexia. ch 24. In: Snowling, M.; Hulme, C., editors. *The science of reading: A handbook*. Oxford: Blackwell; 2005. p. 453-472.
17. DeFries JC, Fulker DW. Multiple regression analysis of twin data. *Behav Genet* 1985;15:467–473. [PubMed: 4074272]
18. DeFries JC, Fulker DW. Multiple regression analysis of twin data: Etiology of deviant scores versus individual differences. *Acta Genet Med Gemellol* 1988;37:205–216. [PubMed: 3254013]
19. Plomin R, Kovas Y. Generalist genes and learning disabilities. *Psychol Bull* 2005;131:592–617. [PubMed: 16060804]
20. Gayan J, Olson RK. Genetic and environmental influences on individual differences in printed word recognition. *J Exp Child Psychol* 2003;84:97–123. [PubMed: 12609495]
21. Betjemann RS, Willcutt EG, Olson RK, et al. Word reading and reading comprehension: Stability, overlap and independence. *Read Writ* 2008;21:539–558.
22. Astrom RL, Wadsworth SJ, DeFries JC. Etiology of the stability of reading difficulties: the longitudinal twin study of reading disabilities. *Twin Res Hum Genet* 2007;10:434–439. [PubMed: 17564501]
23. Byrne B, Samuelsson S, Wadsworth S, et al. Longitudinal twin study of early literacy development: preschool through Grade 1. *Read Writ* 2007;20:77–102.
24. Plomin R, Walker SO. Genetics and educational psychology. *British J Educ Psychol* 2003;73:3–14.
25. Petrill SA, Thompson L, DeThorne L, et al. Reading skills in early readers: Genetic and shared environmental influences. *J Learn Disabil* 2006;39:48–55. [PubMed: 16512082]
26. Gayan J, Olson RK. Genetic and environmental influences on orthographic and phonological skills in children with reading disabilities. *Dev Neuropsychol* 2001;20:483–507. [PubMed: 11892949]
27. Blachman B, Schatschneider C, Fletcher JM, et al. Effects of intensive reading remediation for second and third graders and a one year follow-up. *J Educ Psychol* 2004;96:444–461.
28. Mathes PG, Denton CA, Fletcher JM, et al. The effects of theoretically different instruction and student characteristics on the skills of struggling readers. *Read Res Q* 2005;40:148–182.
29. Byrne B, Olson RK, Samuelsson S, et al. Genetic and environmental influences on early literacy. *J Res Read* 2006;29:33–49.

30. Byrne B, Coventry WL, Olson RK, et al. Genetic and environmental influences on aspects of literacy and language in early childhood: Continuity and change from preschool to grade 2. *J Neurolinguistics* 2009;22:219–236. [PubMed: 20161176]
31. Harlaar N, Dale PS, Plomin R. Telephone testing and teacher assessment of reading skills in 7-year-olds: II. strong genetic overlap. *Read Writ* 2005;18:401–423.
32. Petrill SA, Deater-Deckard K, Thompson LA, et al. Longitudinal genetic analysis of early reading: The Western Reserve Reading Project. *Read Writ* 2007;20:127–146. [PubMed: 19829751]
33. Stevenson J, Graham P, Fredman G, et al. A twin study of genetic influences on reading and spelling ability and disability. *J Child Psychol Psychiatry* 1987;28:229–247. [PubMed: 3584294]
34. Harlaar N, Spinath FM, Dale PS, et al. Genetic influences on early word recognition abilities and disabilities: A study of 7-year-old twins. *J Child Psychol Psychiatry* 2005;46:373–384. [PubMed: 15819646]
35. Light JG, DeFries JC. Comorbidity of reading and mathematics disabilities: Genetic and environmental etiologies. *J Learn Disabil* 1995;28:96–106. [PubMed: 7884303]
36. Hawke JL, Wadsworth SJ, DeFries JC. Genetic influences on reading difficulties in boys and girls: the Colorado twin study. *Dyslexia* 2006;12:21–29. [PubMed: 16512171]
37. Stevenson J. Which aspects of processing text mediate genetic effects? *Read Writ* 1991;3:249–269.
38. Lykken DT, Bouchard TJ Jr, McGue M, et al. The Minnesota Twin Family Registry: some initial findings. *Acta Genet Med Gemellol* 1990;39:35–70. [PubMed: 2392892]
39. Brace, Harcourt. *Stanford Achievement Test: Technical Data Report*. 10th ed.. San Antonio, TX: Harcourt Brace; 2003.
40. Francis DJ, Fletcher JM, Shaywitz BA, et al. Defining learning and language disabilities: Conceptual and psychometric issues with the use of IQ tests. *Lang Speech Hear Serv Sch* 1996;27:132–143.
41. McGue M. When assessing twin concordance, use the probandwise not the pairwise rate. *Schizophr Bull* 1992;18:171–176. [PubMed: 1621065]
42. Townsend G, Hughes T, Luciano M, et al. Genetic and environmental influences on human dental variation: a critical evaluation of studies involving twins. *Arch Oral Biol* 2009;54S:S45–S51. [PubMed: 18715551]
43. Haque FN, Gottesman II, Wong AH. Not really identical: epigenetic differences in monozygotic twins and implications for twin studies in psychiatry. *Am J Med Genet C Semin Med Genet* 2009;151C:136–141. [PubMed: 19378334]
44. Lutz-Bonengel S, Schmidt U, Sanger T, et al. Analysis of mitochondrial length heteroplasmy in monozygous and non-monozygous siblings. *Int J Legal Med* 2008;122:315–321. [PubMed: 18478247]
45. Blakely EL, He L, Taylor RW, et al. Mitochondrial DNA deletion in "identical" twin brothers. *J Med Genet* 2004;41:e19. [PubMed: 14757869]
46. Johnson W, Turkheimer E, Gottesman I, et al. Beyond Heritability: Twin Studies in Behavioral Research. *Curr Dir Psychol Sci* 2009;18:217–220. [PubMed: 20625474]
47. Turkheimer E, Haley A, Waldron M, et al. Socioeconomic status modifies heritability of IQ in young children. *Psychol Sci* 2003;14:623–628. [PubMed: 14629696]
48. Harden KP, Turkheimer E, Loehlin JC. Genotype by environment interaction in adolescents' cognitive aptitude. *Behav Genet* 2007;37:273–283. [PubMed: 16977503]
49. Hayiou-Thomas ME. Genetic and environmental influences on early speech, language and literacy development. *J Commun Disord* 2008;41:397–408. [PubMed: 18538338]



**Figure 1.** Additive genetic (A), Shared environmental (C), and Non-shared environmental influences on variance in SAT-10 Reading for unselected and proband (dyslexic) First Graders.

**Table 1**

Means, intraclass correlations, and probandwise concordances of First Grade SAT-10 Reading by Zygosity and in the Total Sample.

	MZ			DZ			Total Sample		
	Mean (SD)	Twin Resemblance	N	Mean (SD)	Twin Resemblance	N	Mean (SD)	Twin Resemblance	N
Reading									
Unselected sample	556.33 (53.34)	0.79	382	561.19 (50.19)	0.53	642	559.38 (51.41)	0.63	1024
Dyslexia									
Proband	483.86 (16.55)	0.58	72	483.89 (13.57)	0.32	87	483.87 (14.94)	0.44	159
Proband Cotwin	500.99 (30.18)			523.14 (42.72)			513.11 (39.05)		

NOTE: MZ = monozygotic; DZ = dizygotic; N = number of individuals. For the unselected sample Twin Resemblance refers to intraclass correlation. For the dyslexia subsample Twin Resemblance refers to probandwise concordance rate.