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Explaining the Sex Difference in Dyslexia

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Abstract

Background—Males are diagnosed with dyslexia more frequently than females, even in epidemiological samples. This may be explained by greater variance in males' reading performance.

Methods—We expand on previous research by rigorously testing the variance difference theory, and testing for mediation of the sex difference by cognitive correlates. We developed an analytic framework that can be applied to group differences in any psychiatric disorder.

Results—Males' overrepresentation in the low performance tail of the reading distribution was accounted for by mean and variance differences across sex. There was no sex difference at the high performance tail. Processing speed and inhibitory control partially mediated the sex difference. Verbal reasoning emerged as a strength in males.

Conclusions—Our results complement a previous finding that processing speed partially mediates the sex difference in symptoms of attention deficit/hyperactivity disorder (ADHD), and helps explain the sex difference in both dyslexia and ADHD and their comorbidity.

Keywords

Reading; dyslexia; sex difference; processing speed; inhibition; verbal reasoning

Sex differences in prevalence are found in many psychiatric disorders, but none have been fully explained. If not due to an ascertainment or measurement artifact, a valid sex difference in prevalence can provide clues about the etiology or pathogenesis of a disorder. In a previous study, we developed a framework to understand the sex difference in attention deficit/hyperactivity disorder (ADHD; Arnett, Pennington, Willcutt, DeFries & Olson, 2015). The current study applies that same framework to dyslexia, which is frequently comorbid with ADHD. As shown in Figure 1, a key decision in this framework involves

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determining if the sex difference is valid, i.e. not merely an artifact of the sampling or measurement procedure.

If the sex difference is valid, a subsequent hypothesis is that cognitive correlates of the disorder mediate the sex difference, in that they represent risk factors for one sex and protective factors for the other. Although the implication is that the cognitive correlate relates to development of the disorder, we do not test that causal hypothesis in this paper. In order to be considered etiological, the cognitive correlate(s) would need to precede onset of the behavioral phenotype. Moreover, a complete explanation of a sex difference would include a clear understanding of the genetic etiology and related neurological differences that influence behavioral development. The goal of this paper was to take a step towards explaining the sex difference in dyslexia by testing for a cognitive mediator.

It is now clear that we can divide the over-representation of males with dyslexia into two parts: one invalid part explained by referral bias, and one potentially valid residual part found in epidemiological samples (Rutter et al., 2004; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990). The male : female sex ratio in referred samples ranges from about 3:1 to 5:1, whereas the sex ratio in epidemiological samples ranges from 1.5:1 to 3.3:1 (Rutter et al., 2004; Shaywitz et al., 1990), depending in part on the criteria for severity of the reading deficit and minimum-IQ for a diagnosis of dyslexia (Olson, 2002; Quinn & Wagner, 2015). The referral bias for dyslexia was initially detected in a family study by Hallgren (1950), who reported that while the sex ratio among referred dyslexic probands was around 3:1, the sex ratio among relatives of probands was much lower, around 1.5:1. The pattern has since been replicated numerous times (Wadsworth, DeFries, Stevenson, Gilger, & Pennington, 1992).

The sex difference in referred samples of individuals with dyslexia is due in part due to higher rates of externalizing behaviors among males with ADHD (Willcutt & Pennington, 2000), while the smaller sex difference in epidemiological samples remains unexplained. One possibility is test bias. Existing data support measurement equivalence for reading tests across sexes (e.g., McGrew & Woodcock, 2001). However, measurement bias is always conceivable in a study where the participants are not gleaned from the normative sample that was used to develop the measures.

Alternately, a sex difference in reading skill could be due to cultural differences. Previous research found compelling evidence for interactions between culture and sex on cognitive performance (DeFries, Corley, Johnson, Vandenberg & Wilson, 1981). Educational practices could relate to a sex difference in reading (Roivainen, 2011). However, females' advantage in reading performance is apparent at the start of formal schooling, as early as age five (Camarata & Woodcock, 2006) and seven years (Shaywitz et al., 1990; Rutter et al., 2004; Flannery, Liederman, Daly & Schultz, 2000). In addition, the sex difference in reading skill is found across countries with widely differing educational practices (Stoet & Geary, 2013, 2015) and across languages whose orthographies vary in transparency. Importantly, these international data, gathered by the OECD Program for International Student Assessment (PISA), were weighted to accurately reflect sex stratification in the respective nations, so the results are not due to sampling bias related to unequal educational opportunities for females

(OECD, 2014). Moreover, the cognitive predictors of reading skill have similar respective weights for males and females across languages (Hulme, 2015). Thus, neither educational practices nor alphabet differences are likely to explain the effect.

This early emergence and universality of the sex difference in dyslexia implicates underlying cognitive differences that develop before formal schooling begins. Some cognitive precursors to reading skill, such as oral language, emerge much earlier than reading instruction begins (Huttenlocher, Haight, Bryk, Seltzer, & Lyons, 1991). Processing speed, another cognitive predictor of reading skill, shows a female advantage by age five (Camarata & Woodcock, 2006) that is maintained until adulthood (Irwing, 2012; Majeres, 2007). In sum, there is impressive converging evidence that the residual sex difference in dyslexia and reading skill is valid, and relates to sex differences in cognitive correlates that emerge prior to schooling.

The overall goal of this paper is to test a cognitive correlate explanation of the valid sex difference in dyslexia found in epidemiological samples. To do this, we apply the method utilized by Arnett et al. (2015), which found that a sex difference in processing speed mediates the sex difference in ADHD. The framework presented in Figure 1 can be applied to group differences in any psychological trait, where groups could also be defined by ethnicity, SES, country or culture, for example. The pathway on the left side of the flow chart indicates an invalid sex difference (i.e., selection or measurement bias). The method used by Arnett et al. (2015) to explain a valid sex difference falls on the right side of the flow chart. There are three steps in the latter method: first, test whether the observed sex difference is due to a difference in means, variances, or both. Second, test whether cognitive correlates of the trait have a corresponding sex difference in terms of means, variances, or both. Finally, test whether the cognitive correlates mediate the observed sex difference in the trait.

A previous study by Hawke et al. (2009), which included a subset of the current study's sample, found greater variance in males' reading skills. Larger standard deviations in males' reading performance translate to a wider distribution, which should theoretically increase the male : female ratio in *both* tails of the overall group distribution. However, the PISA international sample has repeatedly found that males' greater variance only translates to a sex difference in the low performance tail (Stoet & Geary, 2013; Machin & Pekkarinen, 2008), and Hawke et al. (2009) did not test for a difference at the high extreme. Additionally, while Hawke et al. (2009) found comparable mean performance across sex, the PISA reports a mean difference in most countries, with lower average performance by males.

The current study expands on prior research by first confirming that the sex difference is valid and then testing for both a mean difference and a variance difference in a large sample of unrelated youth recruited as part of the Colorado Learning Disabilities Research Center (CLDRC). We hypothesize that, consistent with repeated results from the PISA, males will demonstrate greater variance and lower mean performance in reading than females. We propose that, following the flow chart in Figure 1 and the methods outlined by Arnett et al. (2015), the sex difference in low reading will disappear when variances are equated. In

contrast, we do not anticipate that equating the means will result in a reduced sex ratio at the low reading tail.

Next, we will test cognitive correlates of reading for mean and variance differences across sex. Those that reflect a sex difference in means or variances will be tested as mediators of the reading sex difference. We expect that phonemic awareness, processing speed, and verbal reasoning will each partially mediate the sex difference, and that the combination of all three mediators will reduce the direct path from sex to reading performance to nonsignificance.

Methods

Participants

Participants included a randomly selected twin or sibling from each family recruited as part of the CLDRC, which studies the etiology of learning disorders, including dyslexia (DeFries et al., 1997). The CLDRC recruits from 27 school districts within a 150-mile radius of the Denver/Boulder area. Case families, in which at least one twin was identified as having a positive history of attention and/or reading problems, were recruited at a rate of about 2:1 relative to control families, in which neither twin was symptomatic. Exclusion criteria were pervasive developmental disorder, history of serious traumatic brain injury, full scale IQ less than 70, and rare major gene disorders. The final sample constituted 2,401 unrelated youth, of whom 1,510 (62.9%) had been recruited as part of a case family (Online appendix, Figure S1).

Participant demographics are described in Table 1. The age of participants ranged from 7 to 24 years old (median = 11.17). Average full scale IQ was significantly higher than that of the normative sample (t[2398]=21.95, p<.001), but still well within the average range. As expected, the proportion of participants with a history of reading problems (37%) or attention problems (21%) as identified by initial teacher screenings was higher than the current U.S. prevalences of diagnosed dyslexia (7–10%) or ADHD (6–7%) (Peterson & Pennington, 2012; Willcutt et al., 2012). Relative to controls, participants with histories of reading problems in school were slightly younger (11.41 versus 11.66, p=.038) and had lower full scale IQs (97.68 versus 110.80, p<.001).

Procedure and Ethical Considerations

Cognitive testing took place during two six-hour days of testing typically scheduled within one month of each other. Parents and youth who were 18 years or older provided consent prior to testing, and minor youth provided written assent. Procedures were in compliance with the institutional review boards at the University of Denver and University of Colorado, Boulder. Neuropsychological testing was administered by trained examiners with at least a bachelor's degree and prior experience working with children.

Measures

Reading—Participants completed the Peabody Individual Achievement Test (PIAT) Reading Recognition and Spelling subtests (Dunn & Markwardt, 1970). There are no time

constraints on these subtests. Additionally, participants completed the Oral Nonword Reading task (Olson, Forsberg, Wise, & Rack, 1994), which requires participants to read 95 one- and two-syllable pseudo-words aloud as they are presented on a computer. Although both response latency and accuracy are measured in this task, only the accuracy score was included for the current study. Finally, participants completed a reading task designed specifically for the CLDRC, the Time Limited Word Recognition test (Olson et al., 1994). This task required participants to read single words as they were presented on a computer screen; correct responses were counted as long as they were initiated within two seconds. Thus, although time was constrained, accuracy, rather than speed of response, was the main measurement.

IQ—Participants completed one of the Wechsler intelligence test series, depending on the year of testing and the age of the participant. A breakdown of sample proportion and test version is described in the online Appendix, Notation S1.

Cognitive Correlates—Candidate cognitive correlates were selected for strong, previously established associations with reading achievement. Individual measures contributing to each cognitive correlate composite have been described in previous research (Bidwell et al., 2007; McGrath et al., 2011; Pennington et al., 2012) and are detailed in the online Appendix, Table S1. Correlations among the reading measures and cognitive composites are included in the online Appendix, Tables S2 and S3.

Data Cleaning

Cognitive measures, except Wechsler scaled scores, were regressed on age and the standardized residuals (mean = 0, standard deviation = 1) were used for the composites. Wechsler subtest scaled scores were converted to z-scores by subtracting the test mean (10) and dividing by the test standard deviation (3). All scores were Winsorized to within three standard deviations of the sample means. Skew and kurtosis absolute values were acceptable (skew < 1.3; kurtosis < 3.0) except for the Gordon Diagnostic Scale Vigilance Commission Errors, which was subsequently log transformed to achieve a normal distribution. Given that normalization of the data could potentially influence our primary hypothesis, we tested our analyses on three alternative reading composites. The results were largely consistent with our primary approach, and are described in the online appendix: Notation S2, Figures S2–S3, and Table S4.

Results

Sex Difference in Reading

The first step was to establish that a sex difference reading did indeed exist in the current sample. As expected, males demonstrated lower reading performance than females: $t(2320) = -3.06$, $p=.002$, Cohen's $d = -0.13$, 95% CI $d = -0.05 – .21$ (equal variances not assumed). This difference was not found in Hawke et al. (2009), and may be due to our larger sample, or may relate to lack of scalar invariance in reading measurement (see below). Consistent with previous research, males also showed significantly greater variability in reading performance (SD = 0.97) relative to females (SD = 0.84): Levene's F(1, 2397) = 30.76, $p<$.

001. Figure 2 shows the distributions of reading performance by sex. Next, we tested for sex differences at the extreme tails by establishing performance cutoffs of −1.5 SD and +1.5 SD relative to the entire sample mean. As expected, males were over represented in the low performance tail (11.6% vs. 6.1% females; $\chi^2(1) = 22.17$, $p<.001$; OR = 2.01, 95% CI = 1.49 – 2.69), but at the high performance tail, the proportion of males (4.9%) and females (4.5%) did not differ: $\chi^2(1) = 0.17$, $p=.678$; OR = 1.08, 95% CI = 0.74 – 1.58. Results were similar using a two standard deviation cutoff.

Measurement Invariance

As illustrated in Figure 1, an early step in determining whether a sex difference is due to valid neuropsychological differences is to rule out the possibility that measurement structure varies across sexes. Using confirmatory factor analysis in Mplus 7.31, we first tested for configural variance by modeling males and females separately; the four reading measures demonstrated strong associations with the general factor in both groups (male β: 0.84 to 0.95; female 0.80 to 0.94). Metric invariance was determined by a nonsignificant χ^2 change when factor loadings were constrained to be equal across sexes ($\chi^2[3] = 7.15$, $p=.067$). Scalar invariance, which we tested by holding the factor loadings and indicator intercepts equal across groups, was not found, as indicated by a significant χ^2 difference between the configural and scalar models: $\chi^2(7) = 100.46$, $p<.001$. Removing one variable at a time from the factor did not change this result; thus, lack of scalar invariance could not be attributed to one particular reading measure. These results held true using variables that were not Winsor-transformed.

Given only partial invariance for the composite, we next examined the individual reading measures for variance and mean differences across sex. All measures showed greater variance for males. A mean difference favoring females was found for the word recognition and PIAT spelling subtests, but means were comparable for the PIAT reading recognition and the oral nonword subtests. Again, results were replicated using non-transformed variables.

Although the lack of scalar invariance is concerning from a statistical standpoint and from a clinical assessment perspective more broadly, it is not entirely surprising given evidence for a very early female advantage in reading-related skills. Scalar variance reflects a group difference in intercepts, despite equal slopes; thus the theoretical implication is that females are born with an advantage in skills that support reading. To our knowledge, scalar invariance for reading measures across sexes has not been tested in larger samples, such as PISA, although PISA did find scalar invariance across countries. Despite this limitation, the greater rate of males in the low performance tail of the reading distribution, and the greater variance in males' scores, are highly consistent findings in the literature. Moreover, Levene's test of equality of variances is not dependent on scalar invariance. Thus, we decided to move forward with our analyses, with a cautious approach toward interpreting the results of mean comparisons across sex.

Variance Difference Model

The variance difference model (VDM) attributes the sex difference to greater variance in males' reading performance. To test this model, we equalized the variance by dividing males' and females' reading scores by their respective standard deviations so that each group had a standard deviation of one. If the observed sex differences are due to a variance difference, then this transformation should eliminate them. After equating variances, the sex ratio in the low performance tail was reduced, but not eliminated completely (Table 2). At the high end of the reading distribution, females were overrepresented once variances were equated. With the performance cutoffs set at two standard deviations, the sex difference was eliminated at the low tail. As expected, females' average performance on the reading composite was significantly better than that of males even when variances were equated ($t[2397] = 3.091$, $p=.002$), which could be due to a difference in group intercepts. Thus, the VDM partially explained the sex difference in low reading performance.

Mean Difference Model

The mean difference model (MDM) postulates that the sex difference in reading ability is due to males' lower average reading performance. This shift in distribution is illustrated in Figure 3 and would explain the asymmetrical finding of a higher rate of males in the low but not high performance tail of the reading distribution. To test the MDM, we subtracted the mean values for each group so that males and females had equal means (zero). This transformation eliminated the issue of scalar invariance encountered above. Following the transformation, males were still overrepresented in the low performance tail and were now overrepresented at the high performance tail (Table 2). However, when a 2 SD cutoff was used, there was no sex difference at either tail.

Combined Model

Following Arnett et al. (2015), we next equated both means and variances across sex and examined whether this combined transformation fully accounted for the sex difference at the low performance tail. Males' and females' RD composite scores were transformed into z-scores, within sex, to establish means of 0 and standard deviations of 1 for each group. This transformation does not set up a mathematical tautology due to the fact that differences in skew or kurtosis across male and female distributions could still exist despite establishing comparable scaled scores (i.e. z scores). Following these transformations, the sex difference at the low performance tail was no longer significant at either cutoff (Table 2). At the high performance tail, a difference favoring females only emerged at the 2 SD cutoff.

In sum, the observed male : female ratio of 1.90 in the 1.5 SD low reading tail, (i.e., 11.6% of males and 6.1% females) was reduced to 1.30 (9.6% males, 7.4% females) when the variance was equalized, but only to 1.49 (10.4% males, 7.0% females) when the means were equalized. When both means and variances were equated, the difference was fully accounted for, with a resulting ratio of 1.05 (8.3% males, 7.9% females).

Mediation by Cognitive Correlates

To test the hypothesis that cognitive correlates mediate the sex difference in reading performance, we first examined the cognitive composites for mean and variance differences

across sex. Processing speed (PS) was the only composite that demonstrated the expected sex difference in both means and variances, with males performing lower on average (t[2320] = 3.06, p=.002) and showing greater variance than females (F[1, 2397] = 30.76, p<.001). Females also outperformed males on the inhibition composite (INH): t(1121) = 2.65, p=.008, but variances were comparable. In contrast, males outperformed females on verbal reasoning (VR): t(2397) = −3.62, p<.001, with comparable variances. There was no sex difference on phonemic awareness or working memory (Table S1).

Next, we used the "MODEL INDIRECT" command, with default maximum likelihood estimation in MPlus 7.3, to test for mediation of the association between sex and reading by PS, INH and VR. Consistent with our hypothesis, inclusion of the cognitive mediators reduced the direct effect of sex on reading to non-significance (standardized β = .03, p=.078), whereas the indirect effects via the cognitive correlates were significant (PS β = .06, p<.001; INH β = .01, p=.013; VR β = −.03, p<.001; total indirect β = .04 p=.005; Figure 4). Thus, it appears that males' overall lower reading performance relates to a combination of cognitive risk factors, i.e. PS and INH, and a cognitive protective factor, VR.

Discussion

This study tested whether the valid sex difference in dyslexia found in population-based samples was due to sex differences in the mean, variance, or both; and whether the sex difference was mediated by cognitive correlates of reading skill. Consistent with Hawke et al. (2009) and PISA (Stoet & Geary, 2015), we found that sex differences in the variance of reading skill explained more of the sex difference at the lower performance tail of the distribution than did the mean difference. However, the sex difference at the low performance tail was only reduced to nonsignificance when both the means and the variances were equated. We also found that PS, INH and VR mediated this sex difference. In contrast, PA did not mediate the sex difference, despite the fact that PA is a strong predictor of later reading skill in many studies. Our results imply that the net sex difference may be explained by both risk and protective factors in cognitive correlates. This amounts to a complex interplay of average sex differences in cognitive functioning, which undoubtedly reflects even more heterogeneity at the individual level.

Our results for dyslexia are similar to a previous paper on the valid sex difference in ADHD (Arnett et al., 2015), in that PS partially mediated the sex difference in both disorders. We have previously demonstrated that PS mediates the comorbidity between dyslexia and ADHD both phenotypically (McGrath et al., 2011) and genetically (Willcutt et al., 2010). This interesting convergence of results across these four studies indicates that PS plays an important role in the development of both disorders. Further support for this conclusion are findings that a speeded naming measure (rapid automatized naming) shows bidirectional, longitudinal influences with both inattention (Arnett et al., 2012) and reading (Peterson, Arnett, Pennington, Byrne, Samuelsson & Olson, In submission) during early childhood.

The effect size for the average sex difference in reading performance was small (d = 0.13). Without establishing scalar invariance in our reading measures, we cannot be certain about the meaning of this difference; it may indicate bias in measurement that favors females'

particular reading-related skills. It may also reflect a female advantage in reading that is present from birth or, at least, from the start of formal reading instruction. We had a very large sample size, whereas a previous study using similar methods but a smaller sample (Hawke et al., 2009) found only a trend. Yet, a recent report by the Brown Center on Educational Policy at Brookings (Loveless, 2015) supports our findings: across two U.S. and two international samples of youth, males consistently had lower reading performance on standardized measures. Moreover, males showed a pattern of larger variance in reading performance than females in most of these samples, with male : female SD ratios ranging from 1.00 – 1.11. This ratio is slightly smaller than that of the current study (1.15), but the consistency of the finding across international samples and timepoints is compelling and argues for a neurobiological, rather than a cultural, etiology. Among these samples, both mean and variance differences increased with age (fourth grade: 5–11 standard points and : 0.13 t– 0.16 SD; 8th through 12th grades: 8 – 31 standard points and 0.19 – 0.34 SD). Thus, a future direction would be to test age as a covariate in these analyses and examine whether cognitive correlates demonstrate differential mediation effects over development.

A related question is whether our results extend to younger ages. There is substantial evidence that the sex difference in PS is present earlier than the typical age of dyslexia diagnosis (e.g., age 5; Camarata & Woodcock, 2006), and there exists a very early sex difference favoring females in language skill (Huttenlocher et al., 1991), which is consistent with our lack of scalar invariance. Our results are consistent with prior findings of a male advantage in VR in school age youth, despite a female advantage in writing (Camarata & Woodcock, 2006), verbal production and fluency (Geary, 1998; Halpern, 2000). Sex differences in verbal skills have been inconsistent in the literature and may depend on the particular skill measured as well as developmental stage. The lack of scalar invariance for the reading composite in our study underscores the possibility of developmental differences that could account for this variability in the extant literature. The clinical and educational implications of these findings are that research on diagnostic criteria and intervention for dyslexia should explicitly evaluate sex as a moderating factor.

PS mediated the associations between reading and ADHD severity in two prior studies (Shanahan et al.,2006; McGrath et al., 2011), suggesting that a substantial PS deficit may relate to comorbid dyslexia and ADHD. However, PS may play a lesser role in explaining the symptoms of youth who develop either ADHD or dyslexia in isolation. A future direction could be to test whether, among youth with comorbid dyslexia and ADHD, PS more fully mediates the sex difference in the ADHD symptoms than among youth with only ADHD.

The logical next step is to identify the shared neurobiological endophenotype that underlies the comorbidity and sex differences in dyslexia and ADHD. There is considerable cross-sectional evidence for a specific relation between myelination in the brain and PS across the lifespan (Raghubar, Barnes, & Hecht, 2010). Thus, a likely neurological candidate to explain sex differences in PS is the development of white matter tracts. Males are known to have greater volumes of white and grey matter relative to females; given that males demonstrate slower PS, this would suggest that greater volume alone is not a valid explanation for improved PS or related behavioral symptoms. Rather, this apparent contradiction may

suggest that smaller brains have more efficient, faster processing. Or, consistent with the results of the current paper, variability in white matter tract integrity may increase as brain volume increases. For example, Perrin and colleagues (2009) found that growth of white matter during adolescence was more rapid among males than females, and that pubertal stage predicted white matter growth. Although sex differences in variance of white matter volumes were not reported, it is plausible that faster growth could introduce more variability, possibly secondary to differences in pubertal development.

The current study did not address the etiology of the sex difference in dyslexia, but previous work in this sample indicates genetic covariation between reading skill and the cognitive correlates studied here (e.g., Willcutt et al., 2010; Christopher et al., 2016). Earlier sex differences may more likely be caused by neurological factors. For instance, a male advantage in mental rotation is apparent by five months (Moore & Johnson, 2008; Quinn & Liben, 2014) and has been linked to the organizing effect of prenatal testosterone on the developing male brain (Miller & Halpern, 2014; Hines, 2011). We do not know if the sex difference in PS is likewise due to prenatal hormonal influences; hence that is an important topic for future research.

Conclusions

The current study found that the higher prevalence of males with reading difficulties can be explained by a combination of males' slower and more variable PS and worse inhibitory control, although these are partly offset by males' better verbal reasoning. We did not find support for a female advantage in verbal skills, which had been previously hypothesized to explain the sex difference in reading. Our results are consistent with a prior study in which PS similarly mediated the sex difference in ADHD, which is frequently comorbid with dyslexia and also more common in males. We propose that PS is a cognitive correlate of reading that serves as a proxy for sex differences in brain development that support more efficient reading performance among females.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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KEY POINTS

- Males are diagnosed with dyslexia more often than females, even among epidemiological samples.
- We developed a strategy for determining the validity of sex differences in developmental disorders, which includes a test of cognitive correlates as mediators.
- The sex difference in reading ability is valid, and is due to males' lower mean and more variable performance relative to females'.
- The sex difference in reading is fully mediated by processing speed, inhibition and verbal reasoning.
- Processing speed, which is also slower and more variable in males, mediates the sex difference in both dyslexia and ADHD.

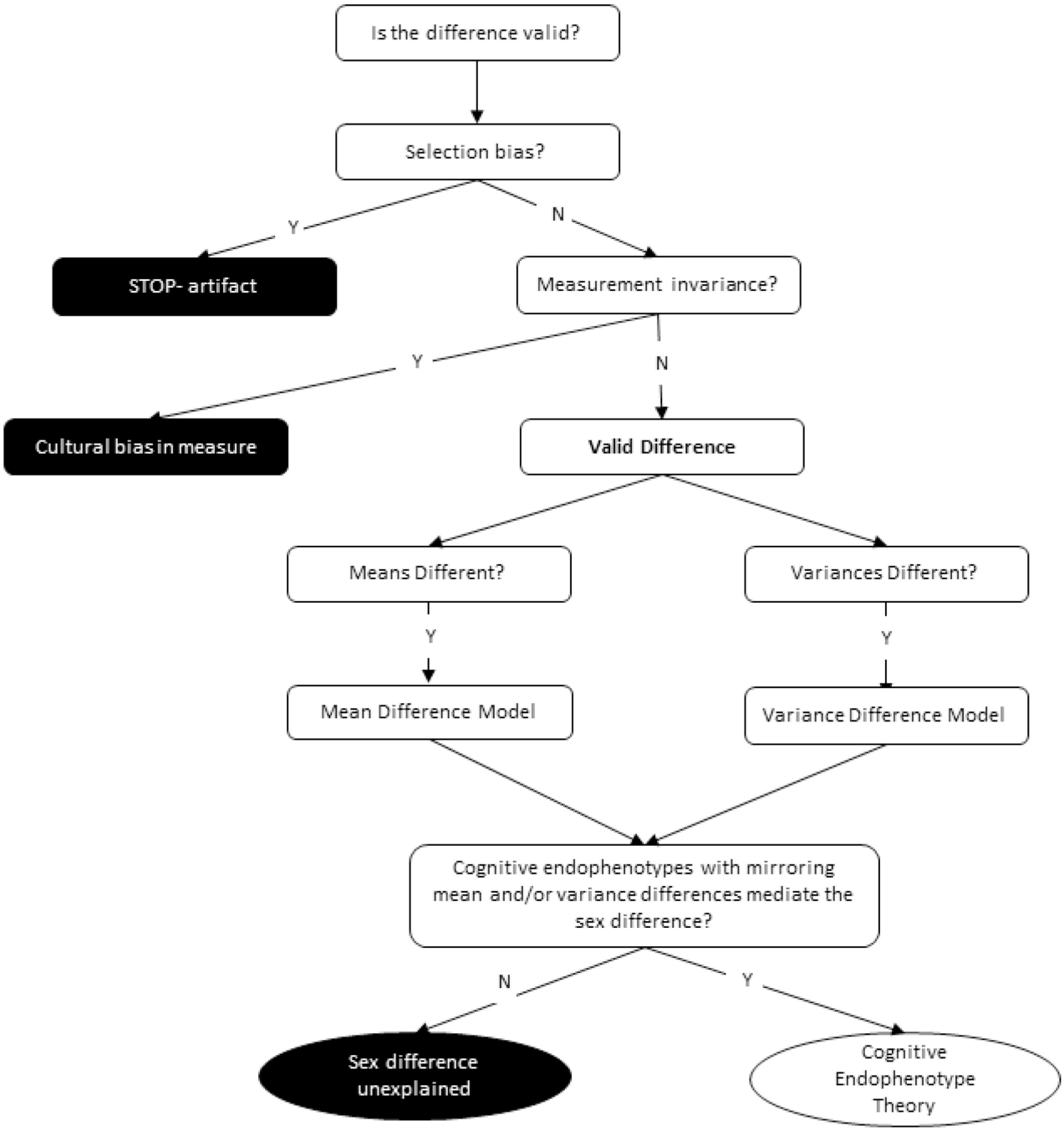

Figure 1.
Framework for Analyzing Group Differences

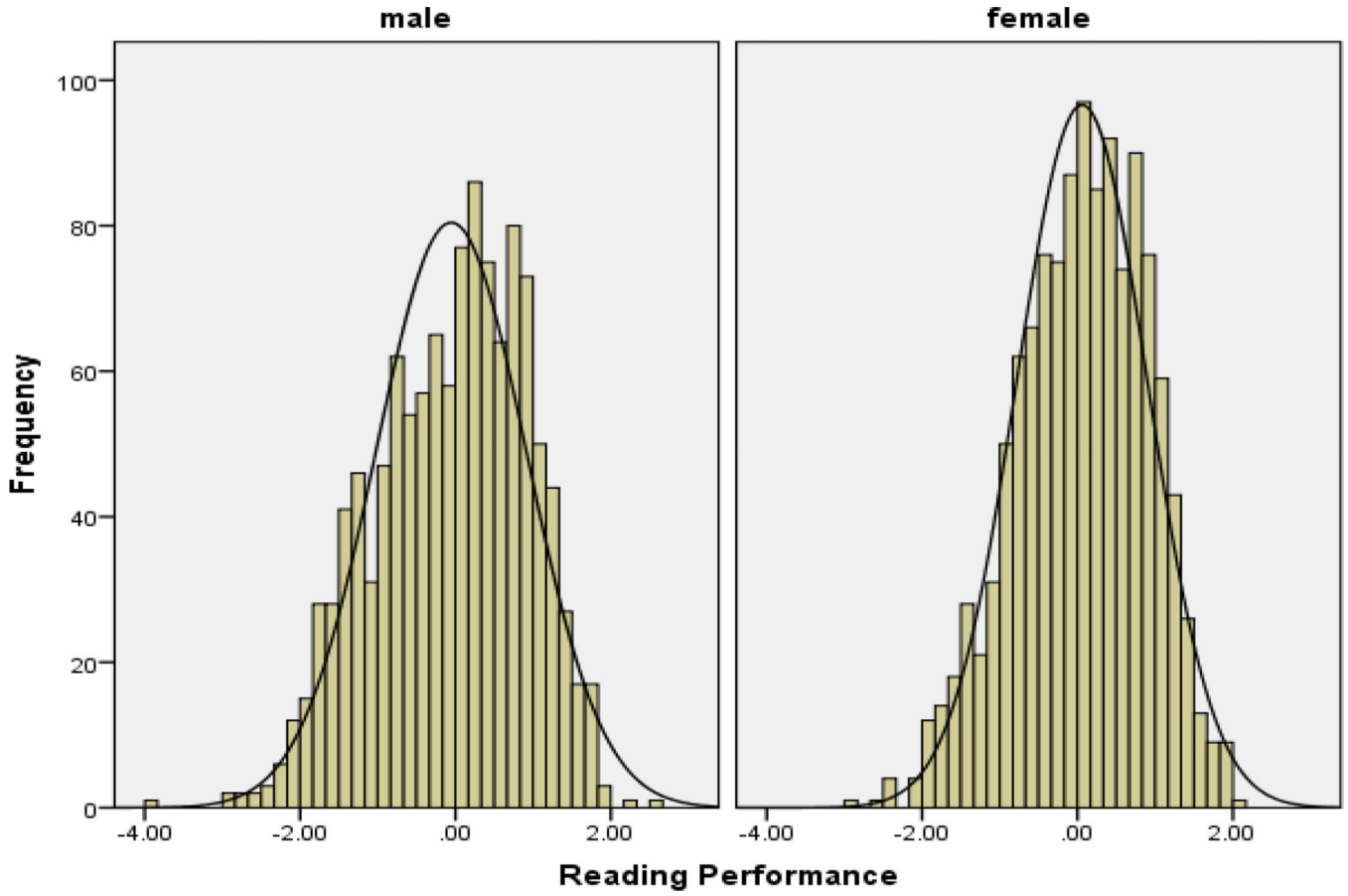

Figure 2.
Reading skill distributions by sex. X-axis values are mean z-scores for the reading composite.

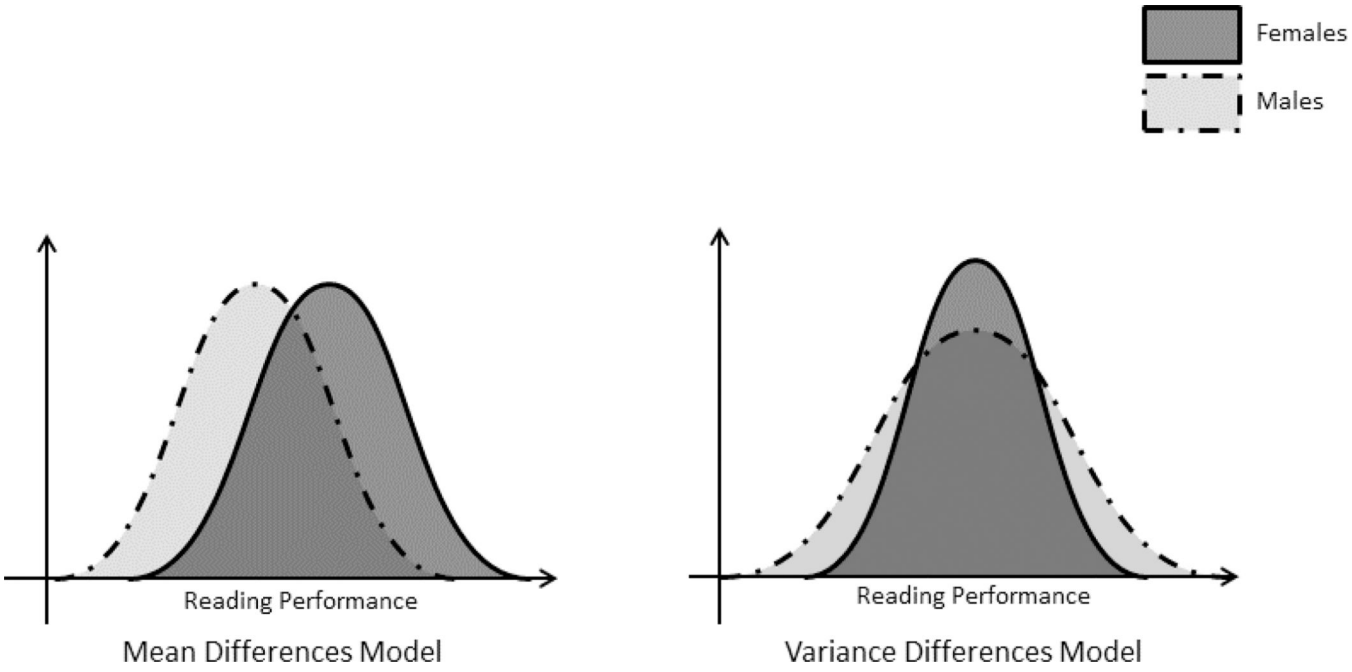

Figure 3.
Schematic of the Mean Difference and Variance Difference Models.

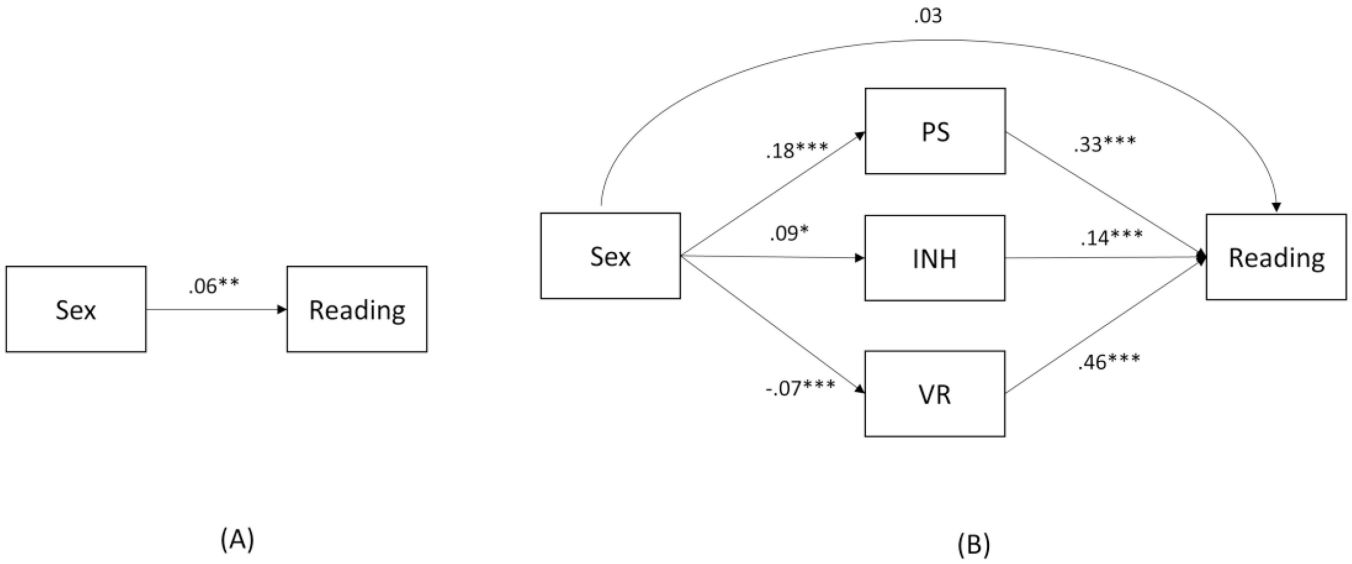

Figure 4.

Mediation of the Sex Difference in Reading by cognitive endophenotypes. PS = processing speed; INH=inhibition, VR=verbal reasoning. (A) Total effect of sex on reading. (B) Direct and indirect effects of sex on reading. Values are standardized beta coefficients. *p<.05, **p<.01, ***p<.001.

Table 1

Sample Demographics

N	2,399
Mean age in years (SD)	11.85 (2.87)
% female	51
% monozygous twins	40
% siblings of twins	9
% primary caregiver Caucasian	90
Median years of education (mother)	15
Median years of education (father)	14
Mean Full Scale IQ (SD)	105.97 (13.32)
% history of reading problems in school	37
% history of attention problems in school	21

Table 2

Sex Differences in Extreme Reading Tails Following Mean and Variance Transformations

	% of Males	% of Females	χ^2 (*1*)	*p*	Odds Ratio	95% CI
VDM: Variances Equated						
Low 1.5 SD	9.62	7.35	***3.97***	***.046***	***1.34***	***1.00 – 1.79***
Low 2.0 SD	2.89	2.70	0.09	.769	1.08	0.66 – 1.78
High 1.5 SD	3.49	5.64	***6.32***	***.012***	***0.61***	***0.41 – 0.89***
High 2.0 SD	0.17	1.47	***12.26***	***<.001***	***0.26***	***0.07 – 0.92***
MDM: Means Equated						
	% of Males	% of Females	χ^2 (*1*)	*p*	Odds Ratio	95% CI
Low 1.5 SD	10.38	7.03	***8.53***	***.003***	***1.53***	***1.15 – 2.05***
Low 2.0 SD	3.40	2.12	3.67	.055	1.62	0.98 – 2.68
High 1.5 SD	6.04	3.59	***7.87***	***.005***	***1.72***	***1.17 – 2.53***
High 2.0 SD	0.94	0.65	0.61	.435	1.44	0.58 – 3.58
Combined: Variances and Means Equated						
	% of Males	% of Females	χ^2 (*1*)	*p*	Odds Ratio	95% CI
Low 1.5 SD	8.34	7.92	0.14	.710	1.05	0.79 – 1.42
Low 2.0 SD	2.47	2.94	0.51	.476	0.84	0.51 – 1.37
High 1.5 SD	4.43	4.82	0.21	.645	0.91	0.62 – 1.34
High 2.0 SD	0.26	0.98	***5.07***	***.024***	***0.23***	***0.07 – 0.92***

Note. Values in bold italics indicate p<.05.