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On the aetiology of reading ability

A twin study on the differences in genetic architecture between stronger and weaker readers

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Date of Submission: April 18th, 2024

Word count: 5,999

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Abstract

Learning to read transforms lives, yet millions of people worldwide are unable to read and spell at a sufficient level. This weakens their educational and employment prospects and prevents them from fully participating in the complexities of modern society. In order to better understand the acquisition of reading, this thesis investigated whether the cognitive processes best known to associate with reading have the same genetic relations in novice and expert readers alike. Genetically sensitive data from the Brisbane Adolescent Twin Study was employed, and the sample consists of 2,190 same-sex and different-sex twins and one of their sibling from 915 families. Multivariate ACTE Cholesky decomposition model was used to partition the genetic and environmental covariance structures between five cognitive traits and nonword reading. As an exploratory method, a twin-specific environmental component was estimated. The study revealed several distinct differences in the genetic and environmental architectures between reading groups, with stronger readers' decoding being supported by additive genetic effects common to general cognitive ability, while weaker readers' decoding ability showed more commonalities with verbal working memory. The results are consistent with past research, replicating many differences previously found to differ between high and low-end readers. Methodological considerations and potential sources of bias in estimates will be discussed, and the implications of the study's findings will be contextualised against the wider literature on the genetics of reading ability.

1 Introduction

The ability to read—and read well—is a skill unlike anything else, facilitating one's capability to cope with the increasingly information-ridden world. Alas, children with reading difficulties often enter a downward spiral whereby their poor literacy leads to low educational attainment and poor employment prospects (Castles et al., 2018; Hulme & Snowling, 2016). In adulthood, the ability to play this language-game is so fundamental in everyday functioning that functionally illiterate adults have difficulties in basic tasks such as reading medicine labels, filling out applications, or understanding bank statements (World Literacy Foundation, 2023). The more severe consequences of poor literacy include the inability to understand government policies and election results. Overall, the World Literacy Foundation estimates that nearly 800 million people worldwide cannot read or write and that the global cost of illiteracy amounts to USD $1.19 trillion. This then begs the question *how* do those with poor literacy differ from the general population – what cognitive and environmental processes underlie their inability to participate in this process, and what could be done to remedy this?

1.1 On Writing

There are various ways to characterise reading: while Goodman (1967) has referred to it as a "psycholinguistic guessing game", Stephen King (2000, p. 103) says it is an exercise in telepathy, where readers and writers participate in a mentalist routine over time and distance. The innateness of this process obfuscates its complexity, and unpacking the above passage requires the coordinated effort of multiple mental faculties. In order for us to become fluent readers, it is essential we learn not just print words—what are the language's orthographic subunits and how can they be bound together to create lexical entries (Castles & Nation, 2022)—but also what are their grammatical functions (e.g., verb, noun), the phonological mappings between letters and sounds (Hulme & Snowling, 2014), and how do words' semantic content change in coordination with what precedes and follows them – a skill that is essential in acquiring sentence-level comprehension. The absence of causal explanations at a physiological level or when a failure to automate these processes cannot be explained by environmental factors might suggest the presence of a specific language impairment (SLI), which often co-occurs with dyslexia (Logan et al., 2011). It is worth noting that reading, like most cognitive traits, follows a normal distribution in a population. As such the criteria used for diagnosis are somewhat arbitrary, and usually those scoring more than 1.5 standard deviations (SD) below the mean in their reading ability qualify for a diagnosis, resulting in roughly 7%

of the population being identified as dyslexics (Hulme & Snowling, 2016). Moreover—and considering how much of behaviour genetic research has treated reading skill as a single phenotype—dyslexia is not homogeneous disorder, and it is vital to distinguish between different types of impairment, whereby different processes may be differentially impaired in different individuals (Castles & Coltheart, 1993; Castles et al., 2006).

1.2 Behaviour genetics of reading ability

Castles and colleagues (2006) argue that a key requirement in uncovering factors that influence normal acquisition of reading ability requires well-specified cognitive models explaining the sources of variance in reading and writing. Two prominent classes of models examining the causal pathways in word-reading exist today (Hulme & Snowling, 2014): connectionist models like the 'triangle' model of reading (Harm & Seidenberg, 2004) and dual-route models (Coltheart et al., 2001). Dual-route model (DRM) posits that skilled readers utilise at least two pathways for word-reading, which in DRM terms are referred to as lexical and sublexical routes. Reading aloud via the lexical route requires the retrieval of the phonological form appropriate to a particular word from the *orthographic lexicon* (Figure 1). As this lexicon contains only representations of previously encountered words, it is unable to process nonwords, which are instead processed through the sublexical route (*letter-sound rule application*). In their study, Castles and Coltheart (1993) assessed lexical and sublexical reading of 56 developmental dyslexics and 56 typically developing readers, revealing distinct varieties of developmental dyslexia, one showing impairment in lexical processing (*surface dyslexia*) and the other on sublexical processing (*phonological dyslexia*). This emphasises the aforementioned need to assess reading at the level of an individual, as one child's impairment may have been caused by factors different from another child (Castles, 2006; Ziegler et al., 2008).

While the reasons for reading impairment might vary from child to child, it has been shown that reading disabilities run in families, and siblings and parents of reading-disabled children perform worse on reading assessments compared to siblings and parents of control children (DeFries et al., 1987). Analyses from the Jyväskylä Longitudinal Study of Dyslexia (Puolakanaho, 2007) show that children with familial history of dyslexia have roughly a fourfold chance to acquire a reading disability compared to children without. Outwith familial incidence, the best early predictors of dyslexia in children include phonological awareness, letter knowledge, and rapid naming (Lohvansuu, 2021; Lyytinen et al., 2015; Puolakanaho et al., 2007). Still, this is not necessarily an indication of genetic influences, as parents and home literacy environment are highly

influential in children's early learning before children begin formal schooling (Niklas et al., 2016; Niklas & Schneider, 2013). In order to circumvent the coalescing effects of nature and nurture on traits like reading (dis)ability, genetically sensitive research designs have to be adopted (Bishop, 2015; Plomin & Daniels, 1987). The twin method partitions the relative contribution of these components by comparing genetically identical monozygotic (MZ) twins to dizygotic (DZ) twins who share on average 50% of their segregating genes. If a trait is influenced by genetic effects, its MZ within-pair correlation should be greater than DZ correlation.

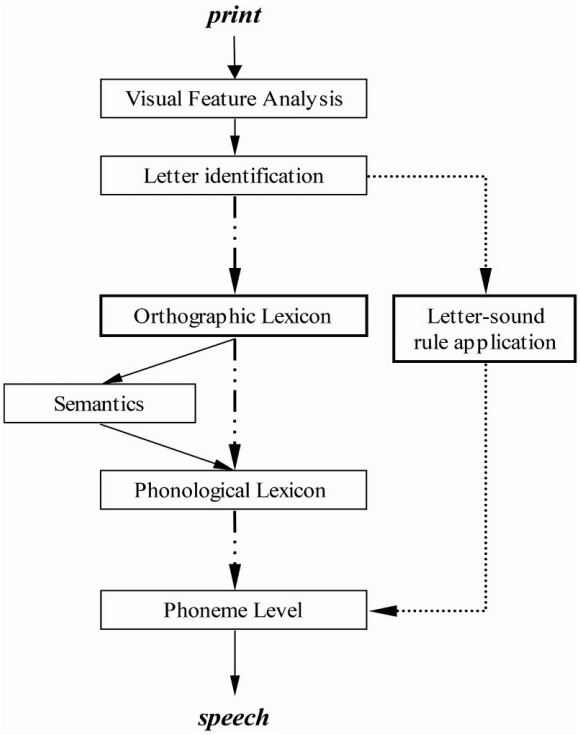

Figure 1: The dual-route model of reading. The series of processes constituting the lexical route are connected with dashed lines (–..–), while the nonlexical route components are connected with dotted lines (...). Inputs to and outputs from the system are shown in solid lines. Figure and caption from Castles and collagues (2006).

Castles and colleagues (1999) used a set of twin-pairs (272 MZ; 320 DZ) of whom one had reading disability and compared them to 423 control pairs (211 MZ; 212 DZ) to explore the genetic basis of processes influencing lexical and sublexical reading processes. The study provided evidence for the distinct genetic influences across subtypes, with genetic and environmental variance components for lexical ($h^2 = 0.31$, $c^2 = 0.63$, $e^2 = 0.06$) and sublexical reading ($h^2 = 0.67$, $c^2 = 0.27$, $e^2 = 0.06$) showing significant deviation from each other. However, this does not explain if these genetic effects over-

lap. Later, Bates and colleagues (2007) conducted a multivariate analysis using on a set of Australian twins, confirming a general additive genetic factor influencing both irregular and nonword reading (Figure 2).

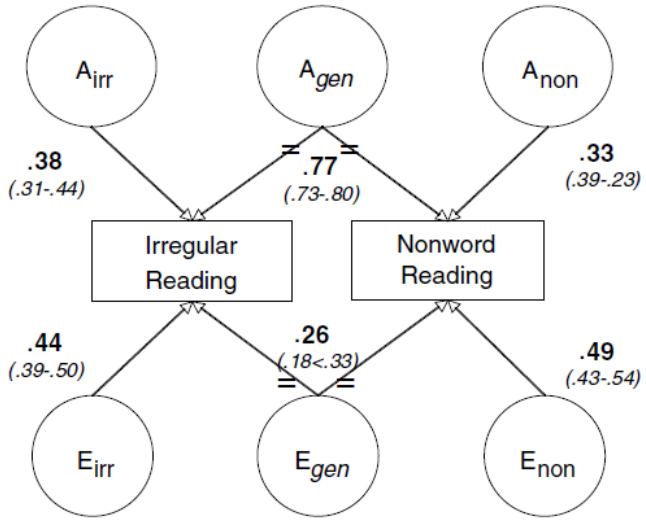

Figure 2: The model shows a general additive genetic factor as well as general unique environmental factor influencing both reading and nonword reading. Common environmental factor was dropped without a significant drop of fit. Figure from Bates and colleagues (2007).

Yet, while these types of studies highlight distinct genetic aetiologies for various subtypes of reading and spelling, they do not necessarily reveal details about the relative importance of different cognitive mechanisms at different skill levels or age groups. Common to practically all behaviour genetic models is the assumption that phenotypes' degree of heritability is constant across the distribution (Berentsen et al., 2020; Logan et al., 2012). Thus the h^2 estimation of variance explained due to genetic effects is assumed to be identical whether the phenotype is measured at the low end, middle, or upper end of the phenotype's distribution. As an example, performance on a nonword repetition task, which is used to assess phonological short-term memory, is a strong diagnostic predictor of language impairment, and higher heritability of reading problems has been observed in children who perform poorly on this task (Logan et al., 2012; Meir & Armon-Lotem, 2017). Similarly, while intelligence and phonological decoding have been shown to work mostly independent from one another (Scholz & Scheer, 2020), Alarcón and DeFries (1997) found that genetic and phenotypic variances and covariances between intelligence and reading ability were larger for twins with reading difficulties compared to a control group, with genetic correlation between reading performance and general cognitive ability for these groups being 0.52 and 0.81, respec-

tively, leading to the assumption that there lies a difference in the genetic covariation in these measures between high and low-end readers.

1.3 The present study

This thesis aims to understand whether the cognitive processes associated with reading have the same genetic relations in weaker and stronger readers. The cognitive processes consistently related to reading skill include phonological working memory and processing speed with some contribution from general cognitive ability. Multivariate models will be fit using genetically sensitive twin data, which allows the estimation of "the relative influences of nature and nurture on a particular trait, in a particular population at a specific time" (Haworth et al., 2008, p. 1006). Heritability estimates for the cognitive measures are expected to be greater in weaker readers. As an exploratory measure, non-twin siblings will be included in the analyses, which allows for the a twin-specific environmental component to be estimated. I hypothesise that this accounts for a small amount of the variance in all cognitive and reading measures, consequently reducing the effect of the common environmental component (Koeppen-Schomerus et al., 2012). By examining whether there exists differential patterns in the genetic architecture of reading ability across the distribution, the present study aims to increase understanding of reading acquisition from novice to expert (Castles et al., 2018).

2 Materials and methods

I conducted a secondary data analysis using data from the Brisbane Memory, Attention and Problem Solving (MAPS) study, the 16-year old wave of the Brisbane Adolescent Twin Study (Wright et al., 2001; Wright & Martin, 2004). The present study uses a sample of 2,190 same and different-sex twins and one of their sibling across 915 families. Five variables for various cognitive abilities hypothesised to differ between high and low-end readers were used. In order to differentiate between latent constructs and how they are coded in the dataset, a different typeset will be used (e.g., WM describes working memory as a latent factor while `WM` describes the variable). All analyses were conducted in R (Version 4.3.3 for Ubuntu, http://www.r-project.org). The scripts for this thesis can be found on GitHub (`https://github.com/mrazael/portfolio/`).

2.1 Ethics statement

The original study was carried out in accordance with the recommendations of The Australian Code for the Responsible Conduct of Research, with written informed consent from all subjects, and the protocol was approved by the QIMR Berghofer Human Research Ethics Committee. The present study has ethical approval from the PPLS Ethics Committee of the University of Edinburgh (No. 84-2324/3).

2.2 Linguistic measures

Reading measures were derived from an extended version of Castles and Coltheart test (1993) developed by Bates and colleagues (2004), and five reading measures were used: regular reading (`READREG`), irregular reading (`READIRR`), regular spelling (`SPELLREG`), irregular spelling (`SPELLIRR`), nonword reading (`READNW`). The test includes additional items that make it more difficult and hence more suitable for older participants. As the test was assessed over the phone, some twins had a large proportion of unrecorded answers. Scores for these twins were set to NA; for others, scores were calculated as a ratio of correctly answered items.

2.3 Cognitive measures

Nonword repetition was assessed using tasks developed by Dollaghan and Campbell (1998; `NWDC`) and Gathercole and Baddeley (1994; `NWGB`). **Performance IQ** (`PIQ`) was measured using the Multidimensional Aptitude Battery (MAB), a revised version of Wechsler Adult Intelligence Scale (WAIS-R) (Wechsler, 1981; Jackson, 1984), and it

comprises two subtests (Spatial, Object assembly). **Perceptual processing speed** tends to be impaired in weaker readers (Peter et al., 2018; Stenneken et al., 2011), and this was assessed using the Digit Symbol Substitution test from WAIS-R, which is a composite of graphomotor and perceptual speed, executive function, visual-scanning efficiency, memory, and associative learning (Joy et al., 2003). **Working memory** is a well-established predictor of individual variation in reading comprehension (Nouwens et al., 2017), and it was assessed using two measures from WAIS-III (Wechsler, 1997): Digit Span Backwards (DSB) and Letter Number Sequencing (`LNS`). Finally, Digit Span Forwards (`DSF`) was used to assess **verbal short-term memory** (VSTM), a known deficit in individuals with reading disabilities (Majerus & Cowan, 2016; Melby-Lervåg et al., 2012, van Leeuwen et al., 2009).

2.4 Data screening and sample representativeness

Variables were assessed for normality visually and using the Shapiro-Wilk test (Shapiro & Wilk, 1965); `LNS` and `DSB` showed moderate positive skew; reading data showed high negative skew, which is indicative of a ceiling effect in these measures; these were transformed with square root and inverse sine square functions, respectively. Plots for before and after transformations are found on Appendix (Figures 6–7). Variables were standardised, fixing means to zero and standard deviations to one. Three composite measures were created by averaging the participant scores: (1) reading ability (`RCOMP`) using `READREG`, `READIRR`, `SPELLREG`, and `SPELLIRR`, (2) phonological short-term memory (`PSTM`) using `NWDC` and `NWGB`, and (3) working memory (`WM`) using `DSB` and `LNS`; the last was done to be consistent with Hansell and colleagues' (2015) study using the Brisbane sample. Reliability was assessed using Cronbach's alpha, yielding a reliability coefficient of 0.87, 0.75, and 0.74, respectively, thus all were within acceptable lower-bound of 0.70 and upper-bound of 0.90 (Tavakol & Dennick, 2011). Variables were residualised for sex and age[1], and outliers beyond 4 SD from the mean were Winsorized to ±4 SD. Participants' reading score was used to label them as weaker ($0 <$ `RCOMP`) or stronger ($0 \geq$ `RCOMP`) readers. Subsamples were created from a maximal number of family members—for a maximum of two twins and one sibling—belonging to the same group. The sample did not differ from the full sample in any trait (Table 6, Appendix).

[1]Participants were assessed across three time points, and the average age was 17.99 (SD = 2.83).

2.5 Equality of Means, Variances, and Covariances

Regularity in sampling and measurement was established by testing whether variables could be equated for means, variances, and covariances according to twin order, across twins and siblings, and zygosity[2]. Models were fit on OpenMx 2.0 (Neale et al., 2016) using full-information maximum-likelihood (ML) procedures on raw data. In all models the SLSQP optimizer was used. Sequential models were nested, and each model was compared to the previous, less restrictive model. Model fit was assessed by examining differences in -2 log-likelihood (-2LL) statistic between nested models, which approximates to a χ^2 statistic, and a significant change indicates a loss in model fit and a violation of a constraint. As means, variances, and covariances were tested 17 times for each variable, Bonferroni-adjusted significance threshold was set to $\alpha = 0.003$.

Results are presented in Table 7 (Appendix). Means could be equated across twin order for all traits (1.1). Means could be equated between twins and siblings for all traits except PIQ (1.2). Variances could be equated across twin order and between twins and siblings for all traits (2.1–2.2). Using siblings in a twin design assumes covariance terms involving siblings can be equated with those involving DZ twins (Keller et al., 2005); this assumption holds (3.1). Means and variances could be equated across zygosity (1.3 and 2.3) and across families including single-sex and opposite-sex twins (1.4 and 2.4), suggesting there is no difference in MZ and DZ families for any trait under question. MZ correlations could not be equated with DZ correlations for any trait (3.3), providing strong evidence of genetic influences. Lastly, the covariances for MZ twins, DZ twins, and siblings could not be set to zero for any trait (model 3.4) indicating within-family similarity, whereby twins and siblings within families are more similar to each other than they are to randomly drawn members of the population (Keller et al., 2005).

2.6 Model fitting

Phenotypic correlations according to zygosity are presented in Table 1, and plots for MZ, DZ, and sibling correlations can be found on Appendix (Figures 5–7). MZ twins show greater correlations than DZ twins, which suggests genetic influences in all traits. Twin-sibling correlations were considerably lower than DZ correlations for PIQ, perceptual speed, and PSTM, implying a possible twin-specific environmental influence on these traits (Koeppen-Schomerus et al., 2012). Because MZ correlations were greater than twice the DZ correlations for working memory, dominance effects were investigated.

[2]For a more detailed account on statistical assumption tests that accompany twin modelling, see Wainwright et al. (2005a) or Keller et al. (2005)

Table 1: Twin-twin correlations according to zygosity and twin-sibling correlations.

	Correlations						
	MZFF	**MZMM**	**DZFF**	**DZMM**	**DZFM**	**DZMF**	**Sib**
	(116–157)	(91–136)	(117–140)	(99–144)	(94–124)	(107–138)	(309–850)
PIQ	0.81	0.79	0.41	0.52	0.35	0.24	0.28
PercSpd	0.72	0.73	0.37	0.23	0.36	0.36	0.19
WM	0.48	0.45	0.15	0.15	0.17	0.17	0.16
VSTM	0.48	0.53	0.31	0.18	0.35	0.19	0.18
PSTM	0.66	0.76	0.61	0.49	0.59	0.57	0.46
Decode	0.69	0.76	0.28	0.50	0.39	0.37	0.39

Phenotypic covariation between traits was partitioned into several components: additive genetic (A) factor describes the effects of multiple genes exerting their influence in a non-dominant and non-epistatic fashion, which are instead included in the dominant genetic (D) factor. Common environmental (C) effects make family members more similar to one another (e.g., socioeconomic status). Twin-specific environmental (T) factor represents influences not shared by non-twin siblings like pre-natal or linguistic environment (Trombetta et al., 2019). Unique environmental (E) effects make family members different from each other. The E factor also captures measurement error.

Models were fit for the whole sample (MZ = 609, DZ = 1,146, Sib = 435) as well as the subsamples of weaker readers (MZ = 290, DZ = 448, Sib = 158) and stronger readers (MZ = 271, DZ = 493, Sib = 187) using the Cholesky decomposition. The Cholesky includes a latent variable for each of the model's measured traits which is connected to all the variables on its right. It assesses the shared variance with all the other predictors using the latent variables to the left of each trait before attempting to calculate the residual variance in a trait using the latent structures specific to that trait (Bates et al., 2007; Bates et al., 2019). The model functions in a similar manner to hierarchical regression, meaning the order of variables affects the results (Rimfield et al., 2015). As such, the variable of interest—here, nonword reading—is placed last in the model to assess the variance it shares with other predictors. Conversely, general cognitive ability is placed first to account for its contribution on each trait before assessing their independent influence on the subsequent traits.

Twin designs cannot simultaneously estimate C and D factors[3], thus the full ACTE Cholesky was first compared against full ADTE Cholesky. In all groups, ACTE model had better fit compared to the ADTE model (Table 2). Thus full ACTE models were

[3]For more about parameter indeterminacy, see Keller and Coventry (2005).

Table 2: Model fit statistics for all groups.

	Model statistics					
	-2LL	**df**	**$\triangle$-2LL**	**$\triangle$df**	**p**	**AIC**
Stronger readers						
Full ACTE Cholesky	11333.13	4898	NA	NA	NA	11525.13
Full ADTE Cholesky	11348.39	4898	NA	NA	NA	11540.39
Hypothesised ACTE	**11344.62**	**4931**	**11.08**	**33**	**1.00**	**11470.62**
Weaker readers						
Full ACTE Cholesky	11005.12	4451	NA	NA	NA	11197.12
Full ADTE Cholesky	11014.92	4451	NA	NA	NA	11206.92
Hypothesised ACTE	**11017.67**	**4484**	**12.54**	**33**	**1.00**	**11143.67**
Whole sample						
Full ACTE Cholesky	23044.73	9445	NA	NA	NA	23236.93
Full ADTE Cholesky	23129.70	9445	NA	NA	NA	23321.70
Hypothesised ACTE	**23071.25**	**9478**	**26.51**	**33**	**0.78**	**23197.25**

compared against a hypothesised model. Past literature suggested that a single C factor could sufficiently capture all common environmental variation in each trait (Eaves et al., 1984; Lazaroo et al., 2019), and I hypothesised that this would hold true for the twin-specific factor as well. Compared to the full ACTE Cholesky, the hypothesised model resulted in a significant increase in fit in all groups, and results were analysed from these models.

3 Results

3.1 Stronger readers

Full model estimates are on Appendix (Figures 12, 13, and 14). A path diagram with standardised path estimates and bootstrapped 95% ML confidence intervals is presented in Figure 3. In stronger readers, variation in decoding shows genetic and environmental influences, each accounting for roughly 50% of the variability. Genetic variation in phonological decoding is largely independent of the general cognitive factor ($A_{General}$), and 90% of the genetic variance is specific to decoding (A_{Decode})[4]. Neither memory related traits nor perceptual speed contribute independently from general cognitive ability to the genetic variability in phonological decoding. There exists a twin-specific environmental effect (T) for all traits except PSTM. Most loadings are small, accounting for

[4] 0.63^2 / (0.21^2 + 0.63^2)

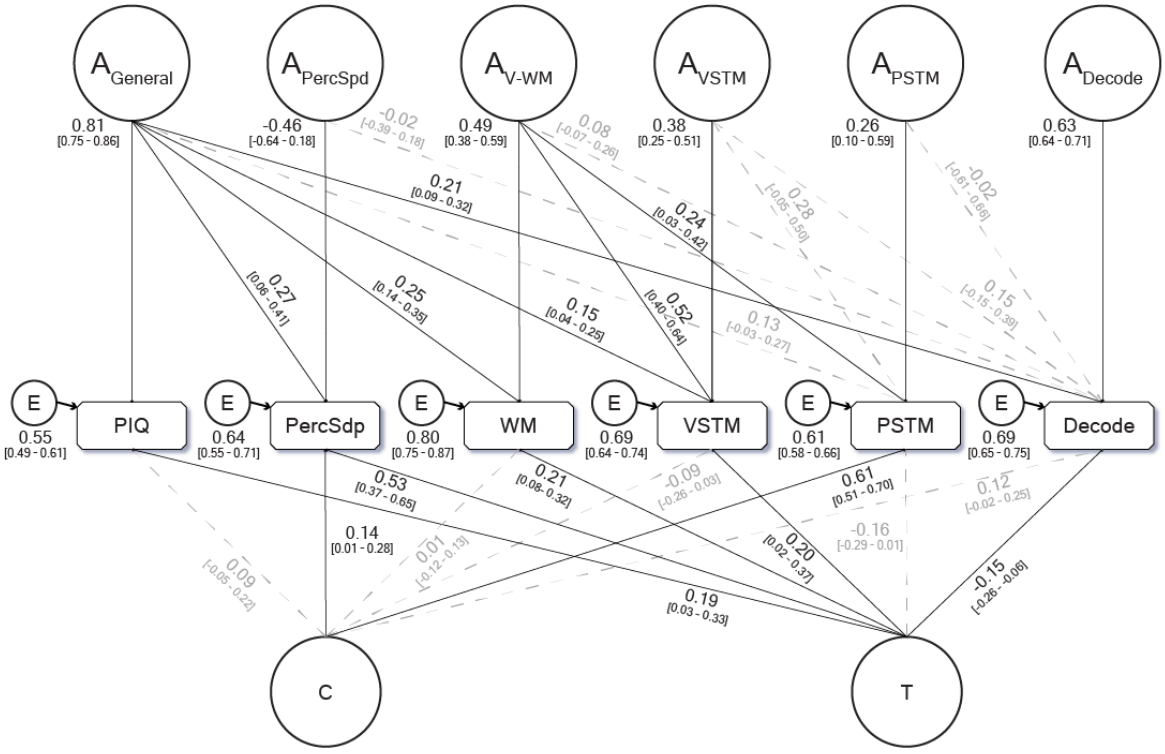

Figure 3: Path diagram for the final model. Non-significant paths are illustrated as dashed lines. Cross-trait unique environmental paths are omitted for clarity. PIQ, performance IQ; PercSpd, perceptual processing speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.

2-4% of the variance, with 28% of the variation in perceptual speed being attributable to this environment. Moreover, the estimate changing from positive to negative implies that a twin-specific environment contributing to better perceptual speed in twins as measured by the Digit Symbol task has a negative influence on their phonological decoding ability.

In general, cognitive measures show no genetic covariance outwith intelligence with the exception of VSTM, for which the genetic covariance it shares with WM is 87% independent of IQ[5]. Verbal working memory traits share a common genetic underpinning ($A_{V\text{-}WM}$), which contributes to the genetic variation in WM, verbal STM, and phonological STM; 62% and 46% of the genetic variation in VSTM and PSTM, respectively, are ascribable to genes common to WM (i.e., genes contributing to better WM contribute to better VSTM and PSTM). The shared environmental component (C) had a negligible effect on stronger readers' perceptual speed but a significant influence on their phonological STM, accounting for 43% of the variability in the trait, with trait-specific genetic influences (A_{PSTM}) having a minimal effect. There is a strong unique environmental effect (E) on each trait, with negligible covariance in E across traits.

Genetic and phenotypic correlations between traits and heritabilities are presented in Table 3. Heritability estimates ranged from 0.22 to 0.65. Genetic correlations be-

[5] (0.49×0.52) / $(0.49 \times 0.52 + 0.25 \times 0.15)$

Table 3: Phenotypic and genotypic correlations and heritabilities for stronger readers.

	PIQ	PercSpd	WM	VSTM	PSTM	Decode
PIQ	**0.65**	0.36	0.19	0.11	0.12	0.15
	[0.56 – 0.74]	[0.31 – 0.40]	[0.11 – 0.24]	[0.05 – 0.16]	[0.06 – 0.18]	[0.07 – 0.20]
PercSpd	0.50	**0.29**	0.19	0.09	0.05	0.05
	[0.14 – 0.74]	**[0.10 – 0.54]**	[0.12 – 0.23]	[0.03 – 0.14]	[-0.01 – 0.10]	[-0.01 – 0.11]
WM	0.45	0.23	**0.31**	0.49	0.16	0.14
	[0.25 – 0.63]	[0.04 – 0.39]	**[0.21 – 0.41]**	[0.45 – 0.52]	[0.12 – 0.20]	[0.09 – 0.18]
VSTM	0.22	0.11	0.80	**0.43**	0.22	0.15
	[0.05 – 0.38]	[0.02 – 0.24]	[0.67 – 0.92]	**[0.33 – 0.55]**	[0.16 – 0.27]	[0.10 – 0.19]
PSTM	0.28	0.14	0.59	0.81	**0.22**	0.18
	[-0.06 – 0.60]	[-0.03 – 0.36]	[0.22 – 0.89]	[0.43 – 0.98]	**[0.10 – 0.36]**	[0.13 – 0.23]
Decode	0.31	0.18	0.25	0.29	0.26	**0.47**
	[0.13 – 0.46]	[-0.08 – 0.57]	[0.06 – 0.44]	[0.12 – 0.47]	[-0.08 – 0.57]	**[0.38 – 0.54]**

Note: Genetic correlations appear below the diagonal, heritabilities appear in **bold** *on the diagonal and phenotypic correlations appear above the diagonal. 95% confidence intervals (bootstrapped) presented in brackets. PIQ, performance IQ; PercSpd, perceptual speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.*

tween cognitive measures and phonological decoding were modest, ranging from 0.18 to 0.31; however, most of the confidence intervals are wide and contain zero, reflecting small power to detect these effects. Bivariate heritability—the proportion of covariance between traits that is explained by genetic factors (e.g., de Vries et al., 2021)—between phonological decoding and other traits ranged from 5% for PSTM to 79% for PIQ[6]. Likewise, genetic correlations between PIQ and other traits ranged from 0.22 to 0.50, and 54–97% of the phenotypic correlations could be accounted for by genetic effects.

3.2 Weaker readers

A path diagram for the final model is presented in Figure 4. In the weaker reading group, additive genetic influences account for 52% of the variability in decoding ability, and the rest is attributable to unique environmental effects. Of the total genetic variance

[6] $0.81 \times 0.21/(0.81 \times 0.21 + 0.09 \times 0.12 + 0.19 \times -0.15 + 0.55 \times -0.01)$

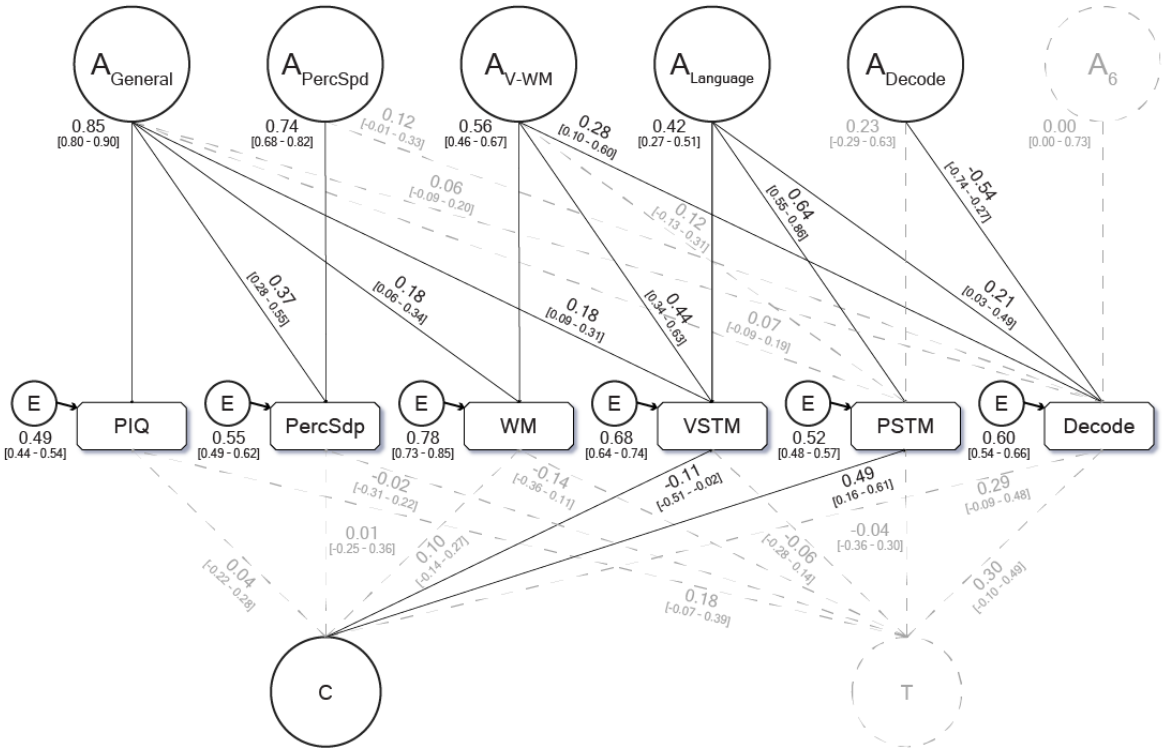

Figure 4: Path diagram for the final model. Non-significant paths are illustrated as dashed lines. Cross-trait unique environmental paths are omitted for clarity. PIQ, performance IQ; PercSpd, perceptual processing speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.

70% is not shared with other phenotypes in the model, and rest of the genetic variation on reading is attributable to additive genetics factors representing verbal working memory ($A_{V\text{-}WM}$) and general language components ($A_{Language}$). Genetic covariance shared between phonological decoding and verbal STM is 34% independent of genes coding for verbal WM. Weaker readers' phonological short-term memory shows moderate environmental influences, which account for 26% of the total variability; genetic variation in PSTM accounts for 44% of the total trait variability and is independent of genetic contributions from other cognitive traits.

Genetic and phenotypic correlations between traits and heritabilities are presented in Table 4. Heritabilities ranged from 0.35 to 0.73, with a noticeable change in estimates for perceptual speed and phonological STM. Phenotypic correlations between phonological decoding and all cognitive measures were modest, ranging from 0.09 to 0.26, with PSTM showing the greatest phenotypic correlation with reading. Genetic correlations between decoding and WM and decoding and VSTM were 0.43 and 0.53, respectively; likewise, verbal STM had strong genetic correlations with other verbal memory measures, with estimates being 0.74 for WM and 0.76 for PSTM; across these traits, 47–79% of their phenotypic correlations could be accounted for by genetic effects. Genetic correlations between PIQ and other measures ranged from 0.10 to 0.45, and phenotypic correlations were largely accounted for by genetic effects with the exception of decoding (36%).

Table 4: Phenotypic and genotypic correlations and heritabilities for weaker readers.

	PIQ	**PercSpd**	**WM**	**VSTM**	**PSTM**	**Decode**
PIQ	**0.73**	0.34	0.13	0.09	0.08	0.15
	[0.65 – 0.82]	[0.28 – 0.39]	[0.07 – 0.19]	[0.03 – 0.16]	[0.02 – 0.14]	[0.07 – 0.21]
PercSpd	0.45	**0.69**	0.11	0.07	0.02	0.09
	[0.33 – 0.65]	**[0.64 – 0.80]**	[0.05 – 0.15]	[0.03 – 0.13]	[-0.04 – 0.09]	[0.02 – 0.15]
WM	0.30	0.14	**0.35**	0.54	0.20	0.17
	[0.08 – 0.60]	[0.04 – 0.31]	**[0.25 – 0.47]**	[0.49 – 0.57]	[0.15 – 0.25]	[0.12 – 0.22]
VSTM	0.28	0.13	0.74	**0.41**	0.30	0.14
	[0.12 – 0.49]	[0.06 – 0.24]	[0.64 – 0.95]	**[0.32 – 0.56]**	[0.25 – 0.35]	[0.09 – 0.19]
PSTM	0.11	0.05	0.19	0.76	**0.48**	0.26
	[-0.14 – 0.30]	[-0.05 – 0.14]	[-0.14 – 0.48]	[0.50 – 0.97]	**[0.32 – 0.72]**	[0.21 – 0.32]
Decode	0.10	0.21	0.43	0.53	0.10	**0.43**
	[-0.18 – 0.30]	[0.01 – 0.48]	[0.19 – 0.84]	[0.38 – 0.84]	[-0.26 – 0.39]	**[0.25 – 0.62]**

Note: Genetic correlations appear below the diagonal, heritabilities appear in **bold** *on the diagonal and phenotypic correlations appear above the diagonal. 95% confidence intervals (bootstrapped) presented in brackets. PIQ, performance IQ; PercSpd, perceptual speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.*

3.3 Whole sample

The final model is presented in Figure 5. When the whole sample was modelled using the hypothesised ACTE model, additive genetic influences account for 34% of the variance in phonological decoding. Genetic variation in decoding is largely independent of the general factor, which accounts for 8% of the genetic variance, with the rest of genetic variation being specific to decoding. The genetic covariance verbal STM shares with working memory is 90% independent of IQ, and the genetic covariance verbal STM shared with phonological STM is 58% independent of working memory.

The common environmental factor shows a significant loading to all variables, accounting for 39% and 27% of the variance in decoding and phonological short-term memory, respectively. Additionally, a moderate to small loadings from the twin-specific environmental component influence variability in perceptual speed and working memory, accounting for 16% and 4% of the variance in these traits. There exists moderate

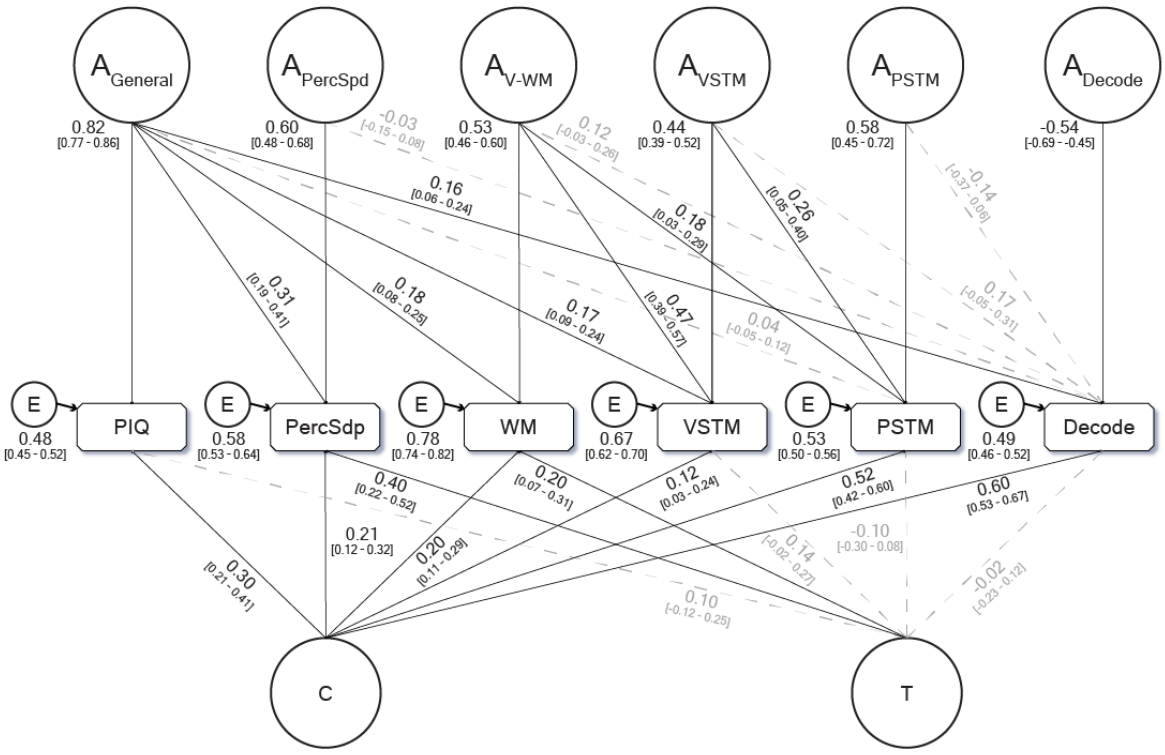

Figure 5: Path diagram for the final model. Non-significant paths are illustrated as dashed lines. Cross-trait unique environmental paths are omitted for clarity. PIQ, performance IQ; PercSpd, perceptual processing speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.

to strong unique environmental effects on each trait, with negligible covariances in E. Heritabilities ranged from 0.32 to 0.67; these are presented alongside genetic and phenotypic correlations in Table 5. Phenotypic correlations between phonological decoding and all cognitive measures were greater than in the subsamples, ranging from 0.16 to 0.35. Genetic correlation between decoding and other measures ranged from -0.02 to 0.38, and 15–70% of the phenotypic correlations were accounted for by genetic effects. Genetic correlation between PIQ and other traits ranged from 0.06 to 0.46, with 15–63% of the phenotypic correlations being accounted for by genetic effects.

Table 5: Phenotypic and genotypic correlations and heritabilities for the whole sample.

	PIQ	PercSpd	WM	VSTM	PSTM	Decode
PIQ	**0.67**	0.38	0.20	0.16	0.18	0.31
	[0.59 – 0.74]	[0.35 – 0.42]	[0.17 – 0.25]	[0.12 – 0.20]	[0.14 – 0.22]	[0.26 – 0.34]
PercSpd	0.46	**0.45**	0.19	0.11	0.08	0.16
	[0.31 – 0.59]	**[0.29 – 0.58]**	[0.16 – 0.23]	[0.08 – 0.16]	[0.04 – 0.12]	[0.12 – 0.20]
WM	0.32	0.15	**0.32**	0.53	0.23	0.24
	[0.15 – 0.45]	[0.05 – 0.24]	**[0.22 – 0.40]**	[0.50 – 0.56]	[0.20 – 0.27]	[0.21 – 0.28]
VSTM	0.26	0.12	0.75	**0.45**	0.31	0.23
	[0.13 – 0.37]	[0.05 – 0.19]	[0.65 – 0.84]	**[0.39 – 0.54]**	[0.27 – 0.34]	[0.20 – 0.27]
PSTM	0.06	0.03	0.27	0.47	**0.44**	0.35
	[-0.08 – 0.18]	[-0.04 – 0.08]	[0.06 – 0.44]	[0.25 – 0.67]	**[0.33 – 0.56]**	[0.31 – 0.38]
Decode	0.26	0.07	0.26	0.38	-0.00	**0.38**
	[0.09 – 0.38]	[-0.16 – 0.24]	[0.01 – 0.46]	[0.16 – 0.56]	[-0.20 – 0.12]	**[0.29 – 0.49]**

Note: Genetic correlations appear below the diagonal, heritabilities appear in **bold** *on the diagonal and phenotypic correlations appear above the diagonal. 95% confidence intervals (bootstrapped) presented in brackets. PIQ, performance IQ; PercSpd, perceptual speed; WM, working memory; VSTM, verbal short-term memory; PSTM, phonological short-term memory; Decode, phonological decoding.*

4 Discussion

The purpose of this thesis was to examine the genetic relations in cognitive processes associated with reading ability and whether distinct aetiological differences in these relationships exist between weaker and stronger readers. The hypothesised ACTE model offered best fit in all groups, and there is a substantial genetic component specific to nonword reading ability. Most of the genetic variance in nonword reading was explained by the additive genetic factor specific to decoding in stronger (90%) and weaker readers (70%) as well as the whole sample (92%). Yet, while the rest of the genetic variance in stronger readers was influenced by the general cognitive factor, in weaker readers these were attributable to genes influencing verbal and phonological working memory.

In the present study, perceptual processing speed exhibited markedly higher heritability among weaker readers (0.69) compared to stronger readers (0.29), but remarkably it shared no genetic variance with nonword reading. Posthuma and de Geus (2008) say that as impaired processing speed has a significant effect in disorders like reading disability, they are expected to overlap in heritability. As group classification in the present study was based on regular and irregular reading and spelling scores, the weaker reading group comprises a blend of weaker readers and surface dyslexics. This suggests a distinct role for processing speed in lexical rather than sublexical reading. This aligns with Stefanac and colleagues' (2019) findings, which indicate a pronounced decline in processing speed specifically in lexical but not in sublexical dyslexia. The whole sample estimates for heritability and genetic/phenotypic correlations between perceptual speed and PIQ were nearly identical to those observed by Posthuma and colleagues (2001). In weaker and stronger readers, 69% and 28% of the total variance in perceptual speed was explained by genetic effects, respectively.

This finding might also explain why phonological decoding showed large influence from genes common to verbal working memory in weaker readers; due to their difficulties in processing visual stimuli—thus preventing lexical access—weaker readers take longer to decode information. This prolonged processing places additional strain on their working memory, causing the system to serve as a bottleneck for learning (Cohen-Mimran & Sapir, 2007; van Leeuwen et al., 2009). Weaker readers experience greater influence in their nonword reading from genes associated with verbal working memory, which accounted for 30% of the genetic variance in decoding. Conversely, in the whole sample only 5% of the genetic variance in decoding was attributable to genes common to verbal working. This is in line with the generalist genes approach introduced by Plomin and Kovas (2005) which follows from the hypothesis that some cognitive disabilities are

merely manifestations of the extreme low-end of normally distributed abilities. Thus while verbal working memory storage might play an important role in early reading acquisition (Nouwens et al., 2017; van Leeuwen et al., 2009), its role is diminished once language skills develop, leading to processes becoming more automated and independent of verbal working memory (Rogers et al., 2022). On the other hand, there seems to be a greater dependency on general cognitive abilities at the higher end of the distribution of reading ability, and 79% of the phenotypic correlation between PIQ and decoding was mediated by common genetic factors in stronger readers. This might then reflect a distinctive feature in stronger readers, whereby their essential processes for decoding are at an adequate level and any differences between individuals can be attributed to general cognitive ability, which allows them to consume more challenging texts, thus facilitating greater vocabulary acquisition.

Unfortunately, interventions focusing on working memory training have been shown to produce only short-term and specific effects which do not generalise to other domains (for a meta-analysis, see Melby-Lervåg & Hulme, 2013). An alternative strategy might involve procedures focusing on improving processing speed and attention, and a randomised individual or group-based neuropsychological intervention by Nukari and colleagues (2019) showed improved effects in dyslexic adults which lasted even after a 5-month follow-up. However, it must be noted that the subjects were Finnish, and as the language is strictly phonemic, with near 1:1 correspondence with orthography and pronunciation, the efficacy of this intervention might not translate to other languages.

Genetic correlations between memory variables tended to be smaller in weaker readers, especially between WM and PSTM. This probably reflects genetic effect specific to phonological processing, which have consistently been found to be a specific source of impairment in weaker readers. Phonological STM showed significant genetic and environmental differences between stronger ($h^2 = 0.22$, $c^2 = 0.37$, $e^2 = 0.38$) and weaker readers ($h^2 = 0.48$, $c^2 = 0.24$, $e^2 = 0.28$), echoing previous work that establishes higher genetic influences for those at the lower end of reading ability (Logan et al., 2012). Genetic correlations between PSTM and nonword reading were not significant in any group, and it did not account for any of the genetic variance in decoding ability. This echoes findings from Logan and colleagues (2011; see also Peterson, 2013), where the same Gathercole and Baddeley's nonword repetition test used in the present study did not correlate with any of the reading measures in their study, including nonword reading. Atop of this, the heritability estimate for it was 0.36, which is within the CI for the present study. Similarly, in a multicohort genome-wide association study (GWAS) by Eising and colleagues (2022) five psychometric reading measures were assessed (word

reading, nonword reading, spelling, phoneme awareness, and nonword repetition), and nonword repetition shared genetic variance with word reading only. They conclude that nonword repetition is genetically more distinct, and it had the highest genetic residual variance not captured by the model. Thus while phonological STM's underlying aetiology might be distinct from sublexical reading, it might be a good behavioural marker of an underlying impairment (Bishop, 2006).

I hypothesised that a single twin-specific component would sufficiently capture all its effects, and this prediction held. Furthermore, it was thought it would have a negative influence on weaker readers' phonological processing. This was based on past literature suggesting postnatal factors influence the amount of linguistic input twins receive (Rutter et al., 2003; Stromswold, 2006; Tomasello et al., 1986; Trombetta et al., 2019). Instead it was found to negative influence decoding in stronger readers atop of positively influencing all other measures outwith phonological memory[7]. This could be explained by synergistic effects, whereby twins with adequate cognitive and linguistic skills are able to provide each other with more developmental opportunities (i.e., conversations, play). However, this does not indicate a special twin-attachment: Mark and colleagues (2017) point out that familial bonds in early childhood and adolescence are relatively forced, whereby siblings have little choice but to engage with each other. Thus given similar context, one would expect to see enhanced socialisation in non-twin siblings too. One way to investigate the validity of this claim would be to compare the twin estimate's magnitude in twins, triplets, and so forth. Should this type of synergistic effect exist, one would expect the T-estimate to increase in nonlinear fashion; if a child's probability to interact with others is influenced by the number of plausible interactees in their vicinity—with the number of possible interactions being represented by the binomial coefficient—then the probability of twins interacting with their sibling(s) is the sum of possible n-way interactions multiplied by the individual encounter likelihood:

$$P(I) = \sum_{k=2}^{n} \binom{n}{k} \cdot p^k q^{n-k}, \text{where } n \geq k \geq 2,\ k \in \mathbb{Z}$$

Unique environmental effects consistently explained greater amount of variance in all traits in stronger readers (30%–64%) than in weaker readers (27%–61%), and this difference was especially large in phonological decoding (48% and 36%, respectively). Thus in stronger readers unique experiences facilitate greater phonological skills. As the participants were late adolescents, majority of them are still engaged with the school

[7]Though the CI indicates this is due to insufficient power only.

system, and the unique experiences can be contextualised around that environment. The strength of these effects might then be an indication of a specific type of evocative gene-environment correlation, whereby brighter students are given more challenging tasks and more opportunities for independent work, thus facilitating unique experiences. Consequently, in accordance with the hypothesis, a single common environmental factor was sufficient to account for all shared environmental effects. Common environmental component was consistently present for PSTM, highlighting the importance of home learning environment for the acquisition of phonological skills.

However, these results must be considered with an asterisk in mind: Derks and colleagues (2004) discuss that while Maximum Likelihood estimation offers a powerful modelling tool for analysing genetic and environmental influences, the method provides biased estimates if the assumption of multivariate normality is not met, leading to the common environmental component to be underestimated and shifting the variance to the unique environmental component. I performed a post-hoc Mardia's test of multivariate normality, and the test rejected the null hypothesis of multivariate normality for all groups at $p < 0.01$. Recovering the correct parameter estimates would require data to be Box-Cox transformed and the use of a liability-threshold model.

Another computational limitation in the present study is that the Cholesky imposes an implicit lower-bound for the variance of each variable, and while this improves the model's interpretability, in multivariate cases this leads to lower than expected Type I error rates, which in turn causes an increase in Type II errors, whereby variance components are falsely judged to be non-significant (Verhulst et al., 2019). In addition to this, the method used to divide people into stronger and weaker readers by splitting the sample in half probably leads to significant overlap in participants within ± 0.5 SD from the mean, thus leading to some differences being hidden from view. Still, as the study's aim was to investigate genetic relations in cognitive processes in a multivariate fashion, a more appropriate threshold would have left the study severely underpowered. An alternative approach would have been to use a regression-based DeFries–Fulker extremes analysis (DF; 1985) to assess the genetic and environmental aetiology in selected and unselected twin-sibling samples. However, DF is not easily extended to the multivariate case (Hawke et al., 2008), and these should be conducted as univariate analyses.

5 Conclusion

This thesis attempted to partition the differences in genetic aetiology between stronger and weaker readers using genetically sensitive twin data. Consistent with previous research, heritabilities for the cognitive abilities known to affect reading most—processing speed and phonological working memory—were higher for those at the lower end of the distribution. Still, most of the genetic variance in decoding was specific to nonword reading, though weaker readers showed more genetic effects related to memory variables. Furthermore, nonword repetition was shown to be genetically distinct from phonological decoding, thus confirming previous studies' findings which suggest that it might be an underlying phenomena for which some weaker readers are able to compensate. Consistent with the predictions, common environmental and twin environmental factor could be uncovered using a single component only. As the twin-specific component is usually integrated into the shared environmental factor in studies not using siblings, these estimates should be examined with caution. While the present study most likely suffered from biased estimates of its own, it benefited from a strong theoretical background, whereby the variables reflected known differences in reading ability across the distribution. The results revealed some distinct patterns between readers of different skill levels which should be investigated further. However, it is worth keeping in mind that reading disabilities do not segregate in families in a simple Mendelian fashion (Astrom et al., 2011), and the heritability of a trait is caused by multiple genetic and environmental factors, with gene-by-environment interactions such as parental education and SES moderating these effects (Bates et al., 2013; Friend et al., 2008; Kremen et al., 2005). As such it is vital to research not just individual traits' influence on reading ability but how they interact with one other. Finally, Eising and colleagues (2022, p. 1) say "our unique capacities for spoken and written language are fundamental features of what makes us human, yet the biological bases remain largely mysterious". With this thesis now complete, it is hoped that the answer to that question is one step closer.

Acknowledgments

Like everything in life, this project would not have been possible without all the people, past and present, who have helped me become who I am today. As there are more people I could ever thank, I will simply mention the selected few: I would like to thank everyone at Eira for believing in me and even moreso for helping me believe in myself, with a special thanks to Leena, Tiina, Teija, Kalle, and Roy. Thanks to Jonna for her constant support and unconditional love – studying abroad, away from her and our dog Nemo has not been easy, and I miss both dearly. Thanks to all the lovely lecturers here in Edinburgh who helped make life during COVID a little less miserable. To Zach I want to extend a special thanks for providing me with an opportunity of a lifetime. A final thanks goes to Michelle for guiding me through this project and for the not-so-simple act of putting up with me.

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6 Appendix

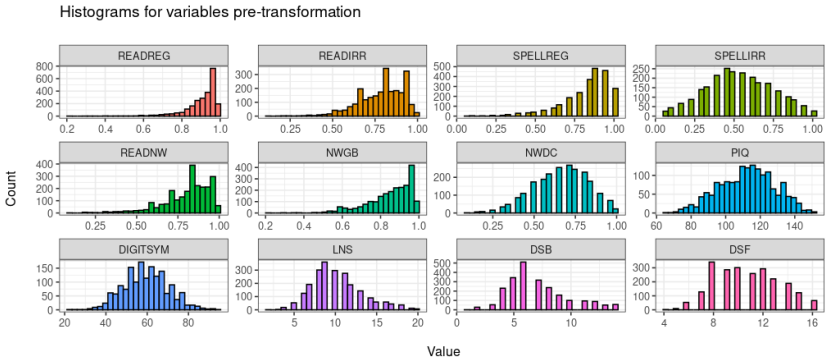

Figure 6: Histograms for variables before transformation and standardisation.

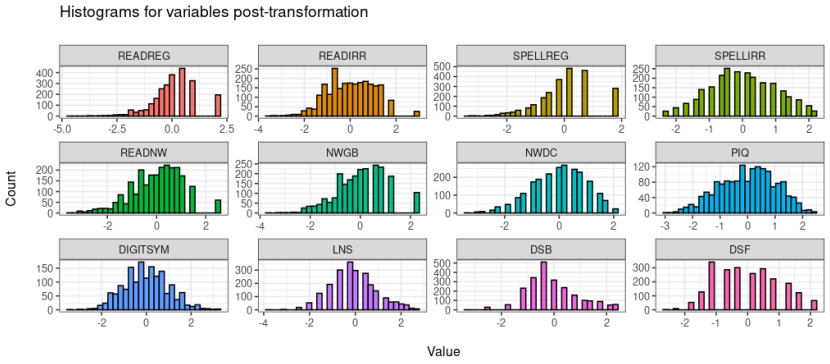

Figure 7: Histograms for variables post-transformation and standardisation.

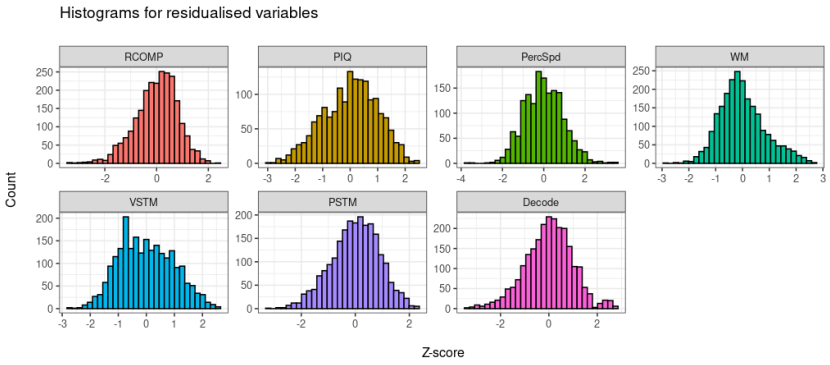

Figure 8: Histograms for final variables, residualised for sex and age.

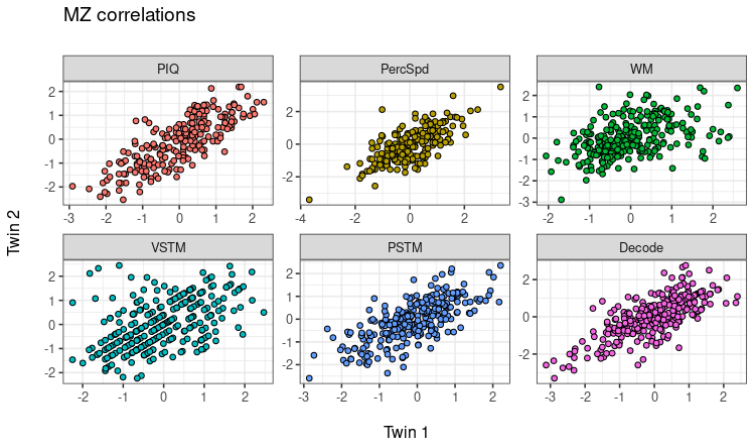

Figure 9: MZ correlations.

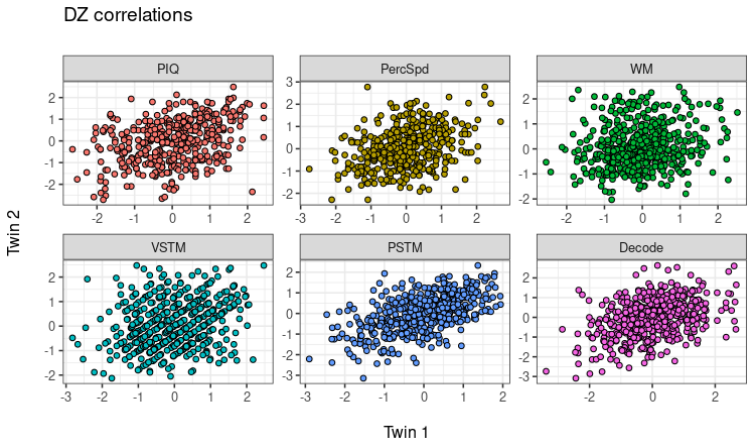

Figure 10: DZ correlations.

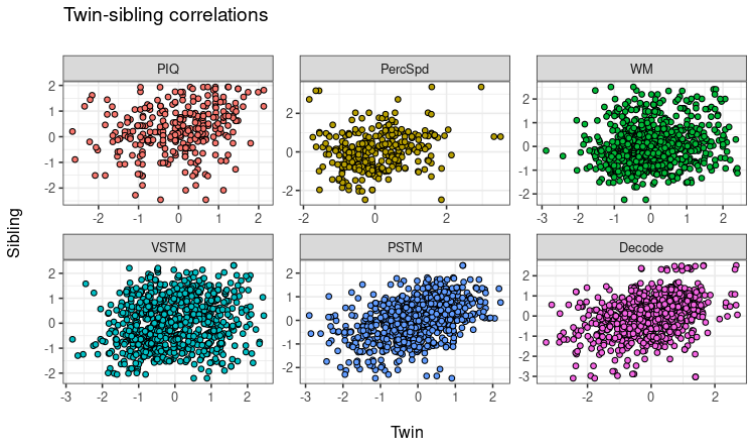

Figure 11: Twin-sibling correlations.

Table 6: T-tests comparing full sample to study sample.

	Group means		Statistics		
	Full	**Study**	**df**	**p-value**	**t-value**
PIQ	-0.00	-0.01	3907	0.75	-0.321
PercSpd	0.00	0.00	2696	0.99	0.015
WM	0.00	-0.01	3722	0.63	-0.484
VSTM	-0.00	-0.01	2658	0.70	-0.385
PSTM	0.00	-0.01	3724	0.69	-0.397
Decode	0.00	-0.01	3717	0.71	-0.376

Table 7: Assumption tests for twin models.

		Difference in model fit, χ^2					
	Δdf	**PIQ**	**PercSpd**	**WM**	**VSTM**	**PSTM**	**Decode**
Means models							
1.1. MZ = MZ & DZ = DZ	6	6.10	5.93	11.55	7.40	4.51	7.18
1.2. MZ/DZ = Sib	6	30.33^b	7.30	5.21	11.62	10.88	4.49
1.3. MZ = DZ	3	2.87	0.02	4.29	4.14	7.02	4.18
1.4. Opposite sex = Same sex	2	0.25	2.10	0.43	1.75	0.92	1.40
Variance models							
2.1. Twin 1 = Twin 2	6	2.44	4.41	3.21	5.42	1.56	10.83
2.2. Twins = Sibling	6	4.77	5.22	8.36	1.13	4.69	17.24*
2.3. MZ = DZ	3	0.89	8.96*	5.12	2.57	2.76	0.98
2.4. Opposite sex = Same sex	2	0.07	1.93	0.94	0.28	2.97	2.83
Covariance models							
3.1. MZ/DZ-Sib = DZ-DZ	10	12.26	22.51*	16.56	3.62	8.57	18.98*
3.2. Opposite sex = Same sex	4	5.52	1.49	3.45	0.91	6.05	4.75
3.3. MZ-MZ = DZ-DZ	3	59.57^b	42.10^b	24.02^b	15.53^b	27.34^b	29.01^b
3.4. All covariances = 0	1	188.95^b	133.75^b	64.25^b	110.01^b	378.23^b	509.22^b

* = $p < 0.05$, ^{b} = $p < 0.003$ (Bonferroni-corrected).

STRONGER READERS

Variable	name	Est.	SE	CI 5%	CI 95%
PIQ	a11	0.81	0.04	0.75	0.86
PercSpd	a21	0.27	0.10	0.06	0.41
WM	a31	0.25	0.06	0.14	0.35
VSTM	a41	0.15	0.07	0.04	0.25
PSTM	a51	0.13	0.09	-0.03	0.27
Decode	a61	0.21	0.07	0.09	0.32
PercSpd	a22	-0.46	0.14	-0.64	-0.18
Decode	a62	-0.02	0.17	-0.39	0.18
WM	a33	0.49	0.07	0.38	0.59
VSTM	a43	0.52	0.07	0.40	0.64
PSTM	a53	0.24	0.12	0.03	0.42
Decode	a63	0.08	0.10	-0.07	0.26
VSTM	a44	0.38	0.10	0.25	0.51
PSTM	a54	0.28	0.17	-0.05	0.50
Decode	a64	0.15	0.17	-0.15	0.39
PSTM	a55	0.26	0.14	0.10	0.59
Decode	a65	-0.02	0.46	-0.61	0.66
Decode	a66	0.63	0.24	0.64	0.71
PIQ	c11	0.09	0.09	-0.05	0.22
PercSpd	c21	0.14	0.08	0.01	0.28
WM	c31	0.01	0.08	-0.12	0.13
VSTM	c41	-0.09	0.09	-0.26	0.03
PSTM	c51	0.61	0.07	0.51	0.70
Decode	c61	0.12	0.09	-0.02	0.25
PIQ	tw11	0.19	0.09	0.03	0.33
PercSpd	tw21	0.53	0.12	0.37	0.65
WM	tw31	0.21	0.07	0.08	0.32
VSTM	tw41	0.20	0.11	0.02	0.37
PSTM	tw51	-0.16	0.09	-0.29	0.01
Decode	tw61	-0.15	0.06	-0.26	-0.06
PIQ	e11	0.55	0.04	0.49	0.61
PercSpd	e21	0.06	0.06	-0.04	0.16
WM	e31	-0.11	0.06	-0.21	-0.02
VSTM	e41	-0.06	0.06	-0.16	0.02
PSTM	e51	-0.01	0.06	-0.11	0.08
Decode	e61	-0.01	0.06	-0.12	0.09
PercSpd	e22	0.64	0.05	0.55	0.71
WM	e32	0.02	0.06	-0.11	0.11
VSTM	e42	-0.07	0.05	-0.16	0.01
PSTM	e52	0.02	0.04	-0.03	0.10
Decode	e62	0.07	0.06	-0.02	0.16
WM	e33	0.80	0.04	0.75	0.87
VSTM	e43	0.19	0.05	0.10	0.26
PSTM	e53	0.04	0.04	-0.01	0.11
Decode	e63	0.09	0.04	0.02	0.16
VSTM	e44	0.69	0.03	0.64	0.74
PSTM	e54	0.07	0.04	0.00	0.12
Decode	e64	0.07	0.04	0.01	0.14
PSTM	e55	0.61	0.03	0.58	0.66
Decode	e65	-0.01	0.05	-0.09	0.07
Decode	e66	0.69	0.03	0.65	0.75

Figure 12: Standardised path estimates.

WEAKER READERS

Variable	name	Est.	SE	CI 5%	CI 95%
PIQ	a11	0.85	0.05	0.80	0.90
PercSpd	a21	0.37	0.09	0.28	0.55
WM	a31	0.18	0.08	0.06	0.34
VSTM	a41	0.18	0.07	0.09	0.31
PSTM	a51	0.07	0.09	-0.09	0.19
Decode	a61	0.06	0.09	-0.09	0.20
PercSpd	a22	0.74	0.06	0.68	0.82
Decode	a62	0.12	0.12	-0.01	0.33
WM	a33	0.56	0.07	0.46	0.67
VSTM	a43	0.44	0.09	0.34	0.63
PSTM	a53	0.12	0.14	-0.13	0.31
Decode	a63	0.28	0.14	0.10	0.60
VSTM	a44	0.42	0.08	0.27	0.51
PSTM	a54	0.64	0.13	0.55	0.86
Decode	a64	0.21	0.15	0.03	0.49
PSTM	a55	0.23	0.26	-0.29	0.63
Phon	a65	-0.54	0.21	-0.74	-0.27
Decode	a66	0.00	0.17	0.00	0.73
PIQ	c11	0.04	0.15	-0.22	0.28
PercSpd	c21	0.01	0.18	-0.25	0.36
WM	c31	0.10	0.12	-0.14	0.27
VSTM	c41	-0.11	0.11	-0.51	-0.02
PSTM	c51	0.49	0.15	0.16	0.61
Decode	c61	0.29	0.17	-0.09	0.48
PIQ	tw11	0.18	0.14	-0.07	0.39
PercSpd	tw21	-0.02	0.16	-0.31	0.22
WM	tw31	-0.14	0.15	-0.36	0.11
VSTM	tw41	-0.06	0.13	-0.28	0.14
PSTM	tw51	-0.04	0.20	-0.36	0.30
Decode	tw61	0.30	0.18	-0.10	0.49
PIQ	e11	0.49	0.03	0.44	0.54
PercSpd	e21	0.04	0.06	-0.06	0.13
WM	e31	-0.01	0.06	-0.11	0.09
VSTM	e41	-0.09	0.06	-0.20	0.01
PSTM	e51	0.00	0.04	-0.06	0.08
Decode	e61	0.05	0.06	-0.04	0.15
PercSpd	e22	0.55	0.04	0.49	0.62
WM	e32	0.07	0.05	-0.01	0.16
VSTM	e42	0.02	0.05	-0.06	0.10
PSTM	e52	-0.02	0.04	-0.08	0.03
Decode	e62	-0.05	0.05	-0.14	0.03
WM	e33	0.78	0.04	0.73	0.85
VSTM	e43	0.33	0.05	0.22	0.39
PSTM	e53	0.09	0.04	0.03	0.18
Decode	e63	0.03	0.04	-0.05	0.07
VSTM	e44	0.68	0.03	0.64	0.74
PSTM	e54	-0.02	0.04	-0.09	0.03
Decode	e64	-0.05	0.04	-0.13	-0.01
PSTM	e55	0.52	0.03	0.48	0.57
Decode	e65	0.14	0.04	0.10	0.23
Decode	e66	0.60	0.04	0.54	0.66

Figure 13: Standardised path estimates.

WHOLE SAMPLE

Variable	name	Est.	SE	CI 5%	CI 95%
PIQ	a11	0.82	0.03	0.77	0.86
PercSpd	a21	0.31	0.07	0.19	0.41
WM	a31	0.18	0.05	0.08	0.25
VSTM	a41	0.17	0.05	0.09	0.24
PSTM	a51	0.04	0.05	-0.05	0.12
Phon	a61	0.16	0.06	0.06	0.24
PercSpd	a22	0.60	0.06	0.48	0.68
Phon	a62	-0.03	0.07	-0.15	0.08
WM	a33	0.53	0.05	0.46	0.60
VSTM	a43	0.47	0.06	0.39	0.57
PSTM	a53	0.18	0.08	0.03	0.29
Phon	a63	0.12	0.09	-0.03	0.26
VSTM	a44	0.44	0.05	0.39	0.52
PSTM	a54	0.26	0.11	0.05	0.40
Phon	a64	0.17	0.11	-0.05	0.31
PSTM	a55	0.58	0.11	0.45	0.72
Phon	a65	-0.14	0.16	-0.37	0.06
Phon	a66	-0.54	0.21	-0.69	-0.45
PIQ	c11	0.30	0.06	0.21	0.41
PercSpd	c21	0.21	0.06	0.12	0.32
WM	c31	0.20	0.05	0.11	0.29
VSTM	c41	0.12	0.06	0.03	0.24
PSTM	c51	0.52	0.06	0.42	0.60
Phon	c61	0.60	0.05	0.53	0.67
PIQ	tw11	0.10	0.11	-0.12	0.25
PercSpd	tw21	0.40	0.11	0.22	0.52
WM	tw31	0.20	0.08	0.07	0.31
VSTM	tw41	0.14	0.09	-0.02	0.27
PSTM	tw51	-0.10	0.12	-0.30	0.08
Phon	tw61	-0.02	0.11	-0.23	0.12
PIQ	e11	0.48	0.02	0.45	0.52
PercSpd	e21	0.06	0.04	0.00	0.12
WM	e31	-0.04	0.04	-0.10	0.03
VSTM	e41	-0.07	0.04	-0.14	0.00
PSTM	e51	0.01	0.03	-0.04	0.07
Phon	e61	0.00	0.03	-0.05	0.06
PercSpd	e22	0.58	0.03	0.53	0.64
WM	e32	0.02	0.04	-0.05	0.08
VSTM	e42	-0.03	0.03	-0.09	0.03
PSTM	e52	0.00	0.03	-0.04	0.05
Phon	e62	0.03	0.03	-0.01	0.08
WM	e33	0.78	0.03	0.74	0.82
VSTM	e43	0.25	0.04	0.18	0.30
PSTM	e53	0.06	0.03	0.02	0.11
Phon	e63	0.05	0.03	0.00	0.09
VSTM	e44	0.67	0.02	0.62	0.70
PSTM	e54	0.05	0.03	0.01	0.10
Phon	e64	-0.01	0.02	-0.05	0.03
PSTM	e55	0.53	0.02	0.50	0.56
Phon	e65	0.07	0.02	0.04	0.11
Phon	e66	0.49	0.02	0.46	0.52

Figure 14: Standardised path estimates.