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Genetic and Environmental Influences on Inattention, Hyperactivity-Impulsivity, and Reading: Kindergarten to Grade 2

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Abstract

Twin children from Australia, Scandinavia and the USA were assessed for inattention, hyperactivity-impulsivity and reading across the first three school years. Univariate behavior-genetic analyses indicated substantial heritability for all three variables in all years. Longitudinal analyses showed one genetic source operating across the time span and a second entering in the second school year for each variable, though possibly not reliable for inattention. Other analyses confirmed previous findings of pleiotropy (shared genes) between inattention and reading, and showed that this genetic overlap is in place from kindergarten onwards and is restricted to one of the genetic sources that affect reading and inattention. The results extend previous conclusions about the developmental trajectories of inattention, hyperactivity-impulsivity, and reading and their relationships. Limitations of this study are discussed, as are educational implications.

We are conducting a longitudinal twin study of early literacy, language, and aspects of behavior to identify genetic and environmental influences on these processes, on their developmental trajectories, and on their interactions. Data come from monozygotic (MZ) and dizygotic (DZ) twin pairs in Australia, Norway, Sweden and the United States starting in the children's final preschool year and continuing through kindergarten, Grade 1 and Grade 2. We refer to the project as the *International Longitudinal Twin Study (ILTS)*. The focus of the current paper is the behavioral measures of attention and hyperactivity-impulsivity, treated as continuous variables rather than as clinical categories. The questions that we address are (1) the genetic and environmental influences on these constructs in each of the first three school years, (2)

developmental change and continuity in them across this time span, and (3) the relationship between these constructs and early reading.

Relevant Findings from the ILTS

At the preschool level, we have shown that the composite measures of phonological awareness, rapid naming, and verbal memory that we developed are each subject to substantial genetic influence (Byrne et al., 2002; Samuelsson et al., 2005), and so are measures of inattention and hyperactivity-impulsivity derived from parent endorsements of ADHD symptoms (Willcutt, Betjemann, Wadsworth et al., 2007). Print knowledge, vocabulary and grammar/morphology, in contrast, are more subject to influence from factors in twins' shared environments, likely to be located in the home and/or preschool. Importantly, each of the preschool variables correlated to a small but significant extent with inattention, and shared genes were the most consistent influence on each of these phenotypic correlations, with genetic correlations ranging from .23 to .48 (Willcutt, Betjemann, Wadsworth, et al., 2007). Hyperactivity did not correlate significantly with any of the preschool variables.

At the end of the first school year (kindergarten) and again in Grades 1 and 2, reading, spelling and rapid naming are seen to be substantially heritable (Byrne et al., 2006, 2007, 2008). There are negligible levels of shared environment influence on literacy, but measures of grammar and vocabulary continue to show shared environment effects. In this paper, we report for the first time on the genetic and environmental influences on inattention and hyperactivity-impulsivity in these early school years, and on their relations with reading.

As well as generating univariate estimates, it is possible to conduct multivariate analyses to estimate the degrees of genetic and environmental overlap and independence among variables. These estimates are quantitatively expressed as genetic correlations. For example, a genetic correlation of 1 would indicate that the same genetic influences are accounting for individual differences in two different variables, or in the same variable across development. A genetic correlation less than 1 suggests at least partially separate genetic influences. It is important to note that these estimates are independent of the magnitudes of genetic influence or heritability for each variable. Heritability could be high on one variable and low on another, but the genetic correlation could still be 1 if the same genetic factors are at play for both variables (Plomin, DeFries, McClearn, & McGuffin, 2008). The genetic correlations between inattention and the preschool variables reported earlier were derived from such multivariate analyses.

Developmental behavior-genetic analyses have shown that there are significant shared genetic influences on word reading at kindergarten and Grade 1, but also new independent genetic influences that emerge in Grade 1 (Byrne et al., 2006, 2007). We have also shown that there is a high phenotypic and a high genetic correlation between word reading and reading comprehension in Grade 1 (Byrne et al., 2007). Because of this latter finding and because studies of clinically significant reading difficulties typically use measures of single-word reading, we are concentrating just on the highly reliable variable of word identification efficiency in this current paper. We report multivariate analyses of this measure, of inattention and hyperactivity-impulsivity, and of the relations among these variables from kindergarten to Grade 2.

The project also allows us to probe the cognitive underpinnings of early literacy development. For instance, Byrne et al. (2008) reported a high genetic correlation between second grade spelling and the ability to fix the orthographic patterns of novel words in memory, evidence, they suggested, for a genetically-influenced learning rate parameter in early literacy growth. In this paper, we address the relationship between reading and inattention across the normal range, an issue that is motivated by the well-recognized comorbidity between inattention and reading disability.

The Potential Importance of Comorbid ADHD

Comorbidity, the co-occurrence of two or more disorders in the same individual, is the rule, rather than the exception, for virtually all complex disorders (e.g., Angold et al., 1999). This pattern holds true for reading difficulties, as 60% of children with reading disability (RD) meet criteria for at least one additional diagnosis (e.g., Trzieniecki, Moffitt, Caspi, Taylor, & Maughan, 2006; Willcutt & Pennington, 2000a, 2000b). The most common comorbidity is attention-deficit/hyperactivity disorder (ADHD), which co-occurs in 20–40% of children with RD (Willcutt & Pennington, 2000a). It is also generally observed that reading difficulties accompany inattention rather than hyperactivity (e.g., Chhabildas, Pennington, & Willcutt, 2001; Massetti et al., 2008; Rietveld, Hudziak, Bartels, Beijsterveldt & Boomsma, 2004; Willcutt & Pennington, 2000a), and again we are able to check on this with the current data within the normal-range. As we argue below, inattention and hyperactivity are best considered continuous variables, as is reading ability, and understanding their relations across the full range can potentially provide clues to clinical comorbidity.

Understanding comorbidity is important for several reasons. First, there is the practical question of which condition to treat first, especially if limited resources are available. In addition, a number of studies suggest that the presence of the second disorder may be a marker for a subgroup of individuals who differ in important ways from individuals with the disorder in isolation. For example, recent results suggest that genetic influences on reading difficulties may be stronger in individuals who also have ADHD than those with RD alone (Willcutt, Pennington, Olson, & DeFries, 2007). Similarly, several studies have found that in comparison to the group with RD alone, the comorbid group exhibited more severe neurocognitive weaknesses, were more likely to experience negative academic and social outcomes, and may be less likely to respond to interventions (e.g., Purvis & Tannock, 2000; Rabiner & Malone, 2004; Rucklidge & Tannock, 2002; Willcutt, Pennington, Chhabildas, Olson, & Hulslander, 2005; Willcutt, Betjemann, Pennington, Olson, DeFries, et al., 2007). In this study we conduct the first longitudinal behavioral genetic analyses of the relationships among reading ability and inattention from kindergarten to second grade.

Categorical versus Continuous Definitions of RD and ADHD

Although many studies of comorbidity have used categorical diagnoses of ADHD or RD, increasing evidence suggests that RD, ADHD, and most other complex disorders are defined by extreme scores on a continuous underlying distribution of reading or attention (e.g., Barkley, 1997; Hay, Bennett, Levy, Sergeant, & Swanson, 2007; Plomin & Kovas, 2005; Plomin, Kovas, & Hayworth, 2007; Willcutt, Pennington, & DeFries, 2000). If the underlying distribution of liability is dimensional, dichotomization into a categorical variable reduces statistical power by eliminating individual differences in severity among those with the disorder, along with variability in subthreshold symptomatology. Alternative methods such as variance components analysis of dimensional measures provide greater statistical power and versatility by using information about the entire continuum of scores.

Factor analytic studies of ADHD provide a second argument for a dimensional measurement approach. The factor structure of DSM-IV ADHD symptoms (see Table 2 for an abbreviated list of these symptoms) has been examined in over 50 samples, and nearly all of these studies have found that symptoms of ADHD are best described by correlated but separable dimensions of behavior characterized by inattention/disorganization and hyperactivity-impulsivity (e.g., Hartman et al., 2001; Wolraich, Feurer, Hannah, Baumgaertel, & Pinnock, 1998). The two symptom dimensions have different developmental trajectories, with mean levels of hyperactivity-impulsivity symptoms declining more than mean levels of inattention across development (Rietveld et al., 2004; Smith, Barkley & Shapiro, 2006). Further support for the external validity of this distinction is provided by differential correlations with measures of

social functioning, comorbid psychopathology, and global impairment (e.g., Lahey & Willcutt, 2002; Molina, Smith, & Pelham, 2001), and studies of school-age samples have consistently found that both phenotypic and genetic correlations with reading difficulties are higher for inattention than hyperactivity-impulsivity (e.g., Lahey & Willcutt, 2002; Molina et al., 2001; Wolraich et al., 1998; Willcutt, Pennington, Olson, & DeFries, 2007).

Based on these lines of evidence, we used continuous measures of reading, inattention, and hyperactivity-impulsivity in the present study. In addition to the primary analyses of the relation between reading and ADHD symptoms, we also tested the factor structure of ADHD in our sample and the longitudinal etiology of inattention and hyperactivity-impulsivity from kindergarten through second grade. Because we have adopted a dimensional approach in an unselected sample of twins, our analyses are informative regarding strengths and weaknesses in attention and activity across the entire distribution. On the other hand, the measure of inattention and hyperactivity-impulsivity is explicitly derived from the symptoms of ADHD described in DSM-IV. Therefore, for clarity and consistency with the clinical literature we have maintained the DSM-IV labels of “inattention” and “hyperactivity-impulsivity” rather than more neutral counterparts such as “attention” and “activity.”

Etiology of ADHD

In a review of studies that together encompass over 12,000 twins pairs aged 6 to 18 years, Willcutt et al. (2005) reported an average heritability for ADHD of .73. The environment that twins share, such as the home and school, appears to exert little influence on ADHD, with the non-heritable component attributable to nonshared environmental influences. There is some evidence that some genetic influences may be involved only in the onset of ADHD and may not contribute to its continuation (Thapar, Langley, Asherson, & Gill, 2007), but other studies suggest that for the most part the stability that is seen in ADHD is largely due to genetic continuity (Kuntsi, Rijdsdijk, Ronald, Asherson, & Plomin, 2005). Our longitudinal data allow us to contribute to this debate by examining possible genetic continuity and change across the three earliest years of school in normal-range variability.

Etiology of Comorbidity between Attention Deficit and Reading Disability

The causes of comorbidity between RD and ADHD remain uncertain. Some longitudinal studies suggest that attention deficits lead to later reading problems, possibly due to interference with reading instruction (e.g., Dally, 2006; Fergusson & Horwood, 1992). In contrast, others suggest that early reading difficulties predict later attention problems (e.g., McGee, Prior, Williams, Smart, & Sanson, 2002). A third possibility is that RD and ADHD may be due to shared genetic or environmental risk factors that increases risk for both disorders. This common genetic etiology hypothesis was supported at preschool in the current sample and in several studies of school-age children (e.g., Trzieniecki et al., 2006; Willcutt, Betjemann, Pennington, et al., 2007a, Willcutt, Pennington, Olson, & DeFries, 2007). Our longitudinal design facilitates a test of these three competing models that attribute the relationship between reading ability and inattention to (1) common genetic influences, (2) a causal relation in which inattention directly affects reading ability, or (3) a causal relation in which reading ability leads to symptoms of inattention.

Method

Participants

Data were collected from 1978 children, 986 members of monozygotic pairs (493 pairs) and 992 members of dizygotic pairs (496 pairs); see Table 1 for details of age and gender. The Australian twins were recruited from the National Health and Medical Research Council's Australian Twin Registry. Twins in the United States were recruited from the Colorado Birth

Registry and twins from Scandinavia were recruited from the Medical Birth Registries in Norway and Sweden. The Australian and Scandinavian families were approached by mail, with an approximate 60% participation rate. In the US, the families of twins were approached by phone when the children were 4. Of the 60% of families who could be contacted, 88% agreed to participate. Families in the US were paid \$100 for participation. A criterion for selection into the study was that participants' first language be that of their country of residence, i.e. English, Swedish or Norwegian. The zygosity of the children was determined by DNA analysis, collected via a cheek swab, or in a minority of cases by items from the questionnaire by Nichols and Bilbro (1966). There were no differences in the mean years of education of the parents across countries; Australia $M = 13.4$, United States $M = 14.1$, Scandinavia $M = 13.9$.

Measures

Reading ability—The test of word reading efficiency (TOWRE; Torgesen, Wagner & Rashotte, 1999) was used to assess reading ability. This test has two subtests; sight-word and phonemic decoding efficiency. Within each subtest the child was required to read as many real words (sight words) or nonwords (phonemic decoding) as they could from a list provided within 45 seconds. Each subtest has two forms; we used both for more reliable estimation and averaged the scores for each child. There is a high correlation between the two subtests, sight words and phonemic decoding (greater than .85 in various estimates from this project—Byrne et al., 2007), so we further averaged the subtest scores to form a single reading composite. The published test-retest reliability for 6 to 9 year olds is .97 for word and .90 for non-word reading (Torgesen et al., 1999).

Attention-deficit/hyperactivity disorder—The Disruptive Behaviour Rating Scale (DBRS; Barkley & Murphy, 1998) was the measure used to identify traits of inattention and hyperactivity-impulsivity. The first 9 questions address symptoms of inattention and questions 10 – 18 were specific to hyperactivity-impulsivity. The DBRS uses a four point scale as: *never or rarely-0, sometimes-1, often-2, very often-3*. Scale items are shown in Table 2, along with the results of a factor analysis of the subscales (see Results). Data for the DBRS for each child was collected from parent and teacher wherever possible. Inter-rater agreement between parent and teacher in Grade 3, where we had the highest number of teacher ratings, was moderate, at .52 averaged across all available observations (maximum N pairs = 209). However the parent version was used for these analyses to maintain consistency with our preschool analyses (Willcutt, Betjemann, Wadsworth, et al., 2007). The DBRS has previously been shown to be a valid predictor of ADHD symptoms in young children (Lahey et al., 2004). Test-retest reliability has ranged from .49 to .75 for periods of one and two years (Willcutt, Betjemann, Pennington, et al., 2007), indicating moderate to high stability.

Testing Procedures

Informed consent was obtained from parents prior to their child's participation and children gave verbal assent. Data were collected from the children during an assessment that lasted approximately one hour in a quiet room at school or home, and children received stickers intermittently as a form of encouragement. The tests included measures other than the TOWRE, but in this report we use only the TOWRE results as the signature measure of reading. For a description of the full testing protocol, see Byrne et al. (2006, 2007, 2008). In Australia and the US, each twin within a pair was examined by a different tester, with both assessments conducted simultaneously. The children in Scandinavia were assessed by a single tester, with twins within a pair being tested on the same day (Samuelsson et al., 2005). The three assessments were given at approximately one year intervals.

The DBRS was mailed to parents shortly after the twins had been assessed each year. There was an overall return rate of 83.2%, with very similar rates for each of the three years and for parents of monozygotic and dizygotic twins.

Analyses

Assumptions and data cleaning—Within this study, structural equations are the primary form of analysis. The assumptions underlying their use include univariate and multivariate normality, linearity, homoscedasticity, multicollinearity (Tabachnick & Fidell, 2007) and stable measurement (Rogosa, 1980). An inspection of the dataset revealed that the behavioural variables were positively skewed and there were a number of outliers. Therefore the data were transformed to produce the greatest adherence to normality, using square root transformations for reading and inattention and log transformations for hyperactivity-impulsivity. The skewness values of the transformed variables were less than 1, and Levene's test for each of the variables was non-significant. Individual scores were truncated to within ± 3 SD from the mean.

Age- and sex- adjusted scores were created by regressing each measure onto age and sex and calculating residuals. Age and sex barely correlated with the measures; for example, for age and TOWRE in kindergarten, $r = -.07$ and for gender and TOWRE in kindergarten, $r = -.09$. Nevertheless, it is standard practice in twin research to adjust for age and sex correlations because these can bias the genetic and environmental estimates.

Scores were also standardized within country because country differences have previously been found for the behavioural and reading variables (Samuelsson et al., 2005, 2008; Willcutt, Betjemann, Wadsworth et al., 2007).

Genetic Modeling

A univariate analysis generated by the MX statistical package (Neale, Boker, Xie & Maes, 2002) was used to provide the estimates of additive (A), common environment (C), and unique environment (E) effects underlying reading ability, inattentive and hyperactive-impulsive traits. Because nonadditive (or dominance, D) genetic effects and C cannot be modeled simultaneously using twins reared together, separate models were used to estimate D when there was evidence of non-additive genetic influence, namely when the MZ correlation was more than twice as large as the DZ correlation. Note that E includes measurement error. For multivariate modeling we employed Cholesky decomposition. Within this model, the effect of genes and environment on one variable are assessed after the effects of other correlated variables are taken into account, in a manner similar to hierarchical regression. For example, in a simple two factor Cholesky model, genetic and environmental estimates for the first factor are the same as would be found in a univariate analysis. The estimates for the second factor include genetic and environmental influences that are shared with the first factor, and also influences specific to the second factor that are not shared with the first factor.

We separately modeled inattention, reading and hyperactivity-impulsivity ratings from kindergarten through to Grade 2 to examine continuity and change of each of these traits across time. We also modeled inattention and reading together across the three time points to estimate genetic and environmental effects shared between the traits over this developmental period.

Direction-of-Causation

We also contrasted a Cholesky decomposition model of the relation between inattention and reading with what is known as a direction-of-causation (DOC) model (Duffy & Martin, 1994; Heath et al., 1993). We did this separately for each school grade, and we employed two DOC models, one capturing a causal link from inattention to reading, the other capturing the

reverse order, reading to inattention. The Cholesky model assumes that common genetic and environmental sources account for the covariation between inattention and reading, represented by paths a_{21} , c_{21} , and e_{21} in Figure 1. The DOC model assumes a causal relationship between the two variables by replacing the shared genetic and environmental paths with a path that represents a direct causal influence of inattention on reading, or reading on inattention, path i in Figure 1. The models can be compared to see if one provides a significantly better fit to the data. A better fit of a DOC model would support the causal direction implied in that model.

Results

Factor Analysis of the DBRS

A principal axis factor analysis of the DBRS with oblique rotation yielded three factors with eigenvalues greater than 1, accounting for 54% of variance. The loadings are presented in Table 2. Factor 1 is characterized by inattention, Factor 2 by hyperactivity, and Factor 3 by impulsivity. If the analysis is constrained to two factors, the impulsivity items (15–18) load with the hyperactivity items (10–14). For the remaining analyses, we constructed two scales, one for inattention and one for hyperactivity-impulsivity.

Descriptive Statistics

Mean inattention and hyperactivity-impulsivity scores by grade, using one twin randomly selected from each pair to avoid bias in variance, are given in Table 3. There was no significant difference between the mean ratings of inattention across kindergarten, Grade 1 and Grade 2. Hyperactivity-impulsivity levels decreased significantly from kindergarten to Grade 1 and from Grade 1 to Grade 2.

Correlations Between Inattention, Reading and Hyperactivity-Impulsivity

The phenotypic correlations in Table 4 between reading and inattention are small to moderate, though in the expected direction. If a child has high levels of inattention their reading score will be reduced. The correlations between hyperactivity-impulsivity and reading are small though significant at $p < .001$ due to the large sample size. However, the strength of the relationship between reading and symptoms of inattention is significantly stronger than between reading and hyperactivity-impulsivity, with p values $< .001$ for each grade based on z transformation for non-independent samples. Inattention and hyperactivity-impulsivity correlate moderately. Each of the relationships appears stable as the correlations are highly similar across the three grades.

Behavioural Genetic Modeling

Univariate analysis of reading, inattention and hyperactivity-impulsivity—The results of the univariate analysis are shown in Table 5. The fact that the MZ correlations for inattention were more than twice the magnitude of the DZ correlations warranted a model which allowed for dominance as well as additive genetic effects, with shared environment fixed to zero (Plomin et al., 2008). We interpret dominant and additive effects jointly as *broad-sense heritability* since the individual components show more bias than their sum (Coventry & Keller, 2005; Keller & Coventry, 2005). (Alternatively, the large differences between the MZ and DZ intraclass correlations for inattention could indicate a contrast effect in which parents overestimate the difference between members of DZ pairs [Rietveld et al., 2004]. To the extent that these are in fact contrast effects rather than dominance, our broad-sense estimates are overstated.)

The results indicate that genes have a substantial effect on children's traits of reading, inattention, and hyperactivity-impulsivity. Shared environment is not influential in the

expression of either inattention or hyperactivity-impulsivity, though it has a small effect on individual differences in reading ability in kindergarten. Unique environment, which includes measurement error, has a small-to-moderate effect on reading and hyperactivity-impulsivity, and a higher effect on inattention.

Longitudinal analyses of reading and ADHD symptoms—Table 6 contains estimates of genetic and environmental contributions to reading, inattention and hyperactivity-impulsivity across the three grades. To illustrate how to interpret the table, consider the genetic factor coefficients for reading. The loadings of Factor A₁ on the three measured variables of .77, .73, and .63 show that a single genetic factor affects all measures, indicating a common source of genetic variation. The proportion of the variance accounted for by this genetic source for each variable is estimated from the squares of the coefficients, .60, .53, and .40, respectively. Factors A₂, with loadings of .51 and .59 on reading in Grades 1 and 2 respectively, indicates that a second genetic source comes into play on top of the genes that are shared with kindergarten reading. The Factor A₃ value of .28 (.08 of variance) suggest a third genetic source for reading coming into play in Grade 2, though the fact that the confidence interval contains zero indicates that it may not be reliable. Common environment is influential in kindergarten only, with a moderate effect. Nonshared environment effects are primarily limited to each grade, consistent with the interpretation that they incorporate measurement error, which by definition is uncorrelated from one occasion to another.

Inattention was modeled as ADE, consistent with the results presented in Table 5. Additive genetic factors are not significant, though some of the values suggest small degrees of influence. Factor D₁ affects inattention in kindergarten and continues to exert an influence in Grades 1 and 2. There is some evidence of a second source of dominant genetic influence, factor D₂, emerging after kindergarten, with loadings of .41 and .44 in Grades 1 and 2, but it, too, may not be reliable given that the confidence intervals contain zero. There are three unique environmental factors that are influential, one is operative when a child is in Kindergarten (E₁), and its influence carries through to Grades 1 and 2, though showing only small effects of around .09 of the variance (.29² and .31²). An independent source accounts for differences in inattention in Grades 1 and 2 (E₂), and a third and separate source is influential when a child is in Grade 2. The pattern of most nonshared effects being new for each grade is also consistent with these effects being in part measurement error.

Hyperactivity shows a pattern similar to that of inattention; there are two significant genetic factors, one continuous throughout the three years and the other coming on stream in Grade 1. Shared and nonshared environment influences also mirrored those on inattention.

Multivariate analysis of inattention and reading across time—The previous analysis provided evidence of the degree to which genetic and environmental influences account for individual differences in the expression of reading, inattention and hyperactivity. In the analysis presented in Table 7, we interleaved reading and inattention into a single longitudinal analysis to examine how the two variables interact with each other across the same time span. We elected to model the genetic influence as additive in this analysis because it is not possible to include both additive (reading) and dominance (inattention) factors in a single analysis. We are thus treating A as representing broad-sense heritability in inattention, a strategy that would be especially appropriate if, as some suggest (Rietveld et al., 2004), the substantial differences in MZ and DZ correlations from parent ratings for ADHD represent a contrast effect rather than true dominance.

We include just the A (genetic) matrix—shared environment is a negligible overall influence, and nonshared environment, although exhibiting some overlap across occasions for inattention (Table 6), shows almost no overlap between inattention and reading. The results show that the

genetic overlap between inattention and reading all occurs in the “generalist” factor that affects both variables (A_1) across the three school years (negative loadings reflect the fact that higher scores on inattention tend to go with lower scores on reading). Put another way, the second genetic factors that come into play in Grade 1 for both reading (A_3) and inattention (A_4) do not affect the other variable—zero loadings of inattention in A_3 and reading in A_4 . This pattern continues to hold if inattention is entered into the model before reading; only in factor A_1 is there genetic overlap between reading and inattention (results not shown). Thus the genetics of the comorbidity between inattention and reading is in place from the first school year and does not change with development. The other feature of the results is that only part of the genetic influence on inattention in kindergarten is shared with reading; factor A_2 documents a genetic influence on inattention (loading of .49 out of a total genetic influence of .61 [.12 + .49]) that is independent of reading.

Genetic Examination of Causation

We compared the fit of the Cholesky model versus the two DOC models, one representing a causal effect on inattention on reading, the other representing a causal effect of reading on inattention. The results shown in Table 8 are the differences in fit of the Cholesky models against each DOC model separately, for each school grade. They indicate that in kindergarten and Grade 1 the Cholesky model provided a better fit for the data—the parameters of the best-fitting DOC models generated expected values that were more distant from the observed data than was the case with the best-fitting Cholesky model ($-2\Delta LL > 3.84$, $p < .05$). However, in Grade 2, the DOC model representing a causal effect on inattention on reading was not a poorer fit than the Cholesky ($-2\Delta LL < 3.84$). Despite this, the weight of the results, we suggest, are consistent with the proposition that the covariation between reading and inattention results from shared genes. We do note, however, that “measurement error greatly reduces the statistical power for resolving alternative causal hypotheses” (Gillespie, Zhu, Neale, Heath, & Martin, 2003, p. 385), so further caution in interpreting the analyses in Table 8 is called for.

Discussion

This twin study examined the developmental trajectories of reading, inattention, and hyperactivity across the first three school grades, kindergarten to Grade 2, in children in Australia, Norway, Sweden, and the USA. It also examined phenotypic and genetic associations between reading and inattention over this time span.

Factor analysis of the parent-completed DBRS ratings confirmed the partial independence of inattention and hyperactivity-impulsivity, consistent with other analyses of the dimensional structure of the ADHD complex (e.g., Hartman et al., 2001). We also observed that endorsement of inattention symptoms by parents remained, on average, constant across the three years we included. In contrast, mean hyperactivity-impulsivity levels declined, consistent with others’ observations (Hart et al., 1995; Smith, Barkley, & Shapiro, 2006). The convergence of our results with these previous studies supports the generalizeability of our findings despite the use of a twin sample, and provides additional evidence supporting the distinction between inattention and hyperactivity-impulsivity.

As we have shown previously for this sample, reading is substantially heritable in the early school years (Byrne et al., 2006, 2007, 2008; Samuelsson et al., 2008). This is the first analysis of reading development over three continuous school years, kindergarten to Grade 2, in our sample. It shows that a new genetic factor becomes operative in Grade 1 in addition to the genetic factor that is continuous through the three years. Previous work has indicated that this newly-emerging factor stems from the US and, particularly, the Scandinavian twins (Samuelsson, 2007, 2008). The Australian twins show higher heritability in kindergarten and the entry of no new genes in Grade 1. We have attributed the country differences to the fact

that reading instruction is the most intensive and extensive in Australian kindergartens of all the samples and hence recruits genetically-based cognitive and behavioral processes right from the start of school. In contrast, the kindergarten curricula in the US, and, particularly, Scandinavia, either do not devote as much time to reading instruction because school attendance is half-day (US) or largely avoid it in favor of an emphasis on social development (Scandinavia).

Inattention and hyperactivity-impulsivity were as heritable in this sample as they have been shown to be in others (Willcutt et al., 2005), with point estimates ranging from .56 to .79. Overlapping confidence intervals (Table 5) suggest that each variable is as heritable as the other, and that there are no reliable changes in heritability over these three years of school. But neither is genetically singular in that for both new genes emerge later in addition to those operating already at kindergarten, although the reliability of this new genetic factor in the case of inattention is uncertain.

As already stated, inattention showed the strongest relationship with reading, and consequently our final analyses were restricted to describing the inattention-reading relationship in more detail. In the first of these, we found that the genetic basis of the relationship between reading and inattention is in place already in the first school year, and that no new genetic source contributes to the relationship after that (at least until the end of Grade 2). In the second analysis, we attempted to illuminate further the causal relation between inattention and reading, pitting a common-gene account against two versions of a more direct causal connection between the two variables, one with inattention as the primary cause and one with reading as the primary cause. For the most part, the common-gene account prevailed, with a superior fit to the data of the Cholesky model compared to either of the two Direction-of-Causation models in kindergarten and Grade 1, though in Grade 2 the Cholesky and inattention-to-reading models provided equal fit (Table 8). In contrast to the influential early results described by Pennington et al. (1993), there was no support for the idea that low levels of reading themselves trigger an inattentive profile in children. To the contrary, if there is a direct causal relation it is inattention undermining reading development as children achieve higher levels of reading skill in Grade 2. It is known that the cognitive demands to read successfully begin to expand in Grade 2, with a wider-ranging vocabulary in reading material and more independence in decoding expected from the child (Freebody & Byrne, 1988; Byrne, Freebody, & Gates, 1992). One hypothesis worth investigating is that an inattentive child becomes vulnerable at that point because of a relative decline in the amount of print being processed. However, we wish to emphasize the speculative nature of this suggestion.

Limitations

Seen from a clinical perspective, one limitation of this study may be the use of a community sample and the consequent treatment of ADHD as a continuum. There may be aspects of ADHD pathology that cannot be captured within such a sample, notwithstanding our earlier arguments that clinical manifestations of the disorders are best thought of as extremes of normal distributions. Other limitations may be the differing ascertainment methods for the twins a—voluntary registry in Australia versus birth records in the other countries—and the use of payment in the US versus none elsewhere. We have attempted in part to address these factors and the mean differences in behavioral and achievement levels across countries by standardizing within country prior to genetic analyses. We also note that the US sample, the largest with about half the twins pairs, has produced mean scores on standardized tests such as the TOWRE that are very close to the national norms in terms of means and variances: In Grade 2, the sight-word and phonemic decoding components of the TOWRE had means (and standard deviations) of 101.97 (14.69) and 99.80 (13.07) respectively, with 100 (15) as the tests' norms (Byrne et al., 2009). The Australian sample tended to score about 2/3 *SD* above

the norm, though it is unclear if this reflects ascertainment or national differences. But at least the US sample does not appear to be unrepresentative on account of participant payments.

In combining the country samples we may have ignored possible country differences in the relations of inattention and literacy. This may apply particularly in kindergarten, where, as previously described, lower levels of heritability for reading hold in Scandinavia (Samuelsson et al., 2008). The size of the Norwegian and Swedish samples mean that separate country analyses would suffer from being underpowered, but future research could address this issue.

Educational Implications

It appears that the genetic basis for the co-occurrence of attentional and reading problems is in place early in schooling. In fact, as Willcutt, Betjemann, Wadsworth, et al. (2007) have shown, it is in place prior to schooling. Knowing this should forearm teachers to tailor their instruction to compensate for the attention-based difficulties. This in turn requires early diagnosis of these problems, a refinement in our understanding of the precise cognitive deficits that accompany them, and the development of adequate compensation strategies. Merely knowing that a child's attention may wander during reading lessons, however, should give teachers sufficient cause for concern, and motivate the search for ways to maintain attention. It should also alert teachers to the broader but related issue of motivation, a pervasive concern throughout education (Byrne, Khlentzos, Olson, & Samuelsson, in press). Thus, although the focus of much research into early literacy development has been on the cognitive and linguistic processes that are involved and on ways to compensate for deficiencies in them, the extensive literature on attentional and motivational processes in instruction is pertinent, and should be part of recipes for optimal instruction in early literacy.

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Cholesky decomposition model

Direction-of-causation (DOC) model

Figure 1.

Cholesky decomposition model and direction of causation (DOC) model, shown for one member of a twin pair. Latent variables are represented by circles, measured variables by rectangles. In the Cholesky model, ACE factors load on both inattention and reading as well as on reading. In the DOC model, ACE factors load independently on inattention and reading, with a path, i , that represents the causal effect of inattention on reading (or reading on inattention). For our analyses, the Cholesky and the DOC models for reading and inattention were compared separately for each of the three school grades.

Table 1

Child's Mean Age at Testing (Years) and Numbers by Zygosity and Sex within Each Country

	Age (SD)			
	Australia	United States	Norway	Sweden
Time				
Kindergarten	6.0 (0.4)	6.3 (0.3)	6.7 (0.3)	6.2 (0.2)
Grade 1	7.0 (0.4)	7.4 (0.3)	7.7 (0.3)	7.7 (0.4)
Grade 2	7.9 (0.4)	8.4 (0.3)	8.7 (0.3)	8.7 (0.3)
Zygosity & Sex				
MZ females	138	256	50	64
MZ males	164	194	36	84
DZ females	106	236	58	66
DZ males	120	292	60	54
Total	528	978	204	268

Table 2

Factor Analysis of Disruptive Behavior Rating Scale

Item	Factor 1	Factor 2	Factor 3
1. Fails to give close attention to detail	.67	.00	.06
2. Difficulty sustaining attention	.73	-.03	-.03
3. Does not seem to listen when spoken to	.50	.14	-.10
4. Does not follow through on instructions	.72	-.03	.00
5. Has difficulty organizing tasks	.74	.01	.06
6. Avoids tasks that require mental effort	.64	-.11	-.04
7. Loses things necessary for tasks	.54	.01	-.03
8. Is easily distracted	.62	.10	-.06
9. Is forgetful in daily activities	.61	.10	.00
10. Fidgets with hands or feet	.30	.13	-.34
11. Leaves seat in classroom	.20	-.03	-.52
12. Runs and climbs excessively	-.06	-.06	-.81
13. Has difficulty playing quietly	.05	.07	-.62
14. Often on the go, as if driven by a motor	-.02	.18	-.58
15. Talks excessively	-.05	.59	-.16
16. Blurts out answers before question is finished	-.04	.69	.02
17. Has difficulty waiting turn	.10	.61	-.07
18. Interrupts or intrudes on others	.06	.72	.07

Note. Bold values indicate highest factor loadings on each factor.

Table 3

Means (and Standard Deviations) for Behavioral Measures from Kindergarten to Grade 2.

Measure	Kindergarten	Grade 1	Grade 2
Inattention	4.86 (4.20)	5.02 (4.51)	5.12 (4.56)
Hyperactivity-Impulsivity	5.09 (4.50)*	4.56 (4.46)*	4.14 (4.28)*
Participants (<i>N</i>)	743	692	585

* Signifies a significant difference with the adjacent mean score at $p < .05$.

Table 4

Correlations Between Reading, Inattention and Hyperactivity-impulsivity in Kindergarten Grade 1 and Grade 2

	Inattention.	Hyperactivity-Impulsivity
Kindergarten		
Reading	-.24**	-.13**
Inattention	-	.58**
Grade 1		
Reading	-.31**	-.12**
Inattention	-	.56**
Grade 2		
Reading	-.28**	-.12**
Inattention	-	.56**

** *Note.* $p \leq .001$. One twin from each pair was randomly selected for these analyses to avoid bias due to within-pair correlations, hence *N*s are approximately half of the full samples at each grade. Sample sizes also depended on having both Inattention and Reading data for each child, with about 83% availability (see text). Kindergarten, $N = 743$; Grade 1, $N = 691$; Grade 2, $N = 585$.

Table 5

Univariate Estimates of Additive Genetic (A), Common Environment (C) and Unique Environment (E) Influential in ADHD Subtypes and Reading in Kindergarten

Measure	Correlations				
	MZ	DZ	a^2	c^2	e^2
Kindergarten					d^2
Reading	.87	.57	.61 (.46, .78)	.26 (.09, .16)	.13 (.09, .18)
Inattention*	.65	.16	.06 (.00, .50)	--	.33 (.28, .41)
Hyperactivity-Impulsivity	.77	.41	.79 (.62, .83)	.00 (.00, .16)	.20 (.17, .25)
Grade 1					
Reading	.83	.45	.86 (.68, .89)	.00 (.00, .18)	.14 (.11, .16)
Inattention*	.65	.14	.03 (.00, .48)	--	.30 (.25, .37)
Hyperactivity-Impulsivity	.76	.37	.73 (.54, .81)	.03 (.00, .22)	.23 (.19, .28)
Grade 2					
Reading	.84	.45	.87 (.73, .90)	.00 (.00, .14)	.13 (.10, .16)
Inattention*	.55	.18	.12 (.00, .56)	--	.44 (.36, .53)
Hyperactivity-Impulsivity	.74	.32	.74 (.61, .79)	.00 (.00, .11)	.26 (.21, .32)

Note. Broad-sense heritability = .66, .69, and .56 for inattention in kindergarten, Grade 1 and Grade 2, respectively.

Table 6

Cholesky Decomposition of Inattention, Reading and Hyperactivity-Impulsivity Separately Across Time

Reading			
	A ₁	A ₂	A ₃
Kindergarten	.77 (.68, .88)		
Grade 1	.73 (.62, .82)	.51 (.40, .58)	
Grade 2	.63 (.51, .73)	.59 (.49, .69)	.28 (.00, .39)
	C ₁	C ₂	C ₃
Kindergarten	.52 (.32, .64)		
Grade 1	.22 (.00, .40)	.20 (.00, .33)	
Grade 2	.20 (.00, .37)	.00 (.00, .25)	.00 (.00, .26)
	E ₁	E ₂	E ₃
Kindergarten	.36 (.33, .40)		
Grade 1	.14 (.10, .17)	.33 (.30, .37)	
Grade 2	.01 (.00, .14)	.17 (.14, .22)	.28 (.26, .32)
Inattention			
	A ₁	A ₂	A ₃
Kindergarten	.31 (.00, .71)		
Grade 1	.10 (.00, .61)	.22 (.00, .57)	
Grade 2	.02 (.00, .62)	.25 (.00, .56)	.00 (.00, .47)
	D ₁	D ₂	D ₃
Kindergarten	.75 (.39, .85)		
Grade 1	.68 (.33, .85)	.41 (.00, .60)	
Grade 2	.57 (.20, .78)	.44 (.00, .60)	.00 (.00, .37)
	E ₁	E ₂	E ₃
Kindergarten	.58 (.53, .64)		
Grade 1	.29 (.22, .35)	.47 (.43, .52)	
Grade 2	.31 (.24, .39)	.21 (.14, .27)	.53 (.49, .58)
Hyperactivity-Impulsivity			
	A ₁	A ₂	A ₃
Kindergarten	.88 (.78, .91)		
Grade 1	.67 (.55, .77)	.49 (.33, .62)	
Grade 2	.61 (.51, .70)	.43 (.25, .62)	.43 (.00, .53)
	C ₁	C ₂	C ₃
Kindergarten	.14 (.00, .42)		
Grade 1	.00 (.00, .49)	.28 (.00, .45)	
Grade 2	.00 (.00, .33)	.00 (.00, .32)	.00 (.00, .30)
	E ₁	E ₂	E ₃
Kindergarten	.45 (.41, .50)		
Grade 1	.14 (.10, .68)	.45 (.40, .49)	

Reading			
Grade 2	.17 (.10, .22)	.10 (.00, .17)	.46 (.42, .51)

Additive Genetic Factors from Cholesky Decomposition of Inattention and Reading Across 3 Time Points

Table 7

Measure	A ₁	A ₂	A ₃	A ₄	A ₅	A ₆
<i>Kindergarten</i>						
Reading	.73 (.64, .81)					
Inattention	-.35(.47, .22)	.70(.58, .78)				
<i>Grade 1</i>						
Reading	.69 (.59, .79)	.06 (-.06, .21)	.50 (.37, .59)			
Inattention	-.36 (-.49, -.22)	.49 (.34, .61)	-.09 (-.27, .06)	.52 (.39, .60)		
<i>Grade 2</i>						
Reading	.61 (.50, .71)	.06 (-.06, .21)	.58 (.46, .68)	.02 (-.11, .17)	.28(-.38, .38)	
Inattention	-.34 (-.48, -.19)	.36 (.18, .50)	-.09 (-.16, -.02)	.20 (.14, .27)	-.06(-.12, .00)	.53(.49, .58)

Table 8

Comparison of the Cholesky Model Fit with Each of the Direction of Causation Models at Each Grade

Time point	DOC Model	
	Inattention → Reading	Reading → Inattention
	$-2\Delta LL$	$-2\Delta LL$
Kindergarten	24.04*	21.75*
Grade 1	5.84*	3.87*
Grade 2	3.47	10.37*

Note: The comparison of the direction of causation models to the Cholesky results in one degree of freedom. The fit index used is minus twice the difference of the log likelihood ($-2\Delta LL$). critical ratio: $-2\Delta LL = 3.84$, $p = .05$, $N = 1978$.