A longitudinal twin study on the association between ADHD symptoms and reading

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Background: Attention deficit hyperactivity disorder (ADHD) and reading disability commonly co-occur because of shared genetic risk factors. However, the stability and change of these genetic influences and the predictive relationships underlying this association longitudinally remain unclear. Methods: ADHD symptoms and reading were assessed as continuous dimensions in a UK general population sample of approximately 7,000 twin pairs. Parent ratings of ADHD symptoms and teacher ratings of reading were obtained at two ages: middle childhood (ages 7–8 years) and early adolescence (ages 11–12 years). Cross-lagged quantitative genetic analyses were applied. Results: ADHD symptoms and reading significantly predicted each other over time. However, ADHD symptoms were a significantly stronger predictor of reading than vice versa. Inattentive and hyperactive-impulsive symptoms of ADHD both contributed to the prediction of reading, but inattentiveness was a significantly stronger predictor. Furthermore, ADHD symptoms and reading were highly heritable, and their association was primarily attributable to shared genetic influences. Despite notable genetic innovation for each trait, genetic factors involved in the association of ADHD symptoms and reading over time were highly stable. Conclusions: ADHD symptoms may put children at increased risk for reading problems and vice versa. Moreover, enduring genetic mechanisms appear to be important in the association of ADHD symptoms and reading over time. Keywords: ADHD, inattentiveness, hyperactivity-impulsivity, reading, longitudinal, twin, genetics.

Introduction

Children with attention deficit hyperactivity disorders (ADHD) often experience reading disability. ADHD and reading disability co-occur more often than expected by chance, and in clinical and non-clinical samples (Semrud-Clikeman et al., 1992; Willcutt & Pennington, 2000), which suggests that their co-occurrence is not because of a sampling artefact.

Twin studies have shown that ADHD symptoms and reading are highly heritable (Faraone et al., 2005; Olson, 2006) and analysis of cross-sectional twin data suggests that their association is largely attributable to genetic influences shared between reading and inattentive symptoms of ADHD (Greven, Harlaar, Dale, & Plomin, 2011; Paloyelis, Rijsdijk, Wood, Asherson, & Kuntsi, 2010; Willcutt, Pennington, Olson, & DeFries, 2007). However, although this cross-sectional evidence is highly consistent in demonstrating genetic overlap between ADHD symptoms and reading in samples including the childhood and adolescent ages, genetic influences on ADHD symptoms and reading are not perfectly stable (Harlaar, Dale, & Plomin, 2007; Kuntsi, Rijsdijk, Ronald, Asherson, & Plomin, 2005).

This gives rise to the possibility that different genetic influences may be involved in genetic overlap between ADHD symptoms and reading over time. This is an important issue as it addresses whether the aetiologies of this association are stable or differ over time.

To date, only one twin study has examined the stability of genetic and environmental influences on the association of inattentiveness and reading (Ebejer et al., 2010). Based on a population-based sample of 989 twin pairs followed across ages 6–9, the study concluded that the genes involved in the association between inattentiveness and reading at study onset did not change with development. In the present study, we aim to replicate this finding in a substantially larger sample (around 7,000 twin pairs), as well as to extend this finding to a period spanning middle childhood (ages 7–8) to early adolescence (ages 11–12).

The present study also sets out to examine the direction of effects underlying ADHD symptoms and reading over this period. It has previously been suggested that ADHD symptoms may lead to the development of reading disability, possibly because attention deficits interfere with reading instruction (e.g. Daily, 2006). On the other hand, reading disability may lead to the development of ADHD symptoms, possibly as a result of the academic failure experienced by the child (e.g. Cunningham & Barkley, 1978).

Longitudinal studies of phenotypic (observed) data across childhood and adolescence have provided...
evidence for both directions of effects. For example, ADHD symptoms have been shown to predict later reading disability (e.g. Dally, 2006; McGee, Prior, Williams, Smart, & Sanson, 2002; Rabiner & Cole, 2000), and some evidence also suggests that reading disability predicts later ADHD symptoms (e.g. McGee et al., 2002; Rabiner, Cole & CPPRG, 2000).

An early influential study compared the neuropsychological profiles of children from three groups (ADHD only, reading disability only and both conditions) and found that the neuropsychological profile of children from the comorbid group resembled the profile of children with reading disability only (Pennington, Groisser, & Welsh, 1993). Based on this, the study hypothesized that reading disability might be the primary disorder, which causes secondary symptoms of ADHD without the aetiology typically associated with ADHD. However, later neuropsychological studies using the same design did not replicate these findings (e.g. Reader, Harris, Schuerholz, & Denckla, 1994; Willcutt et al., 2001). Instead, several cognitive deficits, including processing and naming speed, were later shown to be shared between the disorders (e.g. Willcutt et al., 2010), in line with a common aetiology model.

Twin research has also helped address whether ADHD symptoms and reading may influence the development of each other. For example, a genetic association has been shown to exist already in preschool children between inattentive symptoms and prereading skills (Willcutt et al., 2007), which suggests that the association between ADHD symptoms and reading is not attributable, at least not exclusively, to interference with formal reading instruction or academic frustration during the school years. Moreover, the longitudinal twin study of ADHD symptoms and reading mentioned before (Ebejer et al., 2010) concluded that a common genes account best explains the relations between inattentiveness and reading at the end of kindergarten (around ages 6–7) and first grade (ages 7–8); however, from second grade (ages 8–9 years) inattentiveness may begin to play a more direct causal role in reading ability, possibly because cognitive demands to read successfully begin to expand around that age.

The present study

The present study extends previous research by examining, for the first time, the longitudinal relations between ADHD symptoms and reading from middle childhood (ages 7–8) to early adolescence (ages 11–12) in a quantitative genetic cross-lagged model. This model is well suited for examining the issues raised above, as it allows the examination of direction of causation and common aetiology hypotheses in a single model. Data came from an unselected population-representative sample of around 7,000 twin pairs from the United Kingdom.

Thus, rather than focusing on the clinical extreme, the present study examines the full range of the normal distribution of ADHD symptoms and reading ability.

Aims and hypotheses

The first aim was to explore the direction underlying the associations between ADHD symptoms and reading from middle childhood to early adolescence to test for predictive, potentially causal, relations. We expected bidirectional effects between ADHD symptoms and reading.

The second aim was to examine the genetic and environmental aetiologies of the association between ADHD symptoms and reading across this period. We hypothesized that there would be evidence for partial genetic overlap between ADHD symptoms and reading, and that many of the same genes would be involved in this association over time.

Finally, given the stronger phenotypic and genetic association of reading with inattentive than hyperactive-impulsive ADHD symptoms (Greven, Harlaar, Dale & Plomin, 2011; Willcutt & Pennington, 2000), we addressed aims one and two separately for inattentiveness and hyperactivity-impulsivity. We hypothesized that the longitudinal relationship with reading would be stronger for inattentiveness than hyperactivity-impulsivity.

Method

Participants and procedure

Data came from the Twins Early Development Study, a UK population-representative sample of twins born in England and Wales between 1994 and 1996 (Oliver & Plomin, 2007). Zygosity of twins was initially based on physical similarity ratings and later verified using genotyping (Freeman et al., 1997; Oliver & Plomin, 2007). Families were excluded from the present study following severe prenatal or perinatal complications or if one or both twins suffered from a severe medical condition (e.g. autism, Down’s syndrome). Uncertain sex or zygosity and failure to provide information at recruitment were also grounds for excluding families. After exclusions, a total of 1,346 monozygotic (MZ) male, 1,299 dizygotic (DZ) same-sex male, 1,503 MZ female, 1,360 DZ same-sex female and 2,626 DZ opposite-sex twin pairs were included in the model-fitting analyses.

ADHD symptoms and reading were assessed based on postal questionnaires, returned via postage-free envelope. Written consent, including consent to contact the twins’ teachers, was obtained from parents at each assessment.

ADHD symptoms

ADHD symptoms (i.e. a total score of inattentive and hyperactivity-impulsive symptoms), inattentiveness and hyperactivity-impulsivity were assessed in middle childhood (mean age = 7.87 years, SD = .51) and early
adolescence (mean age = 11.34 years, \(SD = .65\)). Ratings were made using the DSM-IV-based ADHD items of the Revised Conners’ Parent Rating Scale, which assesses hyperactivity-impulsivity and inattentiveness with nine items each (Conners, Sitarenios, Parker, & Epstein, 1998). Parents rated each child’s behaviour on a 4-point Likert scale from (0) ‘not true at all’ to (3) ‘very much true’. Cronbach’s alphas were high: .91/.91 for ADHD symptoms, .89/.90 for inattentiveness and .84/.83 for hyperactivity-impulsivity in middle childhood and early adolescence, respectively.

**Reading**

Reading ability was assessed in middle childhood (mean age = 7.20, \(SD = .28\)) and early adolescence (mean age = 11.54, \(SD = .66\)) based on UK National Curriculum criteria (National Curriculum, 2004). The National Curriculum specifies learning goals for 5- to 16-year-old children at state-funded schools in England and Wales. Teachers rated each child according to these learning goals, based on their knowledge of the child’s performance over the past academic year (for further details, see Chamorro-Premuzic, Harlaar, Greven, & Plomin, 2010; Kovas, Haworth, Dale, & Plomin, 2007). The National Curriculum teacher ratings demonstrate substantial agreement with reading tests (e.g. Dale, Harlaar, & Plomin, 2005).

**The twin method**

The twin method capitalizes on the fact that similarities between children raised together can be attributed to shared genetic or shared environmental factors. Genetic sharing is 100% for MZ (identical) twins, who share 100% of their inherited DNA sequence, but DZ (fraternal) twins on average only share 50% of their additive genes, like any other siblings. Shared environmental influences are 100% similar for MZ and DZ twins, and make children in the same family more alike. In contrast, nonshared environmental influences, which contribute to differences between children in the same family, are specific to each child. Based on this, structural equation models of the expected covariation between twins can be constructed, which can be fitted to the observed data using maximum-likelihood iterative methods. This makes it possible to estimate the relative contributions of genetic heritability \(A\), shared environmental \(C\) and nonshared environmental \(E\) influences to individual differences in ADHD symptoms and reading and to the covariation between them.

**The cross-lagged twin model**

At the centre of the quantitative genetic cross-lagged model (first described by Burt, McGue, Krueger, & Iacono, 2005) are the stability paths \(b_{11}, b_{22}\), which connect the same traits across time, and the cross-lagged paths \(b_{12}, b_{21}\), which connect different traits across time (Figure 1). The stability and cross-lagged paths take the form of partial regression coefficients, which take into account the preexisting association between ADHD symptoms and reading in middle childhood, as well as controlling for stability or cross-lagged effects. Figure 1 also depicts genetic \((a_1–a_4)\), shared environmental \((c_1–c_4)\) and nonshared environmental \((e_1–e_4)\) contributions to ADHD symptoms and reading. Nonshared environmental contributions include measurement error. Lastly, Figure 1 presents genetic and environmental correlations \((r_{A1}, r_{A2}, r_{C1}, r_{C2}, r_{E1}, r_{E2})\). These can range from 1 to 1, and represent the extent to which genetic or environmental influences on the measures are shared.

**Analyses**

Standard regression-based corrections for sex and age were applied to raw scores (McGue & Bouchard, 1984) and residual scores were analysed. The ADHD measures were positively skewed and transformed using the command ‘lnskew0’ in STATA (StataCorp. Statistical Software: Release 9.0. Stata Corporation; College Station, TX: 2005). Reading was normally distributed. Full-information maximum likelihood estimation was used, which allows the inclusion of missing data with minimized bias (Finkbeiner, 1979). The significance of estimates was assessed using likelihood-based 95% confidence intervals (the inclusion of zero indicates nonsignificance). For the cross-lagged paths, significance was confirmed by fixing one cross-lagged path at a time to zero and assessing the drop in model fit using a chi-squared test (conclusions from confidence intervals were confirmed, although these chi-squared tests are not presented due to space limitations). Models were fitted to the data in Mx (Neale, Boker, Xie, & Maes, 2006).

Descriptive statistics, twin and cross-twin cross-trait correlations are available from previous publications based on the same data (Greven, Rijsdijk, & Plomin, 2011; Harlaar, Kovas, Petrill, Dale, & Plomin, 2011; Harlaar et al., 2007; McLoughlin, Ronald, Kuntsi, Asherson, & Plomin, 2007). In line with these publications, we modelled shared environmental influences and variance sex differences (the latter only for ADHD symptoms); however, these publications provided no...
evidence for qualitative or quantitative sex differences, or sibling interaction.

Results
Phenotypic correlations
Phenotypic across-trait correlations between ADHD symptoms and reading ranged from -.23 to -.25, and were of similar magnitude within age and across age (Table 1). These correlations were negative because the measures were scored so that high scores refer to high ADHD symptoms and high reading ability. Phenotypic within-trait across-age correlations were .67 for ADHD symptoms and .48 for reading.

Model fit
The cross-lagged model of ADHD symptoms and reading (Figure 2) fitted the data well based on fit indices which heavily reward parsimony, as indicated by the negative values on the Bayesian information criterion (BIC = -586.18) and the deviance information criterion (DIC = 413.42). However, based on the significant chi-squared and positive Akaike’s information criterion (AIC), the model provided inadequate fit compared with the baseline saturated model \( \chi^2(188) = 520.36, p < .001; \)\( \text{AIC} = 144.36 \). Poor fit for these statistics is common for multivariate modelling in large samples.

Phenotypic cross-lagged model results
The stability paths pertaining to ADHD symptoms \((b_{11} = .66)\) and reading \((b_{22} = .46)\) were high and significant (Figure 2). Moreover, the cross-lagged paths from ADHD symptoms to reading \((b_{12} = -.12)\) and from reading to ADHD symptoms \((b_{21} = -.07)\) were negative and small, albeit significant. Of note, the cross-lagged path from ADHD symptoms to reading was significantly larger than the reverse path [confidence intervals did not overlap, and equating these paths resulted in a significant chi-squared difference test; \( \chi^2(1) = 58.80, p < .001 \)]. This suggests that ADHD symptoms are a somewhat, albeit significantly, stronger predictor of reading than vice versa.

Genetic and environmental cross-lagged model results
Heritabilities in middle childhood were high for ADHD symptoms \((a_1^2 = .71)\) and reading \((a_2^2 = .68;\) Figure 2). The genetic correlation between ADHD symptoms and reading in middle childhood was modest \((r_{A1} = -.30)\), which suggests that ADHD

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Table 1 Phenotypic correlations between attention deficit hyperactivity disorder (ADHD) symptoms and reading

<table>
<thead>
<tr>
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<th>Middle childhood</th>
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<tr>
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<td>ADHD symptoms</td>
<td>Reading</td>
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<tr>
<td>Middle childhood</td>
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<td>-.25 (-.26/-23)</td>
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<td>ADHD symptoms</td>
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<td>Reading</td>
<td>-.23 (-.25/-22)</td>
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<td>-.25 (-.27/-24)</td>
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Ninety-five per cent confidence intervals are provided within parentheses.
ADHD, attention deficit hyperactivity disorder; \( r_p \), phenotypic correlation; 95% confidence intervals are provided within parentheses.

Proportions of the phenotypic correlation due to total genetic or environmental influences (i.e. transmitted plus age-specific effects).

Proportions of the phenotypic correlation due to newly emerging genetic or environmental influences specific to early adolescence.

Table 2: Proportions of the phenotypic correlations between ADHD symptoms and reading due to genetic (A), shared environmental (C) and nonshared environmental (E) influences

<table>
<thead>
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<th>Middle childhood</th>
<th>Early adolescence</th>
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<tbody>
<tr>
<td>( r_p )</td>
<td>-.25 (-.26/-23)</td>
<td>-.25 (-.27/-24)</td>
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<tr>
<td>% of ( r_p ) due to</td>
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<tr>
<td>A</td>
<td>.85 (.72/.98)</td>
<td>.68 (.58/.78)*</td>
</tr>
<tr>
<td>C</td>
<td>.08 (-.04/.20)</td>
<td>.18 (.09/.27)*</td>
</tr>
<tr>
<td>E</td>
<td>.07 (.04/.10)</td>
<td>.14 (.11/.17)*</td>
</tr>
</tbody>
</table>

Cross-lagged model results separately for inattentive and hyperactive-impulsive ADHD symptoms

The cross-lagged model was then fitted separately to inattentiveness and reading, and hyperactivity-impulsivity and reading in two bivariate models (see Tables S3–S6; Figures S1 and S2). As expected, phenotypic correlations with reading were significantly higher for inattentiveness (range from -0.25 to -0.29) than hyperactivity-impulsivity (-0.15 to -0.17; Table S3).
ness to reading, and from hyperactivity-impulsivity to reading were significantly larger than the reverse paths [chi-squared difference test equating cross-lagged paths in Figure S1: \( \chi^2(1) = 58.58, p < .001 \); chi-squared difference test equating cross-lagged paths in Figure S2: \( \chi^2(1) = 26.08, p < .001 \). This suggests that predictive relations were somewhat stronger in the direction from the ADHD symptom dimensions to reading. Notably, the cross-lagged path from inattentiveness to reading (standardized path estimate of \(-.12\)) was significantly larger than the cross-lagged path from hyperactivity-impulsivity to reading \((-0.08\); confidence intervals did not overlap). This suggests that compared with hyperactivity-impulsivity, inattentiveness was a stronger predictor of reading.

Heritabilities were high for inattentiveness (.78, .72) and hyperactivity-impulsivity (.73, .67) in middle childhood and early adolescence, respectively (Figures S1 and S2). At each age, reading showed significantly higher genetic correlations with inattentiveness \((-0.42, -0.31\) than with hyperactivity-impulsivity \((-0.05, -0.13\); Figures S1 and S2). For hyperactivity-impulsivity, the phenotypic correlations with reading were substantially mediated by shared environment (76% and 50% due to shared environment; Table S6). Phenotypic correlations between inattentiveness and reading were largely mediated genetically and negligibly by shared environment.

Of the 76% of the phenotypic correlation between inattentiveness and reading in early adolescence due to genetic influences, a nonsignificant proportion of only 12% was due to newly emerging genetic influences specific to adolescence, whereas the remaining 64% was due to enduring genetic influences transmitted from middle childhood (Tables S4 and S6). For hyperactivity-impulsivity, of the 46% due to genes, the majority of this proportion was due to transmitted genetic effects (Tables S5 and S6). Thus, genes involved in the association of reading with inattentiveness and hyperactivity-impulsivity were highly stable.

Lastly, a trivariate cross-lagged model of inattentiveness, hyperactivity-impulsivity and reading was fitted to the data (Figure S3). Analyses revealed that the cross-lagged path from hyperactivity-impulsivity to reading was significant in the trivariate model (standardized path estimate of \(-.03\); Figure S3). This suggests that hyperactivity-impulsivity predicts reading even after accounting for the strong phenotypic correlation between hyperactivity-impulsivity and inattentiveness in middle childhood (.60; Table S3). Likewise, inattentiveness predicted reading independently of hyperactivity-impulsivity (standardized path estimate of \(-.10\); Figure S3). Of note, our bivariate analyses (Figures S1 and S2) were also important given the debate as to whether inattentiveness co-occurring with hyperactivity-impulsivity may be unrelated to inattentiveness without hyperactivity-impulsivity (Milich, Balentine, & Lynam, 2001).

**Discussion**

ADHD symptoms and reading showed reciprocal relationships across middle childhood to early adolescence. However, ADHD symptoms were a significantly stronger predictor of reading than vice versa. Both inattentiveness and hyperactivity-impulsivity contributed to the prediction of reading, although inattentiveness was a significantly stronger predictor of reading.

ADHD symptoms and reading were highly heritable and yielded no sex differences in their genetic and environmental aetiologies as shown previously for these data (Greven, Rijsdijk, & Plomin, 2011; Harlaar et al., 2007). The association between ADHD symptoms and reading was largely attributable to shared genetic influences. ADHD symptoms and reading each demonstrated notable genetic innovation; however, the genes involved in their association over time were highly stable. This is consistent with a previous study (Ebejer et al., 2010), although we extend the result from middle childhood to early adolescence. As predicted, reading showed significantly stronger genetic overlap with inattentiveness than hyperactivity-impulsivity. Genetics was largely responsible for associations between inattentiveness and reading, whereas genetic as well as shared environmental influences were important in explaining associations between hyperactivity-impulsivity and reading.

**Limitations**

One possible weakness of this study is its use of continuous ratings of ADHD symptoms in a population-based sample rather than focusing on the clinical extreme. However, the use of an unselected sample is also a strength as it facilitated the collection of data from a large sample, thereby increasing power, and helped avoid possible biases associated with clinical samples.

Furthermore, effect sizes of the cross-lagged paths were small. However, small cross-lagged paths are the rule rather than exception (Burt et al., 2005; Gregory, Rijsdijk, Lau, Dahl, & Eley, 2009; Greven, Asherson, Rijsdijk, & Plomin, 2011; Hallett, Ronald, Rijsdijk, & Happe, 2010), and reflect the fact that the cross-lagged model is conservative, as cross-lagged paths are partial regression coefficients. The full predictive relationship between ADHD symptoms and reading is stronger, as indicated by the phenotypic correlations.

Moreover, to move from correlation to causation in cross-lagged analyses, synchronicity is required, that is, traits should be measured at the same time (Kenny, 1975). However, reading was assessed earlier than ADHD symptoms in middle childhood (7.2
vs. 7.9 years), and somewhat later than ADHD symptoms in early adolescence (11.5 vs. 11.3 years). To explore this possible bias, analyses in Figures 2 and S1–S3 were rerun after replacing the National Curriculum teacher ratings at age 7 with National Curriculum teacher ratings made at age 9 (mean child age = 9.04 years, SD = .29), nearly 2 years after ADHD symptoms were assessed. Results (not shown) supported all conclusions drawn in the present article.

An additional limitation is that the aetiologies underlying the cross-lagged effects are unclear, as cross-lagged paths are merely weighted by genetic and environmental influences (Luo, Haworth, & McGuffin, 2008) and of the cross-lagged model (Kenny, 1975), and the presence of statistically significant, albeit small, attrition biases linked to the twin method (Plomin, DeFries, McClearn, & Ploomin, 2010). For example, the cross-lagged path from ADHD symptoms to reading was attributable to genes and environments, exactly to the extent that ADHD symptoms in middle childhood were attributable to genes and environments.

Finally, limitations include standard limitations of the twin method (Plomin, DeFries, McClearn, & McGuffin, 2008) and of the cross-lagged model (Kenny, 1975), and the presence of statistically significant, albeit small, attrition biases linked to the presence of ADHD symptoms (Greven, Rijsdijk & Ploomin, 2011).

**Implications**

The significant and negative cross-lagged relations between ADHD symptoms and reading suggest that ADHD symptoms may put children at increased risk for reading problems, and vice versa. However, small effect sizes of cross-lagged paths suggest that the clinical implications of cross-lagged effects may be limited, at least for the developmental period under investigation. This is of interest as it suggests that although ADHD symptoms (especially inattentiveness) and reading significantly explain variance in each other over time, any predictive, and potentially causal, relations appear to be small.

Another result worth highlighting is that the association between hyperactivity-impulsivity and reading was substantially mediated by shared environment. This result replicates a previous finding based on reading assessed by tests rather than teacher ratings (Greven, Harlaar, Dale & Ploomin, 2011). An obvious target for this shared environmental mediation is the environments children share at home and school. Importantly, however, associations between hyperactivity-impulsivity and reading were only modest. Furthermore, additional evidence for this shared environmental mediation is needed, which may be complicated by several issues concerning shared environmental influences on ADHD (discussed in Burt, 2010).

A final implication concerns the finding that the genes involved in the association of ADHD symptoms and reading were highly stable across middle childhood to early adolescence. This was in spite of the relatively long follow-up into early adolescence and the large sample and hence increased power to detect newly emerging genetic influences. Moreover, this is by no means a default finding; for example, newly emerging genetic influences have been shown to be almost as important as stable genes in the association of inattentiveness and hyperactivity-impulsivity over time Greven, Asherson, Rijsdijk, & Ploomin (2011). Findings from the present study suggest that enduring molecular, and hence neurobiological, processes may be important for the association of ADHD symptoms and reading across childhood development. The exploration of these processes, for example through molecular genetic and neuropsychological studies, will be an interesting area for future research. Lastly, although speculative, our finding that the aetiologies of this association are largely the same in childhood and early adolescence, may suggest that similar treatments may be effective in alleviating this association across development.

**Conclusion**

ADHD symptoms and reading demonstrated reciprocal relationships, although all effects were small. Furthermore, genetic influences involved in the association of ADHD symptoms and reading were highly stable.

**Supporting information**

Additional Supporting Information may be found in the online version of this article:  
**Figure S1** Bivariate cross-lagged twin model of inattentiveness and reading  
**Figure S2** Bivariate cross-lagged twin model of hyperactivity-impulsivity (hyp-imp) and reading  
**Figure S3** Trivariate cross-lagged twin model of inattentiveness, hyperactivity-impulsivity (hyp-imp) and reading  
**Table S1** Transmission of genetic (A), shared environmental (C) and nonshared environmental (E) influences from middle childhood (MC) to early adolescence  
**Table S2** Genetic and environmental results pertaining to residual variance in attention deficit hyperactivity disorder symptoms and reading  
**Table S3** Phenotypic correlations between inattentiveness, hyperactivity-impulsivity (hyp-imp) and reading  
**Table S4** Genetic and environmental results pertaining to residual variance in inattentiveness and reading  
**Table S5** Genetic and environmental results pertaining to residual variance in hyperactivity-impulsivity (hyp-imp) and reading  
**Table S6** Proportions of the phenotypic correlations between the ADHD symptom dimensions and reading due to genetic (A), shared environmental (C) and nonshared environmental (E) influences

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Key points
- Associations between ADHD symptoms and reading have been attributed to shared genetic influences. The present study examined the stability and change of these genetic influences and the predictive relations underlying this association from middle childhood to early adolescence.
- ADHD symptoms and reading significantly predicted each other over time, although ADHD symptoms were a significantly stronger predictor of reading than vice versa.
- This suggests that ADHD symptoms may put children at increased risk for reading problems and vice versa, although all predictive relations were of small effect.
- Genetic influences involved in the association between ADHD symptoms and reading were highly stable over time.
- Thus, enduring molecular and hence neurobiological processes may be responsible for this association.

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