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A Novel Approach to Genetic and Environmental Analysis of Cross-Lagged Associations Over Time: The Cross-Lagged Relationship Between Self-Perceived Abilities and School Achievement is Mediated by Genes as Well as the Environment

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Abstract

Using longitudinal cross-lagged analysis to infer causal directions of reciprocal effects is one of the most important tools in the developmental armamentarium. The strength of these analyses can be enhanced by analyzing the genetic and environmental aetiology underlying cross-lagged relationships, for which we present a novel approach here. Our approach is based on standard Cholesky decomposition. Standardized path coefficients are employed to assess genetic and environmental contributions to cross-lagged associations. We indicate how our model differs importantly from another approach that does not in fact analyze genetic and environmental contributions to cross-lagged associations. As an illustration, we apply our approach to the analysis of the cross-lagged relationships between self-perceived abilities and school achievement from age 9 to age 12. Self-perceived abilities of 3852 pairs of twins from the UK Twins Early Development Study were assessed using a self-report scale. School achievement was assessed by teachers based on UK National Curriculum criteria. The key cross-lagged association between self-perceived abilities at age 9 and school achievement at age 12 was mediated by genetic influences (28%) as well as shared (55%) and non-shared (16%) environment. The reverse cross-lagged association from school achievement at 9 to self-perceived abilities at 12 was primarily genetically mediated (73%). Unlike the approach to cross-lagged genetic analysis used in recent research, our approach assesses genetic and environmental contributions to cross-lagged associations per se. We discuss implications of finding that genetic factors contribute to the cross-lag between self-perceived abilities at age 9 and school achievement at age 12.

Keywords

cross-lagged association; self-perceived abilities; school achievement

Among the vast body of research focused on identifying causal pathways (e.g., Rutter et al., 2001; Shadish et al., 2001), longitudinal analysis uses time to unravel cause from effect in developmental analyses because time flows in only one direction. However, correlations, even across time, are not necessarily causal. For example, the correlation between X at age 1 and Y at age 2 does not necessarily indicate that X causes Y because the correlation might

be mediated by other factors, most notably Y at age 1. When both X and Y are assessed at both ages, the effect of X at age 1 on Y at age 2 can be estimated independent of Y at age 1, which is the essence of cross-lagged analyses (Kenny, 1975). In this paper, we consider the cross-lagged relationship between children's self-perceived abilities and their school achievement from age 9 to age 12 in order to ask the extent to which self-perceived abilities affect or reflect school achievement.

Quantitative genetic theory and methods attempt to untangle cause and effect at the level of genetic and environmental aetiology (Plomin et al., 2008). One of the most important advances in recent decades has been multivariate genetic analysis. Rather than analyzing the variance of a single variable, multivariate genetic analysis analyzes the covariance between variables, including longitudinal analyses of covariance across time. Applying multivariate genetic analysis to cross-lagged analysis makes it possible to estimate the extent to which the effect of X at Age 1 on Y at Age 2 is mediated genetically or environmentally. Because there are already hundreds of multivariate genetic studies and scores of longitudinal genetic studies, what is new is the application of quantitative genetic analysis specifically to cross-lagged relationships.

In this paper, we present a novel approach that applies quantitative genetic analysis to cross-lagged relationships per se and apply this approach to the cross-lagged relationships between self-perceived abilities and school achievement for the purpose of illustration. Our approach is a refinement of the first attempt along these lines (Neiderhiser et al., 1999). However, subsequent research in this area has used a different approach that incorporates a phenotypic analysis of cross-lagged relationships within a multivariate/longitudinal genetic analysis (Burt et al., 2005; Forsman et al., 2010; Larsson et al., 2008). In our view, assessing the genetic and environmental aetiology of cross-lagged relationships is a key issue for cross-lagged genetic analysis.

In recent years interest has grown in self-perceived abilities — how good people *think* they are — to predict school achievement beyond 'actual' abilities (Marsh & Craven, 1997). In a phenotypic cross-lagged latent variable analysis using the present sample, bidirectional effects were found in that both self-perceived abilities and school achievement at age 9 affected school achievement and self-perceived abilities at age 12 controlling for general cognitive abilities (Charmorro-Premuzic et al., 2010). Although part of the interest in self-perceived abilities comes from an assumption that they are environmental in origin (Wigfield & Eccles, 2000), the first twin study of self-perceived abilities yielded a heritability estimate of about 0.50 in middle childhood (Greven et al., 2009). The same report also presented a multivariate genetic analysis that showed that self-perceived abilities, independent of general cognitive abilities, predicted school achievement at the same age for genetic rather than environmental reasons. Here we apply our new approach to cross-lagged genetic analysis in order to investigate the genetic and environmental aetiologies of the bidirectional relationships between self-perceived abilities and school achievement from age 9 to age 12. The previous phenotypic cross-lagged analysis indicated that we would find bidirectional effects, and the previous multivariate genetic analysis predicted that we might find genetic influence on the cross-lag between self-perceived abilities at age 9 and school achievement at age 12.

Materials and Methods

Sample

The participants in this study were sampled from the Twins Early Development Study (TEDS), a study of twins born in England and Wales between 1994 and 1996 (Oliver & Plomin, 2007). TEDS is reasonably representative of the general population in terms of

parental education, ethnicity and employment status (Kovas et al., 2007). Zygosity was assessed through a parent questionnaire of physical similarity, which is over 95% accurate when compared to DNA testing (Price et al., 2000). For cases where zygosity was unclear, DNA testing was conducted. Ethical approval for TEDS has been provided by the King's College London ethics committee (reference: 05/Q0706/228).

At age 9, TEDS families from the 1994 and 1995 cohorts were invited to participate in the study. At age 12, twins born in 1994, 1995 and 1996 were invited to participate. As described below, self-perceived abilities were reported by children themselves and school achievement was assessed by their teachers. At 9 years, 6770 child booklets were returned complete (83%), and 5836 teacher rating forms were returned complete (76%). At 12 years, 11708 child booklets (69%) and 9905 teacher forms were returned complete (78%). For each assessment we obtained informed consent from the parents of the twins. The mean age at each assessment was 9.04 ($SD = .29$) and 11.54 ($SD = .66$). Not all teachers provided National Curriculum levels, and for some twin pairs we only received a completed form for one member of the pair. In Tables 1 and 2 we present the sample sizes for the measures at each age. Given the complexity of including opposite-sex dizygotic (DZ) twins in multivariate genetic model-fitting (Neale et al., 2006a), our study was based on monozygotic (MZ) and same-sex DZ twins only.

Measures

Self-perceived abilities—Children's self-perceived abilities (SPA) with respect to English, mathematics, and science were assessed using the 9-item Perceived Abilities in School Scale (PASS) (Spinath et al., 2006). The PASS items were formulated to correspond to activities listed in the UK National Curriculum for English, mathematics and science, for instance, 'How good do you think you are at reading?' Responses were made on a 5-point Likert scale, with 1 indicating that the respondents believed they were *Not at all good* at the activity in question, and 5 indicating that they believed they were *Very good* at that activity. Factor analysis of the PASS clearly revealed three factors that corresponded to SPA in English, mathematics and science, and all internal consistencies fell in the moderate range (Spinath et al., 2006).

School achievement—The participants' school achievement in English, mathematics, and science was assessed using teachers' ratings based on UK National Curriculum (NC) criteria, which are uniform assessment guidelines followed by all teachers within the UK school system. Teachers were contacted in the second half of the school year so that they would be familiar with the children's performance. At age 9, the NC Teacher Assessments at Key Stage 2 were used, which are designed for children aged 8–11 years old. At age 12, the NC Teacher Assessments at Key Stage 3 were used, which are designed for children aged 11–14 years old. Teacher-rated achievement ranges from Levels 1 to 8. By the end of Key Stage 2 (age 11), children are expected to reach level 4; and by the end of Key Stage 3 (age 14), children are expected to reach level 5/6. These National Curriculum assessments have been shown to be valid measures of academic achievement (Kovas et al., 2007). Further information about National Curriculum is available at <http://curriculum.qca.org.uk>.

Analyses

The twin method investigates genetic and environmental influences on individual differences (variance) in observed traits. By comparing twin intra-class correlations for monozygotic (MZ) and dizygotic (DZ) twin pairs it is possible to estimate genetic effects, shared environmental effects (which make twins growing up in the same family more similar) and nonshared environmental effects (which do not contribute to familial resemblance). A more comprehensive way of estimating these effects is maximum

likelihood model fitting analysis (Neale et al., 2006 b). Mx software for structural equation modeling was used for standard analyses using raw data (Neale et al., 2006b).

Before genetic analysis, phenotypic correlations of variables were examined to determine whether they were of sufficient magnitude to be included in genetic analyses. Genetic and environmental influences on associations between measures are limited by the size of the phenotypic correlation; that is genetic and environmental influences on the covariance can be present if the two measures covary at least moderately.

The Genetic Cross-Lagged Model—After phenotypic correlation was established, genetic and environmental influences on cross-lagged associations were examined using multivariate genetic model-fitting method. The approach, as an extension of the pioneering work of Neiderhiser et al. (1999), is based on Cholesky decomposition (Loehlin, 1996; Neale & Cardon, 1992). Instead of controlling for contemporaneous correlations at both times as the earlier model did (Neiderhiser et al., 1999), which could lead to overcorrection, only the correlation at the prior age is controlled in the present model. Another advantage of our approach is the adoption of standard Cholesky decomposition, which is more practicable and easier to interpret. However, as discussed later, it should be noted that the cross-lagged models described here and by Neiderhiser et al. (1999) are limited in the same way, in that both cross-lagged paths cannot be estimated in the same model. Figure 1(a, b, c) represents the model that focuses on the cross-lagged association between self-perceived abilities at 9 and school achievement at 12. The right side of Figure 1(d, e, f) focuses on the cross-lagged association between school achievement at 9 and self-perceived abilities at 12.

The genetic cross-lagged model has three components: a multivariate analysis between the two variables at each age, a longitudinal analysis of each variable across the two ages, and the analysis of the cross-lags themselves. Before assessing the cross-lag, possible confounding influences from the stability of each variable, the contemporaneous association between two earlier measurements, and the reverse cross-lag must be removed. This can be done by the Cholesky decomposition, which not only decomposes variance within each variable and covariance between variables into genetic and environmental factors, but also controls confounders by attributing their influences to latent factors before decomposing the target variance or covariance. Therefore, by appropriately ordering measured variables in the standard Cholesky decomposition (e.g., achievement at 9, self-perceived abilities at 9, self-perceived abilities at 12 and achievement at 12), the model specifically studies variables listed second and fourth in the order. In this way, the target cross-lag is analyzed after the adjustment of the correlation between self-perceived abilities and achievement at 9, stabilities of self-perceived abilities and achievement (which may not entirely be adjusted, see the Discussion for details) and the reverse cross-lag. In other words, the cross-lagged analysis is independent of the stabilities, the earlier contemporaneous association and the reverse cross-lagged association. Then genetic and environmental contributions to the target cross-lagged association per se are estimated.

To study the cross-lagged association between self-perceived abilities at 9 and school achievement at 12, we used the model illustrated on the left of Figure 1(a, b, c), which includes 12 latent factors. A_1 , C_1 and E_1 represent additive genetic, shared environmental and non-shared environmental contributions to the contemporaneous association between self-perceived abilities and school achievement at 9 ($a_{11} \times a_{12} + c_{11} \times c_{12} + e_{11} \times e_{12}$), the reverse cross-lagged association which is simply estimated as the correlation between school achievement at 9 and self-perceived abilities at 12 ($a_{11} \times a_{13} + c_{11} \times c_{13} + e_{11} \times e_{13}$), and the stability of school achievement ($a_{11} \times a_{14} + c_{11} \times c_{14} + e_{11} \times e_{14}$). A_2 , C_2 and E_2 represent contributions to the stability of self-perceived abilities ($a_{22} \times a_{23} + c_{22} \times c_{23} + e_{22} \times e_{23}$), and the cross-lagged association between self-perceived abilities at 9 and school achievement at

12 ($a_{22} \times a_{24} + c_{22} \times c_{24} + e_{22} \times e_{24}$, i.e. path b_{21} in Figure 2 which will be described later). A_3 , C_3 and E_3 represent contributions to the contemporaneous association between self-perceived abilities and school achievement at 12 ($a_{33} \times a_{34} + c_{33} \times c_{34} + e_{33} \times e_{34}$). Finally, any genetic and environmental contributions specific to school achievement at 12 are represented by A_4 , C_4 and E_4 .

The model on the right of Figure 1(d, e, f) examines the cross-lagged association between school achievement at 9 and self-perceived abilities at 12. The model operates in the same way as the former one, except the order of the variables in the Cholesky decomposition is changed to control for other measures.

The fit of the genetic cross-lagged model is examined by comparing the $-2 \log$ likelihood (-2ll) of it to a saturated model, which estimates variances, covariances and means. The difference is distributed as a chi-square. The degrees of freedom equate the differences between the number of estimated parameters in the saturated model and that in the cross-lagged model. Akaike's information criterion (Akaike, 1987) is also computed, with lower values indicating better fit.

An Alternative Model: The Phenotypic Cross-Lagged Model—This model has been used in recent cross-lagged studies (e.g., Burt et al., 2005; Larsson et al., 2008; Forsman et al., 2010). In this paper, we refer to it as the *phenotypic* cross-lagged model (in contrast to our *genetic* cross-lagged model) because its key assumption is that genetic and environmental factors contribute to cross-lagged associations indirectly through phenotypic-driven processes (Burt et al., 2005). In other words, this model does not directly decompose the cross-lags into genetic and environmental components. For purposes of comparison with our genetic cross-lagged model, we applied the phenotypic cross-lagged model (Figure 2) to the same data.

Results

Descriptive Statistics

Table 1 shows the means and standard deviations for the measures at 9 and 12 years along with the results of a 2×2 (Zygosity $\times$ Sex) ANOVA. Of the 12 main effects and interactions, only three were significant despite the large sample size, and these effects accounted for no more than 0.6% of the variance. The mean NC levels at ages 9 and 12 were consistent with the expected level of achievement for these ages. For further analysis, self-perceived abilities and achievement were corrected for age and sex by means of regression (McGue & Bouchard, 1984) and standardized residual scores were created.

Twin Intraclass Correlations

The twin intraclass correlations for all measures are shown in Table 2 by zygosity. In every case, the MZ twin correlation exceeded that of DZ twins, indicating genetic influence. For self-perceived abilities, heritabilities estimated by doubling the difference between MZ and DZ correlations, were 34% at age 9 and 52% at age 12. Estimates of shared environmental influences — subtracting the above estimates of heritability from MZ correlations — were minimal, while estimates of non-shared environment — subtracting MZ correlations from 1.0 — were substantial. Heritabilities of achievement were 76% at age 9 and 52% at age 12, while shared and nonshared environmental influences were modest. The heritability (A), shared environment (C) and non-shared environment (E) estimates from the intraclass correlations were included in Table 2.

Phenotypic Correlations

The phenotypic correlation between self-perceived abilities at 9 and achievement at 12 was moderate ($r = .26$), as was the correlation between achievement at 9 and self-perceived abilities at 12 ($r = .43$). Self-perceived abilities moderately predicted achievement at 9 and 12 years ($r = .33$ at 9, $r = .41$ at 12). Both self-perceived abilities and achievement were relatively stable from age 9 to 12 (self-perceived abilities: $r = .44$; achievement: $r = .60$). All the correlations above were significant ($p < .01$).

Model Fitting Using the Genetic Cross-Lagged Model

On the left side of Figure 1(a, b, c), the genetic cross-lagged model was applied to the cross-lagged association between self-perceived abilities at age 9 and school achievement at age 12. On the right side (Figure 1d, 1e, 1f), the model was applied to the reverse cross-lag, i.e. the association between achievement at 9 and self-perceived abilities at 12. Comparing to the saturated model, both models fitted the data well ($\chi^2 = 50.08$, $df = 42$, $p = .18$, $AIC = -33.9$). The goodness of the fit of the two models to the same data was confirmed to be equal. 95% confidence intervals (CI) of path estimates are included in Table 3.

Before looking at the cross-lagged associations, the aetiologies of all measures and those of contemporaneous relationships between self-perceived abilities and achievement were examined using estimates from the model. The heritability of self-perceived abilities was moderate at age 9 (31%) and age 12 (49%). Environmental influences for self-perceived abilities were primarily due to nonshared environment (age 9: $C = 10\%$, $E = 59\%$; age 12: $C = 2\%$, $E = 48\%$). School achievement at 9 years was mainly influenced by genetics (68%), with modest environmental contributions ($C = 16\%$, $E = 16\%$); at 12 years, genetic and environmental influences were moderate ($A = 38\%$, $C = 39\%$, $E = 23\%$). Genetic and environmental correlations were used to indicate the extent of overlap between latent factors underlying self-perceived abilities and achievement (Plomin et al., 2008). At 9 and 12 years, the genetic influences on self-perceived abilities were substantially correlated with those on achievement ($r = .61$ at 9, $r = .96$ at 12). There was no significant overlap between their shared environmental influences (age 9: $r = -.06$, 95% CI: $-.86-.51$; age 12: $r = -.35$, 95% CI: $-1.00-1.00$); the non-shared environmental influences on them were modestly correlated ($r = .18$ at 9, $r = .14$ at 12).

The genetic and environmental influences on cross-lagged associations are presented as the percentage of ACE contributions to the model-estimated phenotypic correlations (Neiderhiser et al., 1999). These values were derived from the path estimates shown on the left side of Figure 1, which focuses on the key cross-lag from self-perceived abilities at age 9 to school achievement at age 12. The phenotypic cross-lag controlling for their contemporaneous correlations at age 9, their stabilities from age 9 to 12 and the reverse cross-lag can be estimated as the sum of the products of ACE paths that link self-perceived abilities at 9 and achievement at 12 through latent factors A_2 , C_2 , E_2 , using the formula described before, i.e. $.20 \times .01 + .10 \times .04 + .57 \times .00 = .11$ (95% CI: $.05-.19$). Genetic and environmental contributions to this phenotypic cross-lag can be estimated by dividing the product of related path estimates by the phenotypic cross-lag, although confidence intervals are much larger than for the phenotypic cross-lag. For example, the genetic contribution is $a_{22} \times a_{24} / (a_{22} \times a_{24} + c_{22} \times c_{24} + e_{22} \times e_{24})$, that is, $(.20 \times .01) / .11$. Genetic factors account for about one-quarter of the phenotypic cross-lag (28%, 95% CI: $.00-1.00$). The shared environmental contribution to the phenotypic cross-lag was 55% (95% CI: $.01-1.00$) and nonshared environment accounted for the remainder (17%, 95% CI: $.00-.59$).

For the model depicted on the right side of Figure 1, which focuses on the cross-lagged association between achievement at 9 and self-perceived abilities at 12, the phenotypic cross-lagged correlation was estimated as .24 (95% CI: .04–.36). This phenotypic cross-lag was primarily affected by genetics (73%, 95% CI: .39–.97), with modest influences from shared environment (20%, 95% CI: .00–.78) and nonshared environment (7%, 95% CI: .00–.54). It should be noted that these estimates of the ACE contributions are based on model-fitting results and may not be exactly equal to values derived from Figure 1 where squared path estimates were rounded up.

Model Fitting Using the Phenotypic Cross-Lagged Model

The phenotypic cross-lagged model was also applied to examine the associations between self-perceived abilities and achievement (Figure 2; for 95% confidence intervals of the path coefficients, see Table 4). The fit of this model was examined by a chi-square test against the saturated model ($\chi^2 = 172.09$, $df = 62$, $p < .001$, $AIC = 48.1$).

Genetic and environmental contributions to each measure were estimated by squaring their genetic and environmental path coefficients. At 9 years, the estimates were very close to the estimates from the genetic cross-lagged model (e.g., for self-perceived abilities, $A = 30\%$, $C = 11\%$, $E = 59\%$). At 12 years, the model estimates genetic and environmental influences unique to age 12 (Burt et al., 2005): $A_s = 21\%$, $C_s = 3\%$, $E_s = 47\%$ of the specific variance of self-perceived abilities at age 12; $A_a = 18\%$, $C_a = 20\%$, $E_a = 25\%$ of the specific variance of achievement at age 12.

The aetiologies of the relationships between self-perceived abilities and achievement at each age were also examined using genetic and environmental correlations (Figure 2). At 9 years, the estimates ($r_{a1} = .63$, $r_{c1} = -.05$, $r_{e1} = .16$) were almost the same as estimates from the genetic cross-lagged model. The ACE correlations at 12 years reflected age-specific covariance between the two measures: $r_{a2} = .86$, $r_{c2} = -.99$, $r_{e1} = .12$.

The phenotypic cross-lagged correlations in Figure 2 were comparable to those derived from the genetic cross-lagged models: $b_{21} = .06$ and $b_{12} = .32$. The key issue is that in the phenotypic cross-lagged model these phenotypic cross-lagged correlations are merely weighted by the ACE estimates at age 9 rather than being decomposed into their ACE components of covariance because the model assumes phenotypic transmission (Burt et al., 2005; Larsson et al., 2008). In other words, the phenotypic cross-lagged path from self-perceived abilities at 9 to achievement at 12 is assumed to be affected genetically to the extent that self-perceived abilities at age 9 are heritable (30%). Thus the decomposition of the phenotypic cross-lag was the same as the ACE estimates for self-perceived abilities at 9: 30% A, 11% C and 59% E. Similarly, for the cross-lagged phenotypic path from achievement at 9 to self-perceived abilities at 12, the decomposition merely replicated ACE estimates for achievement at 9: 69% A, 16% C and 15% E.

A Comparison of the Genetic and Phenotypic Cross-Lagged Models

The differences between the two cross-lagged models are highlighted in Figure 3. As can be seen by the blow-up of the genetic paths (from Figures 1 and 2), the genetic cross-lagged model decomposes the cross-lag into its genetic and environmental components of covariance (Figure 3a). In contrast the phenotypic cross-lagged model simply weights the phenotypic cross-lag by the heritability at age 9. As shown in panel c of Figure 3, the two models yield different results. Although both models by chance showed moderate genetic influences (the genetic model: $A = 28\%$; the phenotypic model: $A = 30\%$), the environmental influences behaved differently. In the genetic cross-lagged model, environmental influences were mainly due to shared environment ($C = 55\%$, $E = 17\%$),

whereas non-shared environment contributed most in the phenotypic model ($C = 11\%$, $E = 59\%$). These discrepancies are to be expected because the models differ in their hypotheses about the etiology of a cross-lagged association. Burt et al. (2005) assumed that genetic and environmental influences on a cross-lag entirely reflect those on the previous behavior. In other words, there is no genetic analysis of the cross-lag. However, in the genetic cross-lagged model, the focus is on the genetic analysis of the cross-lag itself and no assumption is made about the mechanism by which the cross-lag emerges.

We also compared the two models on the reverse cross-lagged association between achievement at 9 and self-perceived abilities at 12. In this case, despite the critical differences in the model, the estimates of contributions to the phenotypic cross-lagged path happened to be similar for the two models (genetic model: $A = 73\%$, $C = 20\%$, $E = 7\%$; phenotypic model: $A = 69\%$, $C = 16\%$, $E = 15\%$).

Discussion

A 1999 paper used a model derived from Cholesky decomposition to assess genetic and environmental contributions to the relationships between parenting and adolescent adjustment over time (Neiderhiser et al. 1999). Due to its stringency and complexity, the model yielded conservative estimates and was difficult to understand and to use; it was not used in any subsequent research. A model used in recent studies (e.g., Burt et al., 2005) is also a multivariate longitudinal genetic model but this model does not decompose genetic and environmental influences on the cross-lagged association, which in our view is the critical element of genetic cross-lagged analysis. Instead, uni-variate genetic and environmental contributions at the earlier age are used to weight the phenotypic cross-lagged association.

In this article we introduced a new genetic cross-lagged approach in which the phenotypic cross-lagged effects themselves are decomposed into genetic, shared environmental and nonshared environmental factors using a standard Cholesky decomposition that can be easily understood and adopted. In this way, we can directly estimate the genetic and environmental effects on the cross-lagged paths per se. Applying our genetic cross-lagged model to the aetiology of the reciprocal relationship between self-perceived abilities and school achievement revealed that genetic influences contribute moderately to the association between earlier self-perceived abilities and later school achievement and substantially to the reverse cross-lagged association.

Over the past decade, school achievement has been extensively studied, resulting in many factors that correlate with it (Winne & Nesbit, 2010). Among them, self-perceived abilities (sometimes called academic self-concept) have been acknowledged as an important predictor of school achievement (Marsh & Craven, 1997). Moreover, its relationship with school achievement is reciprocal in that school achievement also has an effect on self-perceived abilities (Guay et al., 2003; Marsh et al., 1999). However, possible genetic contributions to this reciprocal relation have hardly been considered. The only available work has shown that independent of IQ, self-perceived abilities associate with school achievement at the same age for genetic rather than environmental reasons (Greven et al., 2009). This corresponds to our finding that genetic correlations are high at both age 9 and age 12, while environmental correlations are small. Furthermore, our results indicate that genetic influences are important for cross-lagged association between self-perceived abilities at 9 and school achievement at 12, as well as for the cross-lag between school achievement at 9 and self-perceived abilities at 12. This suggests that genetic influences on promoting achievement through self-perceived abilities (and vice versa) should be considered, despite

the long-held belief in the importance of environmental factors (Shavelson et al., 1976; Wigfield & Eccles, 2000).

Limitations

As mentioned before, one major limitation of the genetic cross-lagged approach is that the two cross-lagged associations are represented separately by two different models. As the two models are not nested, it is not possible to test which model better represents the bidirectional relationship, as is typically done in phenotypic cross-lagged analyses. Nor can it compare whether, for example, genetic influences are significantly greater for one cross-lag than the other. However, as a genetic analysis, we are often primarily interested in one causal direction and its aetiology—in our case, the relationship between self-perceived abilities at age 9 and achievement at age 12, rather than the direction of the phenotypic effects.

There is another potential limitation of the genetic cross-lagged model. Although stabilities have been adjusted by independent paths in the model beforehand, the cross-lagged correlation between self-perceived abilities at 9 and achievement at 12 could be somewhat inflated by a possible confounding effect from the stability of self-perceived abilities, because the cross-lagged correlation and the stability are represented by the same latent factors (i.e., A_2 , C_2 and E_2) which include genetic and environmental influences on the cross-lag as well as the stability. Nevertheless, the inflation is rather limited by the moderate magnitude of the stability, which may even be modest given part of the covariance between self-perceived abilities at 9 and those at 12 can be accounted for by latent factors A_1 , C_1 and E_1 . Similarly, the cross-lagged association between achievement at 9 and self-perceived abilities at 12 is potentially inflated with some stability of achievement. To address this problem, we examined several other methods. For instance, to analyse the cross-lag from self-perceived abilities at 9 to achievement at 12, the contemporaneous covariance between self-perceived abilities and achievement at 12 was removed from achievement at 12 by regression to prevent influences from any variance related to self-perceived abilities at 12 including the stability of self-perceived abilities. Then in a trivariate model, that is, a standard Cholesky decomposition of three variables (achievement at 9, self-perceived abilities at 9 and achievement at 12, which are ordered so as to adjust achievement at 9), we decomposed the cross-lag into genetic and environmental factors. Results from this method and others suggested comparable results to those presented in this article, that the cross-lagged association between earlier self-perceived abilities and later achievement was mediated by genes with the environment, as well as for the reverse cross-lag (details of these analyses are available from the first author).

Besides the usual limitations of the twin method (Plomin et al., 2008), one specific limitation of the study is that the phenotypic cross-lagged association is modest between self-perceived abilities at 9 and achievement at 12. In multivariate genetic studies, a moderate phenotypic correlation between variables is necessary to obtain reliable estimates of genetic and environmental contributions to the phenotypic covariance. Here, the phenotypic correlation between self-perceived abilities at 9 and achievement at 12 was moderate (.26), but the cross-lagged phenotypic correlation was halved (.11) after the stabilities and contemporaneous correlation at 9 were taken into account, which represents the ceiling for genetic analysis of the cross-lags.

In addition, the present study includes possible confounding by IQ. Studies have shown that IQ and self-perceived abilities are moderately correlated (Ackerman & Wolman, 2007; Furnham & Chamorro-Premuzic, 2004); IQ and school achievement are substantially correlated (Chamorro-Premuzic & Furnham, 2005). Therefore, the cross-lagged association between self-perceived abilities and achievement may be partly mediated by IQ. However,

IQ contributes modestly to the cross-lagged association between earlier self-perceived abilities and later achievement, although the effect of IQ is considerable on the reverse cross-lag (Chamorro-Premuzic et al., 2010). Nonetheless, future research would profit by extending the cross-lagged genetic model to include other covariates such as IQ.

Conclusions

We present a novel analytic approach for genetic cross-lagged research. This approach can reveal genetic and environmental influences on the cross-lagged associations per se using a standard Cholesky decomposition. The application of the model to the cross-lagged associations between self-perceived abilities and achievement at 9 and 12 years indicates important genetic influences. The superiority of the genetic cross-lagged model to evaluate genetic and environmental contributions to cross-lagged associations is indicated by comparing it to the phenotypic cross-lagged model using the same data.

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Figure 1.

The genetic cross-lagged model.

Note: The model on the left (a, b, c) tests the key cross-lag from self-perceived ability at age 9 (SPA9) to school achievement at age 12 (NC12). The model on the right (d, e, f) tests the reverse cross-lag from NC9 to SPA12. Measured variables are depicted in rectangles. Latent variables A (additive genetic factor), C (shared environmental factor), and E (nonshared environmental factor) are presented in the circles. Standardized path estimates (i.e., $a_{11} \sim a_{44}$, $c_{11} \sim c_{44}$, $e_{11} \sim e_{44}$) are also represented in the diagram. The thickness of each arrow indicates the strength of the corresponding path. Nonsignificant paths are depicted by dashed lines. 95% confidence intervals of path estimates are listed in Table 3. The models represent only one member of the twin pair. If both siblings are illustrated, all genetic factors should be connected by double-headed arrows with values corresponding to the degree of genetic similarity (1.0 for MZ, 0.5 for DZ). The shared environmental factors are set to be the same for both members of the twin, and the non-shared environmental factors are uncorrelated for both members.

Figure 2.

The phenotypic cross-lagged model.

Note: Measured variables are depicted in rectangles: SPA = self-perceived ability; NC = school achievement. The latent variables A (additive genetic factor), C (shared environmental factor), and E (nonshared environmental factor) are presented in the circles. As (additive genetic factor), Cs (shared environmental factor), and Es (nonshared environmental factor) are residual variances specific to age 12. Standardized path estimates for these factors (i.e., $a_1, c_1, e_1 / a_2, c_2, e_2 / a_3, c_3, e_3 / a_4, c_4, e_4$), genetic and environmental correlations (i.e., $r_{a1}, r_{c1}, r_{e1} / r_{a2}, r_{c2}, r_{e2}$), cross-age stability paths (i.e., b_{11}, b_{22}) and cross-lagged paths (i.e., b_{21}, b_{12}) are also represented in the diagram. The thickness of each arrow indicates the strength of the corresponding path. Nonsignificant paths are depicted by dashed lines. 95% confidence intervals of path estimates are listed in Table 4. The model represents only one member of the twin pair.

Figure 3.

A comparison of the genetic and phenotypic cross-lagged models.

Note: SPA = self-perceived ability; NC = school achievement. Here we only show the comparison for the cross-lagged association of SPA at 9 and NC at 12. (a) is the part of the genetic cross-lagged model (see Figure 1) that represents the way genetic factors mediate the cross-lag between SPA at 9 and NC at 12 independent of SPA at 12 and NC at 9. (b) is the part of the phenotypic cross-lagged model (see Figure 2) that indicates that the model merely weights the phenotypic cross-lag from SPA at 9 by the genetic influence on SPA at age 9. (c) represents genetic (A), shared environmental (C) and nonshared environmental (E) contributions to the phenotypic cross-lagged association estimated by the genetic cross-lagged model and the phenotypic cross-lagged model.

Means (and Standard Deviations) by Zygosity and Sex and ANOVA Results Showing Significance and Effect Size by Zygosity and Sex

Table 1

Measure	Zygosity				Sex		ANOVA		
	All	MZ	DZ		Female	Male	Zygosity	Sex	Zygosity × Sex
Age 9									
SPA	3.98 (.61)	3.97 (.60)	3.98 (.62)		3.93 (.61)	4.03 (.61)	$p = .618$	$p < .001$	$p = .030$
	$n = 2230$	$n = 1191$	$n = 1039$		$n = 1222$	$n = 1008$	$2 < .001$	$2 = .006$	$2 = .002$
NC	2.98 (.58)	2.96 (.58)	3.00 (.58)		2.99 (.57)	2.97 (.60)	$p = .086$	$p = .432$	$p = .386$
	$n = 1866$	$n = 973$	$n = 893$		$n = 1004$	$n = 862$	$2 = .002$	$2 < .001$	$2 < .001$
Age 12									
SPA	3.88 (.58)	3.87 (.59)	3.89 (.57)		3.87 (.59)	3.89 (.57)	$p = .348$	$p = .264$	$p = .880$
	$n = 3852$	$n = 2025$	$n = 1827$		$n = 2098$	$n = 1754$	$2 < .001$	$2 < .001$	$2 < .001$
NC	4.39 (.91)	4.35 (.89)	4.43 (.92)		4.39 (.87)	4.39 (.95)	$p = .035$	$p = .895$	$p = .185$
	$n = 2577$	$n = 1349$	$n = 1228$		$n = 1397$	$n = 1180$	$2 = .002$	$2 < .001$	$2 = .001$

Note: Results for one randomly selected member of each twin pair. The number of randomly selected individuals (indicated by n) is bigger than expected as compared to Table 2, because there are some twin pairs for whom data were available for only one member. SPA = self-perceived abilities; NC = school achievement; MZ = monozygotic; DZ = same-sex dizygotic; η^2 = eta squared (effect size).

Table 2
Twin Intraclass Correlations (and 95% Confidence Intervals) and Estimated ACE

Measure	rMZ	rDZ	A	C	E
Age 9					
SPA	.41 (.36–.46), <i>n</i> = 1187	.24 (.18–.30), <i>n</i> = 1040	.34	.07	.59
NC	.85 (.83–.86), <i>n</i> = 975	.47 (.42–.53), <i>n</i> = 884	.76	.09	.15
Age 12					
SPA	.54 (.51–.57), <i>n</i> = 2014	.28 (.24–.33), <i>n</i> = 1824	.52	.02	.46
NC	.82 (.80–.84), <i>n</i> = 1340	.56 (.51–.60), <i>n</i> = 1200	.52	.30	.18

Note: SPA = self-perceived abilities; NC = school achievement; n = number of complete twin pairs; A = additive genetic; C = shared environment; E = nonshared environment; rMZ= intraclass correlations of monozygotic twins; rDZ = intraclass correlations of same-sex dizygotic twins. All correlations are significant at *p* < .01.

Table 3

Path Estimates (and 95% Confidence Intervals) of the Genetic Cross-Lagged Models Depicted in Figure 1

Genetic cross-lagged model (Figure 1a, b, and c)					The second genetic cross-lagged model (Figure 1d, e, and f)				
A	C	E	A	C	E	A	C	E	
$a_{11} = .68 (.59-.77)$	$c_{11} = .16 (.08-.26)$	$e_{11} = .16 (.14-.17)$	$a_{11} = .31 (.16-.44)$	$c_{11} = .10 (.00-.23)$	$e_{11} = .59 (.54-.63)$				
$a_{12} = .11 (.05-.19)$	$c_{12} = .00 (.00-.05)$	$e_{12} = .02 (.01-.04)$	$a_{12} = .25 (.11-.41)$	$c_{12} = .00 (.00-.18)$	$e_{12} = .01 (.00-.01)$				
$a_{13} = .29 (.20-.38)$	$c_{13} = .01 (.00-.06)$	$e_{13} = .01 (.00-.02)$	$a_{13} = .12 (.02-.37)$	$c_{13} = .03 (.00-.39)$	$e_{13} = .00 (.00-.01)$				
$a_{14} = .23 (.13-.36)$	$c_{14} = .25 (.08-.46)$	$e_{14} = .00 (.00-.02)$	$a_{14} = .29 (.15-.50)$	$c_{14} = .01 (.00-.10)$	$e_{14} = .03 (.02-.04)$				
$a_{22} = .20 (.05-.34)$	$c_{22} = .10 (.00-.23)$	$e_{22} = .57 (.52-.61)$	$a_{22} = .43 (.19-.58)$	$c_{22} = .16 (.00-.25)$	$e_{22} = .15 (.13-.17)$				
$a_{23} = .07 (.00-.23)$	$c_{23} = .01 (.00-.10)$	$e_{23} = .02 (.01-.04)$	$a_{23} = .11 (.01-.28)$	$c_{23} = .27 (.00-.47)$	$e_{23} = .00 (.00-.01)$				
$a_{24} = .01 (.00-.14)$	$c_{24} = .04 (.00-.26)$	$e_{24} = .00 (.00-.01)$	$a_{24} = .07 (.02-.16)$	$c_{24} = .01 (.00-.05)$	$e_{24} = .00 (.00-.01)$				
$a_{33} = .13 (.01-.22)$	$c_{33} = .00 (.00-.09)$	$e_{33} = .45 (.42-.49)$	$a_{33} = .14 (.01-.27)$	$c_{33} = .10 (.00-.28)$	$e_{33} = .23 (.20-.27)$				
$a_{34} = .14 (.00-.27)$	$c_{34} = .10 (.00-.28)$	$e_{34} = .00 (.00-.01)$	$a_{34} = .13 (.00-.21)$	$c_{34} = .00 (.00-.09)$	$e_{34} = .01 (.00-.02)$				
$a_{44} = .00 (.00-.18)$	$c_{44} = .00 (.00-.28)$	$e_{44} = .23 (.19-.26)$	$a_{44} = .00 (.00-.16)$	$c_{44} = .00 (.00-.06)$	$e_{44} = .45 (.41-.49)$				

Note: All paths estimates are standardized and squared to refer to the percentage of variance explained. A = additive genetic; C = shared environment; E = nonshared environment.

Table 4

Path Estimates (and 95% Confidence Intervals) of the Phenotypic Cross-Lagged Model Depicted in Figure 2

Measure	A	C	E
SPA at age 9	$a_1 = .30 (.15-.45)$	$c_1 = .11 (.00-.23)$	$e_1 = .59 (.54-.64)$
NC at age 9	$a_2 = .69 (.59-.79)$	$c_2 = .16 (.06-.26)$	$e_2 = .15 (.14-.17)$
SPA at age 12	$a_3 = .21 (.09-.27)$	$c_3 = .03 (.00-.13)$	$e_3 = .47 (.44-.52)$
NC at age 12	$a_4 = .18 (.05-.31)$	$c_4 = .20 (.11-.32)$	$e_4 = .25 (.21-.29)$
	r_A	r_C	r_E
SPA — NC at age 9	$r_{a1} = .63 (.43-.89)$	$r_{c1} = -.05 (-1.00-.58)$	$r_{e1} = .16 (.09-.23)$
SPA — NC at age 12	$r_{a2} = .86 (.29-1.00)$	$r_{c2} = -1.00 (-1.00-.40)$	$r_{e2} = .12 (.02-.23)$
Phenotypic Cross-age Coefficients			
$b_{11} = .34 (.31-.37)$	$b_{21} = .06 (.03-.10)$	$b_{12} = .32 (.28-.35)$	$b_{22} = .58 (.54-.62)$

Note: SPA = self-perceived abilities; NC = school achievement; A = additive genetic; C = shared environment; E = nonshared environment. All ACE paths estimates are standardized and squared to refer to the percentage of variance explained. r_A = genetic correlation; r_C = shared environmental correlation; r_E = nonshared environmental correlation.