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**Externalizing problems in childhood and adolescence predict
subsequent educational achievement but for different
genetic and environmental reasons**

Externalizing problems in childhood and adolescence predict subsequent educational

achievement but for different genetic and environmental reasons

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Abstract

Background: Childhood behavior problems predict subsequent educational achievement; however, little research has examined the etiology of these links using a longitudinal twin design. Moreover, it is unknown whether genetic and environmental innovations provide incremental prediction for educational achievement from childhood to adolescence.

Methods: We examined genetic and environmental influences on parental ratings of behavior problems across childhood (age 4) and adolescence (ages 12 and 16) as predictors of educational achievement at age 16 using a longitudinal classical twin design.

Results: Shared-environmental influences on anxiety, conduct problems, and peer problems at age 4 predicted educational achievement at age 16. Genetic influences on the externalizing behaviors of conduct problems and hyperactivity at age 4 predicted educational achievement at age 16. Moreover, novel genetic and (to a lesser extent) nonshared-environmental influences acting on conduct problems and hyperactivity emerged at ages 12 and 16, adding to the genetic prediction from age 4.

Conclusions: These findings demonstrate that genetic and shared-environmental factors underpinning behavior problems in early childhood predict educational achievement in mid-adolescence. These findings are consistent with the notion that early-childhood behavior problems reflect the initiation of a life-course persistent trajectory with concomitant implications for social attainment. However, we also find evidence that genetic and nonshared-environment innovations acting on behavior problems have implications for subsequent educational achievement, consistent with recent work arguing that adolescence represents a sensitive period for socio-affective development.

Keywords: education; genetics; longitudinal; twin study; behavior problems; SDQ

Introduction

The long-established importance of educational achievement for later-life success (e.g. Sewell & Hauser, 1975) has led to a significant body of work examining the antecedents of school achievement (Deary, Strand, Smith, Fernandes, 2007; Fergusson & Horwood, 1998; Shakeshaft et al., 2013; Vitaro, Brendgen, Larose, & Tremblay, 2005). One area of specific focus has been childhood and adolescent behavior problems. Several studies have reported that genetic and environmental factors underpin individual differences in both educational achievement (Bartels, Rietveld, Van Baal, & Boomsma, 2002; Shakeshaft et al., 2013; Asbury & Plomin, 2013) and child and adolescent behavior problems (Eaves et al., 1997; Lewis & Plomin, 2015). Furthermore, these genetic and environmental factors have been found to be moderately-to-substantially overlapping: that is, some of the same genes and experiences affect both educational achievement and behavior problems (Hicks et al., 2008; Johnson et al., 2005). Less well understood is the extent to which genetic and environmental influences on childhood and adolescent behavior problems predict educational achievement at age 16 (the end of mandatory education in many countries).

In the current study we used a longitudinal twin design to examine whether childhood and adolescent behavior problems share genetic and environmental influences with educational achievement, and how these influences relate over time. Specifically, we sought to estimate the extent to which genetic and environmental influences acting on behavior problems in early childhood, before formal schooling begins, can predict achievement in public examinations at age 16. Moreover, we examined whether novel genetic and environmental influences on behavior problems, emerging over the course of development (Lewis & Plomin, 2015), would provide additional sources of prediction for educational achievement at age 16. Next we briefly introduce phenotypic and behavioral genetic investigations of educational achievement and behavior problems before moving to formal

tests of the role of genetic and environmental influences on behavior problems and educational achievement over childhood and adolescence.

Behavior Problems and Educational Achievement

Behavior problems pose intuitive risks to the prospects of school success. Children and adolescents with externalizing behavior problems (e.g. conduct problems, hyperactivity) will likely find it harder to pay attention in the classroom or to comply with school rules, and so it would be unsurprising to find inverse associations between externalizing behaviors and school success. A range of studies have examined whether behavior problems and educational achievement are inversely associated and have consistently confirmed this expectation. For example, in a large New Zealand birth cohort, conduct disorder at age 8 was found to predict leaving school at age 18 without educational qualifications (Fergusson & Horwood, 1998). Similarly, in a large Canadian community sample, hyperactivity-inattention and aggressiveness-opposition measured in kindergarten were found to predict non-completion of high school (Vitaro, Brendgen, Larose, & Tremblay, 2005). A large number of other studies also provide support for the link between early childhood externalizing behaviors and subsequent low educational achievement (Chen et al., 2010; Masten et al., 2005; Veldman et al., 2014).

Beyond externalizing behaviors, the links between educational achievement and behavior problems are more mixed. For example, one study noted that prosociality (often referred to as a behavioral strength: Goodman, 1997) at age 8 predicted educational achievement 5 years later in mid-adolescence (Caprara et al., 2000). A similar observation in a 5-year longitudinal sample of Chinese school students followed from age 8 also found that prosocial competence predicted academic achievement in subsequent years (Chen, Huang, Chang, Wang, & Li, 2010). In contrast, other studies have failed to observe prosociality as a

predictor of subsequent educational success (e.g. Kokko et al., 2006). In the domain of internalizing problems, similarly mixed results have been noted. For example, one study reported that higher levels of anxiety in the pre-school years was predictive of higher school grades in early adolescence (DiLalla, Marcus, & Wright-Phillips, 2004) but other research has failed to observe such links (van Lier et al., 2012).

Building on the well-replicated phenotypic links between externalizing behaviors and educational achievement have been genetically-informative studies seeking to assess the relative roles of the genetic and environmental factors underpinning this association. In early childhood the link between externalizing behavior and educational achievement has been reported to be mostly attributable to shared-environmental factors, although genetic factors have also been noted to play a role (Newsome, Boisvert, & Wright, 2014). In mid-childhood this pattern appears to shift towards genetic factors accounting for the majority of the phenotypic links between externalizing behaviors and educational achievement. For example, in a large UK cohort (the Twins Early Development Study (TEDS): also used in the current study) hyperactivity and educational success at age 7 were found to share substantial genetic links, alongside more modest nonshared-environmental links (Saudino & Plomin, 2007). Similarly, results from the Minnesota Twin Family Study showed that at age 11 genetic influences on inattention and educational achievement were highly overlapping, although the genetic link between disruptive behavior and educational achievement, while statistically significant, was less pronounced (Johnson et al., 2005). In the same sample, achievement striving, self-control, and aggression at age 17 have been reported to be genetically related to educational success (also at age 17), alongside a modest link via nonshared-environmental influences (Hicks et al., 2008). Finally, work using the TEDS twin cohort reported that standardized UK high-school exam results at age 16 were heritable and genetically associated with many psychological traits including behavior problems, although associations between

educational achievement and specific components of behavior problems were not detailed (Krapohl et al., 2014).

The Current Study

These studies provide insights into common genetic and environmental influences underlying observed relationships between behavior problems and educational achievement. However, this literature is still in its infancy and a number of important questions remain unanswered. Firstly, while childhood externalizing behaviors are phenotypically predictive of adolescent educational achievement (Fergusson & Horwood, 1998; Vitaro et al., 2005), are these phenotypic links explained by genetic or environmental factors? Indeed, conduct problems show stable genetic and shared-environment influences from age 4 to age 16 (Lewis & Plomin, 2015). As such, it is conceivable that either or both of these sources of variance might account for individual differences in their prediction of educational achievement at age 16.

Secondly, recent work has highlighted that childhood and adolescent externalizing behaviors are underpinned both by stable genetic and environmental influences, as noted above, but also by innovative genetic and environmental influences (i.e. effects that emerge across development: Lewis & Plomin, 2015). As such, do early-emerging (i.e. ≤ age 4) and subsequent (i.e. > age 4) genetic and environmental factors independently relate to later educational achievement?

A range of perspectives have been indirectly informative on this issue. Perhaps most prominently, the developmental taxonomy proposed by Moffitt (1993) stresses that antisocial behavior follows one of two main trajectories: life-course persistent or adolescent-limited. The former is argued to reflect disrupted neuropsychological functioning and temperament difficulties, which in turn negatively impact learning and interpersonal relations, and

subsequently can serve to impair life outcomes. The latter trajectory is posited to reflect the

extreme cases of the otherwise normative adolescent desire to attain status and a distinct

personal identity. Here externalizing behaviors are argued to be simply the manifestation of

these goals. Of importance, this sub-set of adolescents is believed to be relatively goal-

directed in their externalizing behaviors: "adolescence-limited delinquents are likely to

engage in antisocial behavior in situations where such responses seem profitable to them, but

they are also able to abandon antisocial behavior when prosocial styles are more rewarding"

(Moffitt, 1993, p. 686). As such, one would expect that genetic and environmental influences

on childhood externalizing would predict educational achievement in adolescence, either as a

result of a deleterious developmental cascade, or because the underpinning psychological

characteristics of the behavior problems are stable over time and create issues for schooling

in a more proximal fashion. In contrast, any genetic and environmental influences on

externalizing that emerge in adolescence would be expected to contribute less to the

prediction of educational achievement, despite the more proximal nature of these effects.

However, recent observations suggest that adolescence is a sensitive period of development

for a range of socio-affective processes – such as emotion regulation and impulse control

(Steinberg, 2007). These processes have well-established links to behavior problems

(Eisenberg et al., 2001; Silk, Steinberg, & Morris, 2003) and so this sensitive period of socio-

affective development may reflect important independent risk factors for subsequent life

success in their own right (Blakemore, 2010). As such, it is an open question whether early or

later emerging genetic and environmental influences on behavior problems exert the greater

impact on educational outcomes.

In addition to our core questions, here we also took the opportunity to examine how

internalizing traits (i.e. anxiety, peer problems) and prosociality related to academic

achievement, both phenotypically and via underlying genetic and environmental pathways.

Establishing the presence and (where relevant) the etiology of such effects is important given the mixed results in these domains, as noted above.

To answer these questions we used a large and population-representative sample of UK monozygotic and dizygotic twins who have been followed since birth as participants in the Twins' Early Development Study (TEDS) (Oliver & Plomin, 2007). TEDS twins have been surveyed on a wide range of behavioral and cognitive characteristics throughout their lifespan. Here we used the Strengths and Difficulties Questionnaire (SDQ: Goodman, 1997) instrument to assess behavior problems with parental ratings obtained at each age in order to maintain a consistent mode of rating across each wave of measurement. We used SDQ scores at ages 4 and 12 for all SDQ scales, and also for age 16 for prosociality, conduct problems, and hyperactivity (SDQ anxiety and peer problems were not measured at age 16 and so were unavailable for analysis). Educational achievement was measured as the mean score of performance at age 16 in the standardised high school completion exams taken in the UK: the General Certificate of Secondary Education (GCSE).

Methods

Participants

Participants were drawn from the Twins Early Development Study (TEDS), an ongoing longitudinal study following monozygotic (MZ) and dizygotic (DZ) twins born in England and Wales between 1994 and 1996 (Haworth et al., 2013). The TEDS sample is representative of the UK population (Kovas, Haworth, Dale, & Plomin, 2007) and the project received approval from the Institute of Psychiatry Ethics Committee (05/Q0706/228). Twin zygosity was determined using a parental rating measure of similarity and DNA genotyping (Price, Freeman, Craig, Petrill, Ebersole & Plomin, 2000). The number of complete twin pairs for each zygosity class across the three measurement points were as follows (also see

Supplementary Table 1 for full details)*:* MZ male pairs: n = 720-1166; MZ female pairs: n = 1028-1350; DZ male pairs: n = 670-1196; DZ female pairs: n = 886-1247; and DZ opposite sex pairs: n = 1513-2352. 52% of those individuals who were assessed at age 4 (for behavior problems) were re-assessed at age 16.

Measures

Strength and Difficulties Questionnaire (SDQ): The Strengths and Difficulties Questionnaire (SDQ) is a short but reliable instrument (25 items: Goodman, 2001; Stone, Otten, Engels, Vermulst, & Janssens, 2010) for measuring psychosocial problems in children (Goodman, 1997). The SDQ consists of five scales measuring anxiety, conduct problems, hyperactivity-inattention, peer problems, and prosocial behavior. Higher scores indicate greater difficulties (i.e. for anxiety, conduct problems, hyperactivity, peer problems) or strengths (i.e. for prosociality). In the current study we used parent-rated scores for the SDQ sub-scales acquired when the child was 4 and 12 years old. Scores for prosociality, conduct problems, and hyperactivity were also acquired by parental rating when the individual was 16. Cronbach's alpha was low for conduct problems (all ages: α range = .52-.57) and for peer problems (age 4: α = .47), although in line with previously reported values (Goodman, 2001). Cronbach's alpha was broadly acceptable for the rest of the SDQ measures (anxiety: α range = .60-.68; hyperactivity: α range = .71-.77; peers age 12: α range = .64; prosociality: α range = .67-.73).

Educational Achievement: GCSEs are graded from A* (the highest grade) to G (the lowest pass grade). We coded these grades from 11 (A*) to 4 (G): this scoring scheme reflects the fact that sub-GCSE levels of attainment represent National Curriculum levels 1, 2 and 3 and a G at GCSE was equivalent to Level 4 attainment. We constructed our measure of educational achievement as the mean score for the three required subjects: English (either the

English language grade, or the mean of the English language grade and the English literature grade where both exams were taken), Science (the mean of all Science GCSEs taken), and Mathematics.

Analysis

Correlations between twins differing in their degrees of genetic relatedness (i.e., MZ and DZ twins) are useful as a guiding heuristic to estimate relative magnitudes of genetic and environmental effects (Plomin, DeFries, Knopik, & Neiderhiser, 2013). Three sources of variance are typically estimated using data from MZ and DZ twins: additive genetic (A), shared-environment (C), and nonshared-environment effects (E). A reflects the aggregate impact of those genetic effects that sum up to influence a phenotype. C reflects the action of environmental factors shared by twins that serve to make them more similar on a particular phenotype. E reflects the action of environmental factors unique to individuals within a twin pair that serve to make them differ from each other on a particular phenotype. The presence of genetic effects on a given phenotype is typically inferred if the correlations between MZ twins are larger than the correlations for DZ twins. The presence of shared-environment effects is inferred if the correlations for DZ twins are larger than half the magnitude of the correlations for the MZ twins. Finally, nonshared-environmental effects are inferred if correlations for the MZ twins are less than unity. As such, this variance component also contains measurement error.

These correlational analyses were extended using formal model-fitting of variance-covariance matrices to the twin data. This approach allows parameter estimates in univariate models to be formally tested for significance as well as allowing multivariate models – the core focus of the current study – to be analyzed. The validity of inferences from twin analyses, as with all methods, rest on certain assumptions. Firstly, that MZ and DZ twins are

equally correlated with regards to environments of etiological importance for the trait under study (i.e. the equal-environments assumption); secondly, the absence of parental assortative mating for the trait under study. Violations of the former will serve to (spuriously) increase estimates of heritability whereas violation of the latter will serve to (spuriously) increase estimates of shared-environment effects.

In the current study, longitudinal analyses were central to our tests: We sought to estimate the extent to which genetic and environmental effects underlying SDQ measures at ages 4, 12, and 16 (for conduct problems, hyperactivity, and prosociality only) were associated with educational achievement at age 16. To perform this analysis, we used the Cholesky decomposition. The Cholesky decomposition specifies as many factors as there are variables for each source of variance, with each subsequent factor having one fewer pathway than the preceding factor (see Figure 1). In other words, for additive genetic effects (A) the first latent factor loads on all of the n measured variables: The subsequent latent factors load on n_{-1}, n_{-2}…n_{-i} variables. In this way each factor accounts for as much of the remaining variance as possible, until the last factor accounts for just the residual variance in the last measured variable. This is repeated for the shared-environment factors (C) and nonshared-environmental factors (E). This design makes it possible to estimate the extent to which early emerging genetic and environmental influences on an SDQ trait predict later educational achievement. Moreover, this design allows us to examine whether innovative genetic and environmental factors – i.e. sources of variance independent of earlier genetic and environmental influences – contribute additional genetic and environmental prediction to educational achievement at age 16. Twin models were fitted using full-information maximum-likelihood in OpenMx 2.0 (Boker et al., 2011; Boker et al., 2013) running within R 3.2 (R Development Core Team, 2015).

----- Insert Figure 1 here -----

Results

Descriptive statistics for all of the study variables are detailed in full in Table 1. Assumption testing using all twins indicated that means and variances could be equated across twin order, zygosity, and sex for most variables, with the small number of significant differences observed consistent with the large number of tests performed. Of note, however, was evidence for modest-to-moderate mean sex differences, particularly for hyperactivity and prosociality. Sex-limitation modelling (testing for quantitative and qualitative genetic and environmental differences across sex) largely indicated that genetic and environmental influences could be equated across sex, with the significant differences that were observed mostly being either small in magnitude or, again, consistent with the large number of tests performed. Following these observations we pooled our sample across sex, but used sex-residualized variables for all twin analyses.

----- Insert Table 1 here -----

Phenotypic Analyses

We first examined whether age 4 SDQ traits predicted educational achievement at age 16. Correlational analyses showed significant negative links with anxiety ($r = -.06$, $p < .001$), conduct problems ($r = -.19$, $p < .001$), hyperactivity ($r = -.23$, $p < .001$), and peer problems ($r = -.09$, $p < .001$). No association between age 4 prosociality and educational achievement was observed ($r = .02$, $p > .05$). These associations remained significant when controlling for parental socio-economic status (indexed by parental education level, occupation, and family income) and sex. The full set of inter-correlations is presented in Supplementary Table 2.

We next sought to test whether behavior problems at age 12 and 16 added to this prediction. As such we built a series of phenotypic Cholesky decomposition models (see Figure 2), which followed the same logic as detailed above for the twin analyses. These analyses indicated that all SDQ traits at age 4 – with the exception of prosociality – were significant predictors of age 16 educational achievement (in line with the correlational analyses reported above). Of importance, we also observed that in all cases SDQ traits provided incremental prediction at subsequent ages. The size of these effects ranged from moderate (hyperactivity$_{age12}$ → educational achievement β = -.29) to modest (prosociality$_{age12}$ → educational achievement β = .06) (see Figure 2). Controlling for parental socio-economic status and sex did not lead to any notable changes in the magnitude or significance of path estimates.

----- Insert Figure 2 here -----

Twin Analyses

Educational achievement showed significant genetic (A=.55, p < .001), shared-environment (C=.35, p < .001), and nonshared-environment (E=.11, p < .001) effects (also see Shakeshaft et al., 2013). The univariate twin analyses for the behavioral problems variables have been reported in other published work (Lewis, Haworth, & Plomin, 2014; Saudino & Plomin, 2007; Shakeshaft et al., 2013) and so are not detailed in full here (but see Supplementary Tables 3-5). In brief, though, SDQ traits were all underpinned by moderate-to-large genetic and nonshared-environmental influences, with modest shared-environmental influences evident for SDQ-conduct problems and SDQ-prosociality.

We next turned to tests of genetic and environmental influences underpinning the phenotypic associations between SDQ traits and educational achievement. We built a series

of Cholesky models with SDQ traits at age 4, age 12, and age 16 (conduct problems, hyperactivity, and prosociality only), and educational achievement score entered in chronological order from left to right (see Figure 1 or Figures 3-5). To test whether SDQ traits were genetically and environmentally associated with educational achievement we examined each of the genetic paths shared between educational achievement and SDQ age 4, age 12, and age 16, respectively. These parameters correspond to A_4, A_{12}, and A_{16} to educational achievement in Figure 1. For conduct problems these genetic paths were significant at all ages: conduct$_{A4}$-educational achievement: $\Delta\chi^2$ (1) = 17.88, p < .001; conduct$_{A12}$-educational achievement: $\Delta\chi^2$ (1) = 6.03, p = .01; conduct$_{A16}$-educational achievement: $\Delta\chi^2$ (1) = 20.67, p = < .001. Similar results were observed for hyperactivity: hyperactivity$_{A4}$-educational achievement: $\Delta\chi^2$ (1) = 98.85, p < .001; hyperactivity$_{A12}$-educational achievement: $\Delta\chi^2$ (1) = 27.22, p < .001; hyperactivity$_{A16}$-educational achievement: $\Delta\chi^2$ (1) = 22.59, p = < .001. No genetic associations were observed between the other three SDQ traits (at any age) and educational achievement: all $\Delta\chi^2$ (1) < 1.21, all p > .27.

Shared- and nonshared environmental influences were examined using the same principles detailed above. For anxiety, conduct problems, and peer problems, shared-environmental influences were significantly associated with educational achievement, and these influences were all reflective of early-emerging shared-environment influences with broadly stable effects thereafter: anxiety$_{C4}$-educational achievement: $\Delta\chi^2$ (1) = 7.81, p = .005; conduct$_{C4}$-educational achievement: $\Delta\chi^2$ (1) = 14.56, p < .001; peer problems$_{C4}$-educational achievement: $\Delta\chi^2$ (1) = 19.91, p < .001. No further shared environmental associations were observed between SDQ traits (at any age) and educational achievement.

Nonshared environmental influences were more nuanced. For conduct problems these paths were significant at ages 12 ($\Delta\chi^2$ (1) = 4.80, p = .03) and 16 ($\Delta\chi^2$ (1) = 7.44, p = .006).

For hyperactivity at ages 4 ($\Delta\chi^2$ (1) = 10.04, p = .002), 12 ($\Delta\chi^2$ (1) = 83.08, p < .001), and 16 ($\Delta\chi^2$ (1) = 95.30, p < .001). For prosociality these overlapping influences were only apparent cross-sectionally at age 16 ($\Delta\chi^2$ (1) = 7.07, p = .008).

Finally, we examined the magnitude of the overlap between genetic and environmental influences on SDQ traits and educational achievement. Genetic influences on conduct problems that were present by age 4 accounted for 3.1% of the genetic effects underpinning educational achievement at age 16. Genetic influences on conduct problems that were present by ages 12 and 16 accounted for a further 1.5% and 5.3%, respectively. Genetic influences on hyperactivity that were present by age 4 accounted for 16% of the genetic effects underpinning educational achievement at age 16. Genetic influences on hyperactivity that were present by ages 12 and 16 accounted for a further 6.2% and 4.9%, respectively. Shared-environmental influences on peer problems fully overlapped (100%) with the shared-environment influences on educational achievement, and these overlapping influences were present from age 4. Shared-environmental influences on anxiety accounted for 25.7% of the shared-environment influences on educational achievement, and these overlapping influences were present from age 4. Nonshared-environmental influences on hyperactivity that were present by age 4 accounted for 1% of the nonshared-environmental effects underpinning educational achievement at age 16. Nonshared-environmental on hyperactivity that were present by ages 12 and 16 accounted for a further 5.8% and 5.8%, respectively. Conduct problems and prosociality at age 16 each showed nonshared-environmental influences that overlapped with educational achievement: < 1% in both cases. Full model parameter estimates for additive genetic, shared-, and nonshared-environmental influences are detailed in Figures 3-5.

Subsidiary Analyses

The above analyses independently addressed each of the behavioral problems and their respective links to educational achievement. Our results indicated that conduct problems and hyperactivity are both genetically linked with educational achievement. This observation gives rise to the question of whether the genetic contribution from conduct problems to educational achievement is specific to conduct problems, or overlaps with hyperactivity. Similarly, conduct problems, anxiety, and peer problems all showed shared-environment links with educational achievement. As such, is the shared-environmental contribution from conduct problems to educational achievement specific to conduct problems, or does it overlap with that of anxiety and peer problems? The Cholesky decomposition is ill-suited to address this issue as the general factor also necessarily includes specific variance to whichever variable is included first in the model. As such we used the independent pathways model (see Supplementary Figure 1). This model specifies both a general factor and specific factors for genetic, shared-, and nonshared-environment effects. Accordingly, if this model shows a good fit to the data (relative to the baseline Cholesky) it provides evidence that genetic and environmental covariance between the measured variables can be accounted for by the general factor. For these analyses we focused specifically on age 4 behavioral problems as not all measures were available at age 16.

We first used this model to examine whether the genetic influences underpinning conduct problems and hyperactivity provide distinct or common genetic prediction for educational achievement. The independent pathways model provided a good fit to the data and was not appreciably different (Burnham & Anderson, 2004) to the Cholesky decomposition ($AIC_{Cholesky} = 15333.01$ vs $AIC_{IPMod} = 15335.40$). We detail the parameter estimates of the independent pathways model in Supplementary Figure 1. As such, this analysis indicates that while conduct problems and hyperactivity (at age 4) are both genetic

predictors of age 16 educational achievement, this prediction reflects a common etiology.

We next examined whether the shared-environment links between behavioral problems – specifically, conduct problems, anxiety, and peer problems – and educational achievement reflected processes specific to each behavioral problem or a more general etiology. The independent pathways model fitted substantially less well ($AIC_{Cholesky}$ = 21546.35 vs AIC_{IP} = 21561.16); however, this result may simply reflect the omission of a specific genetic effect common to conduct problems and educational achievement; i.e. the general genetic factor forces any link between age 4 conduct problems and educational achievement to also explain genetic influences on peer problems and anxiety. Indeed, a modified independent pathway model including this parameter provided a more parsimonious fit to the data than the Cholesky ($AIC_{Cholesky}$ = 21546.35 vs AIC_{IPMod} = 21539.22: see Supplementary Figure 2). In aggregate, this set of analyses indicates that while conduct problems, anxiety, and peer problems (at age 4) are all shared-environment predictors of age 16 educational achievement, these environmental factors reflect generalized rather than specific sources of prediction.

----- Insert Figures 3-5 here -----

Discussion

The current study examined the association between child and adolescent behavior problems and educational achievement at age 16. At the phenotypic level, anxiety, conduct problems, hyperactivity and peer problems (as rated by parents) at age 4 all predicted lower levels of educational achievement at age 16, although the magnitude of these predictions was modest for anxiety and peer problems. For each of these variables incremental prediction for educational achievement was observed at the subsequent measurement points. Prosociality

positively predicted educational achievement from age 12, with incremental prediction at age 16. These results support previous findings reporting early-childhood links from externalizing to school success (Fergusson & Horwood, 1998; Vitaro et al., 2005), as well as help to clarify the role of internalizing behaviors and prosociality on educational achievement in light of mixed findings in the literature (Caprara et al., 2000; DiLalla et al., 2004; Kokko et al., 2006; van Lier et al., 2012).

The etiology (i.e. genetic and environmental underpinnings) of the association for the links between early childhood behavior problems and later educational achievement was largely specific to each of the behavior problems. The link between early-childhood conduct problems and later educational achievement was explained by genetic and shared-environmental factors. The link between early-childhood hyperactivity and later educational achievement was also explained in part by genetic factors, but here nonshared-environmental factors accounted for the remainder of the association. This result is notable in light of the rarity of nonshared-environmental stability over time (e.g. Burt, Klahr, & Klump, 2015; Turkheimer & Waldron, 2000; although see Livingstone et al., 2016). The link between early-childhood anxiety and peer problems and later educational achievement was explained by shared-environmental factors. Of note, the genetic influences linking conduct problems and hyperactivity with educational achievement were themselves substantially overlapping. Similarly, the shared-environmental contribution from conduct problems to educational achievement overlapped substantially with that of anxiety and peer problems. These findings, particularly the observations for the externalizing problems, are consistent with the notion that early-emerging behavior problems reflect longstanding challenges to life outcomes (Moffitt, 1993), here exemplified by the important life variable of educational achievement.

For both conduct problems and hyperactivity we also found evidence for genetic and nonshared-environment innovations that emerged at ages 12 and 16 and provided incremental

prediction for educational achievement. These results are consistent (particularly in the context of genetic innovation) with the notion of adolescence as a sensitive period of socio-affective development with implications in turn for educational outcomes (Blakemore, 2010). In contrast, the associations from anxiety and peer problems to educational achievement did not show genetic or environmental innovations, instead being wholly accounted for by early emerging shared-environment effects.

These findings raise some intriguing questions. Firstly, what processes might explain the shared-environmental influences acting on age 4 conduct problems, anxiety, and peer problems, which in turn impact on later educational achievement? Some possible factors might include parental warmth/support (Roelofs, Meesters, ter Huurne, Bamelis, & Muris, 2006), parental control (Barber, 1996), or family chaos (Hanscombe, Haworth, Davis, Jaffee, & Plomin, 2011). Low levels of parental concern for the welfare and outcomes of the child might plausibly manifest in conduct problems, anxiety, and peer problems, and in turn impact educational outcomes, either directly – e.g. through limited shared book reading or interaction – or indirectly – e.g. as a consequence of behavior problems impairing learning opportunities. Broader shared experiences beyond the home – such as preschool quality or neighbourhood-level deprivation (Caspi, Taylor, Moffitt, & Plomin, 2000; Reijneveld, Brugman, Verhulst, & Verloove-Vanhorick, 2005) – might similarly explain this pattern of shared-environmental effects.

Secondly, what processes might explain the genetic influences common between externalizing problems (i.e. conduct problems and hyperactivity) and educational achievement? One possibility is individual differences in executive functioning, particularly in the context of emotion and impulse management. Such mechanisms are likely to have direct effects on the expression of externalizing behaviors (Barkley, 1997; Moffitt, 1993; Morgan & Lilienfeld, 2000) and may also impair educational development through failures to

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persevere when the workload becomes difficult, or indirectly as a consequence of exclusion from class activities due to poor behavior.

A number of limitations require discussion. Firstly, the classical twin design is subject to a number of assumptions, such as the equal environments assumption (Plomin et al., 2013). Future studies that can capitalize on additional family structures in order to provide more assumption-free estimates would be valuable, although it is noteworthy that research testing whether violations of the equal environments assumption are apparent for psychopathology has found little evidence for this potential source of bias (Kendler, Neale, Kessler, Heath, & Eaves, 1993). Secondly, with the current study design we cannot draw inferences concerning the genetic and environmental mechanisms underpinning the observed links between early-childhood behavioral problems and later educational achievement. For instance, these traits may exert their influence on subsequent school success through the initiation of a deleterious developmental cascade (i.e. bad school behavior early on leads to poor skill development, with the associated knock-on effects for subsequent intellectual development) or because of stable influences that act more proximately. Thirdly, although Cronbach's alpha was consistent with previous work (e.g. Goodman, 2001), these values fell below conventional standards for conduct problems (all ages) and for peer problems (age 4). It is noteworthy, however, that some debate exists over whether modest Cronbach's alpha values signal need for concern. If one uses a broad content coverage and quickly administrable instrument with just a few items per scale, as is the case with the SDQ instrument, one should expect, and perhaps even desire, such 'modest' alphas (e.g., Boyle, 1991).

In summary, in the current study we have shown that genetic, shared-environmental, and (to a lesser extent) nonshared-environmental influences on behavior problems in early childhood are predictive of educational achievement in major public examinations at age 16, consistent with work emphasising life-course persistence of behavior problems and the

concomitant negative life outcomes. Of importance, we also observed that new genetic and nonshared-environmental influences – that is, genetic influences on conduct problems and hyperactivity emerging during childhood and adolescent development – were also predictive of educational achievement at 16, consistent with the notion that adolescence represents a sensitive period for socio-affective development.

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Figure 1. Graphical representation of the longitudinal Cholesky decomposition for SDQ traits and educational achievement.
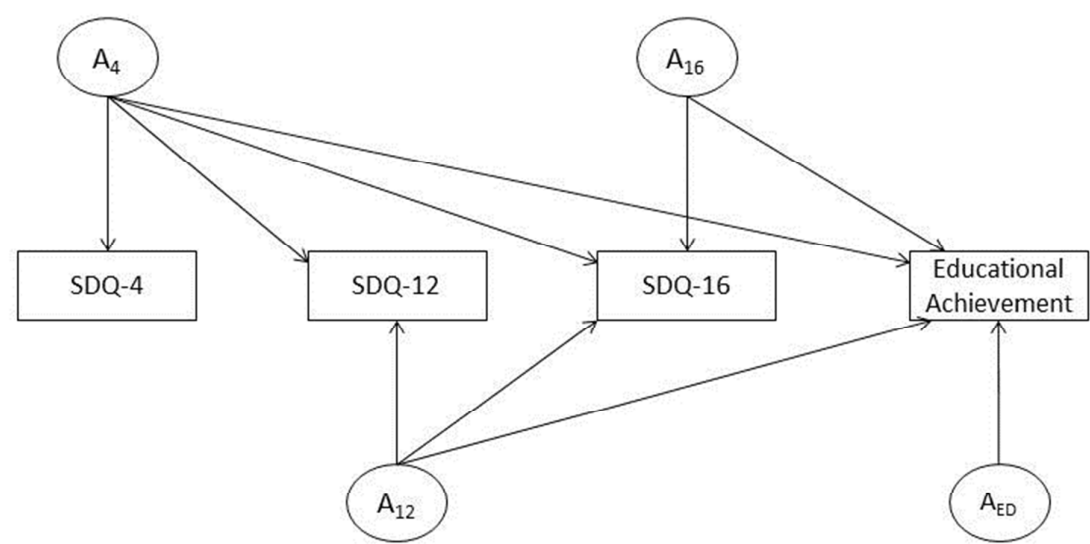
Note. A = additive genetic influences; shared- and nonshared-environmental influences were also modelled, and took the same form as the A pathways (i.e. C_4, C_{12}, C_{16}, E_4, E_{12}, and E_{16}), but are omitted here in the interests of visual clarity; SDQ-16 was only available for conduct, hyperactivity, and prosociality.

Figure 2. Phenotypic Cholesky decomposition modelling results for SDQ traits and educational achievement.
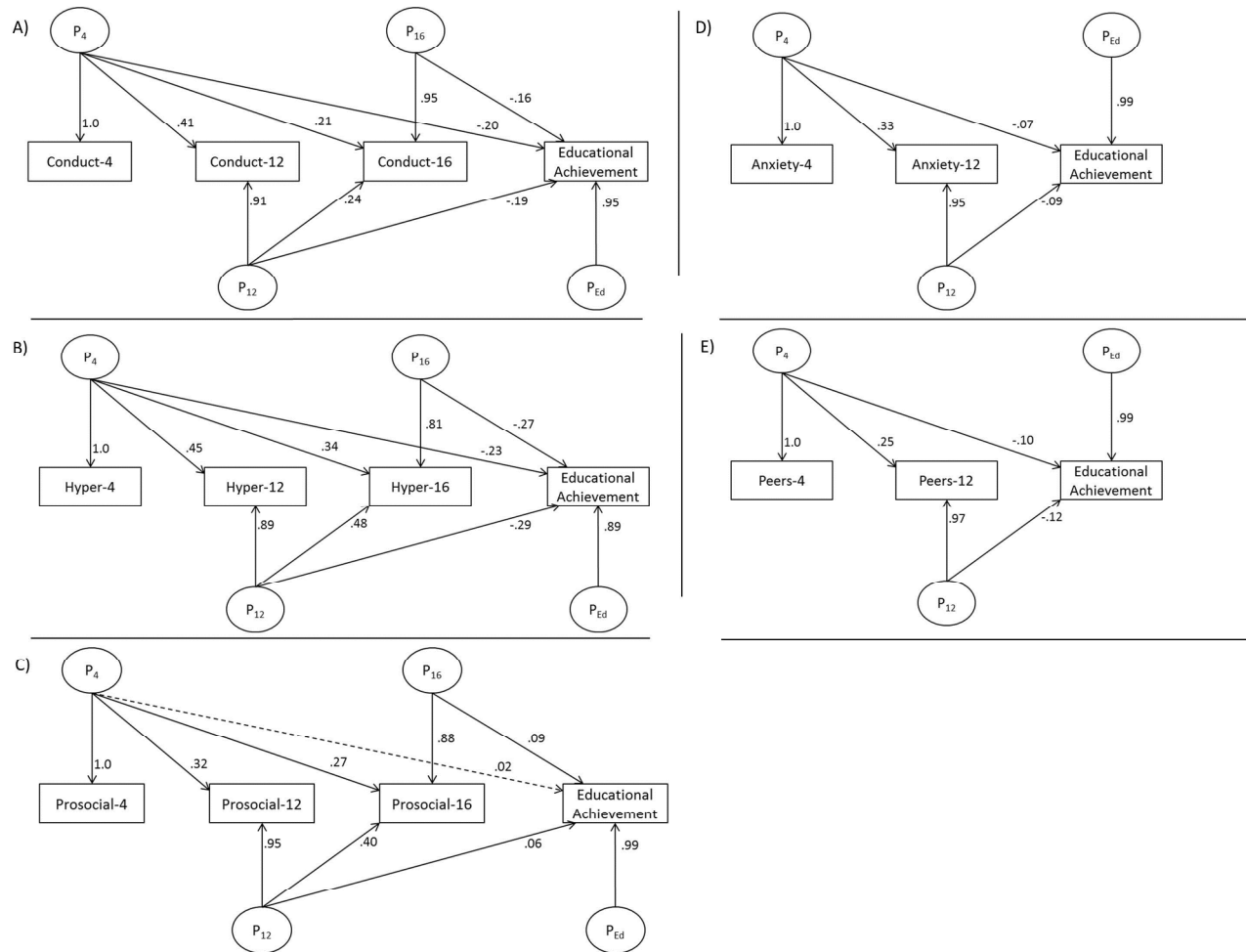
Note. Bolded lines = p < .05; P = phenotypic effects; 4/12/16 = age 4/12/16; values are standardized path loadings.

Figure 3. Longitudinal additive genetic modelling results for SDQ traits and educational achievement.
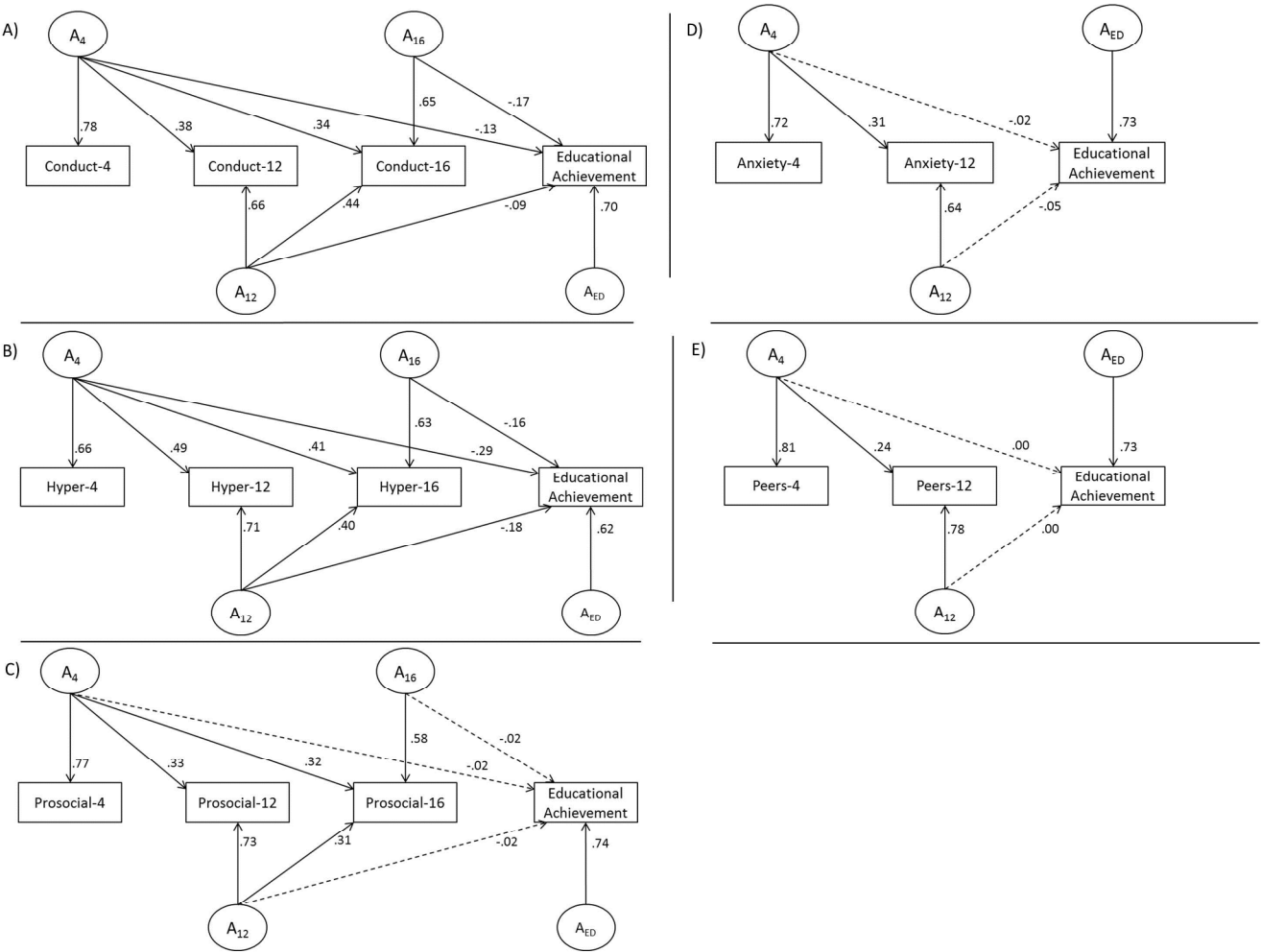
Note. Bolded lines = p < .05; A = additive genetic effects; 4/12/16 = age 4/12/16; values are standardized path loadings.

Figure 4. Longitudinal shared-environment modelling results for SDQ traits and educational achievement.
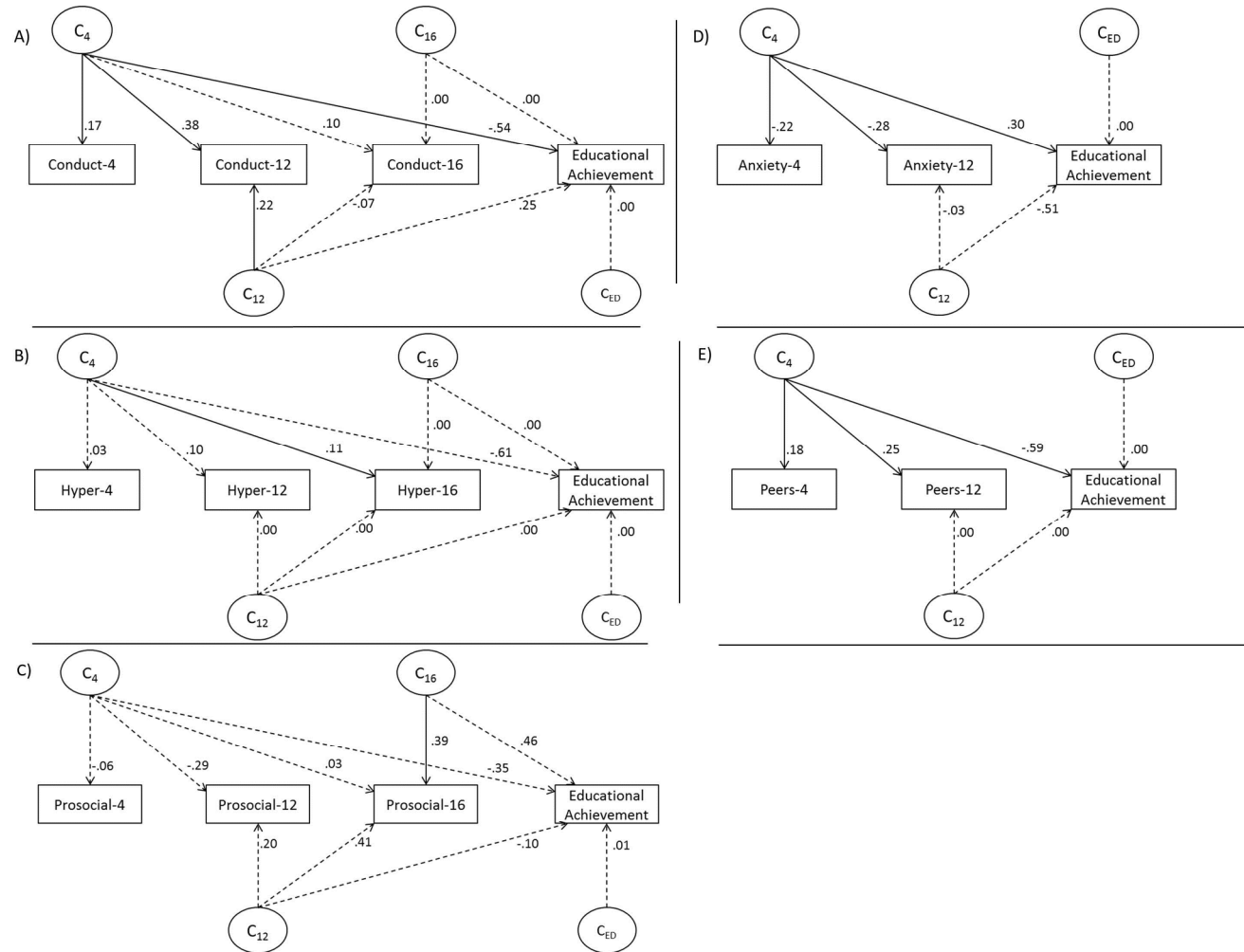
Note. Bolded lines = p < .05; C = shared-environment effects; 4/12/16 = age 4/12/16; values are standardized path loadings.

Figure 5. Longitudinal nonshared-environment modelling results for SDQ traits and educational achievement.

Note. Bolded lines = p < .05; E = nonshared-environment effects; 4/12/16 = age 4/12/16; values are standardized path loadings.

Table 1. *Descriptive statistics for SDQ sub-scales and Educational Achievement.*

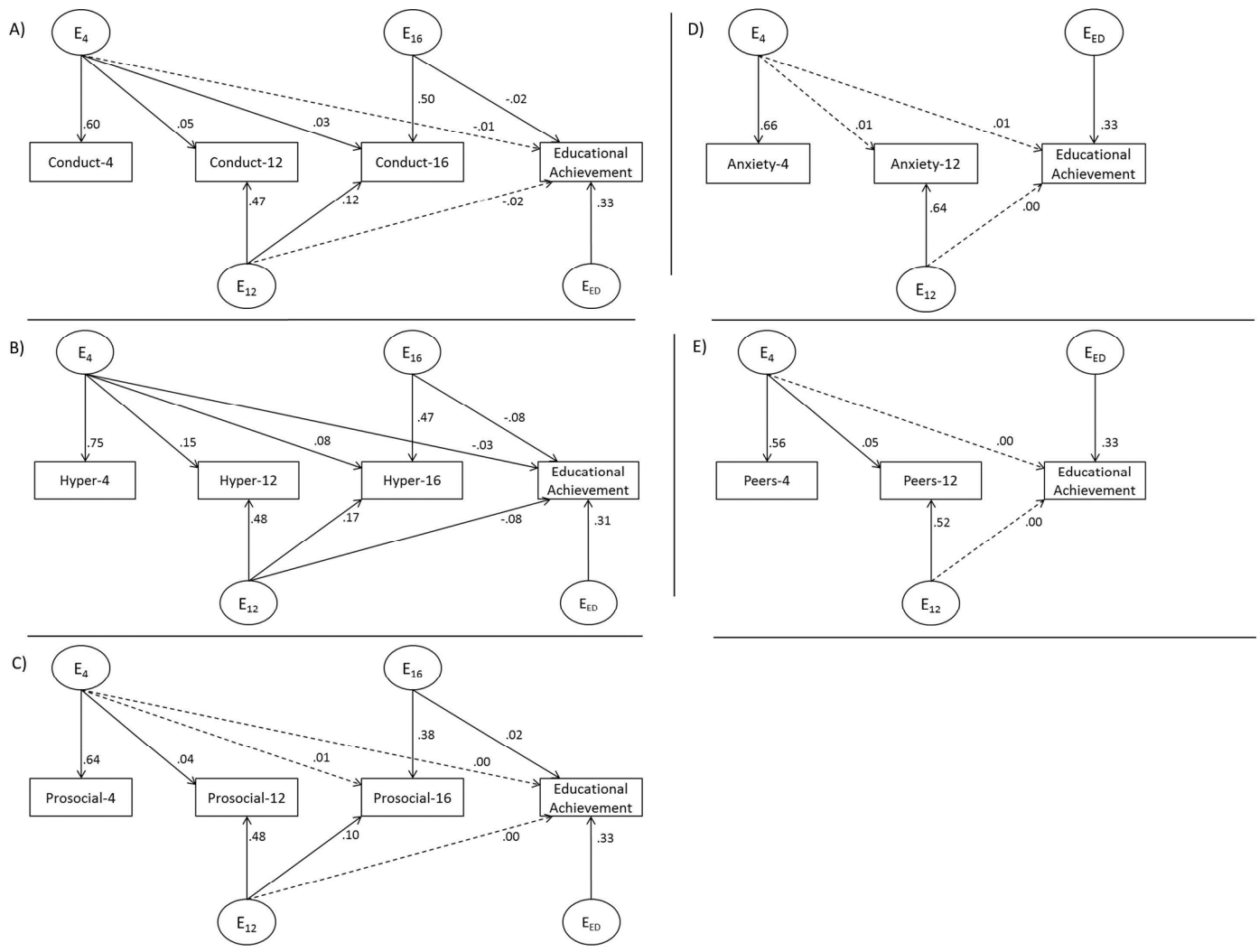| Measure	α	MZm M (SD)	MZf M (SD)	DZm M (SD)	DZf M (SD)	DZosm M (SD)	DZosf M (SD)
SDQ (age 4)							
Anxiety	.60	1.28 (1.35)	1.41 (1.43)	1.39 (1.44)	1.49 (1.49)	1.33 (1.40)	1.32 (1.43)
Conduct	.54	2.26 (1.58)	1.91 (1.46)	2.27 (1.60)	1.97 (1.54)	2.16 (1.60)	1.88 (1.50)
Hyperactivity	.76	4.37 (2.24)	3.74 (2.07)	4.21 (2.44)	3.77 (2.36)	4.38 (2.36)	3.32 (2.18)
Peer problems	.47	1.40 (1.41)	1.23 (1.34)	1.70 (1.57)	1.45 (1.48)	1.63 (1.57)	1.37 (1.42)
Prosociality	.69	7.04 (1.84)	7.56 (1.78)	7.11 (1.90)	7.61 (1.81)	7.09 (1.92)	7.71 (1.77)
SDQ (age 12)							
Anxiety	.68	1.65 (1.80)	1.92 (1.97)	1.65 (1.84)	1.93 (1.94)	1.65 (1.86)	1.89 (1.97)
Conduct	.57	1.43 (1.45)	1.16 (1.34)	1.49 (1.56)	1.21 (1.41)	1.40 (1.53)	1.23 (1.40)
Hyperactivity	.77	3.35 (2.25)	2.29 (1.96)	3.23 (2.39)	2.50 (2.13)	3.48 (2.49)	2.14 (1.87)
Peer problems	.64	1.11 (1.50)	.88 (1.28)	1.23 (1.64)	1.04 (1.44)	1.27 (1.66)	0.96 (1.36)
Prosociality	.67	8.31 (1.72)	8.83 (1.50)	8.20 (1.74)	8.84 (1.49)	8.28 (1.73)	8.78 (1.49)
SDQ (age 16)							
Conduct	.52	1.16 (1.31)	1.12 (1.28)	1.25 (1.34)	1.23 (1.43)	1.26 (1.45)	1.16 (1.35)
Hyperactivity	.71	2.45 (2.01)	1.88 (1.65)	2.53 (2.03)	2.06 (1.99)	2.78 (2.19)	1.84 (1.71)
Prosociality	.73	8.00 (1.95)	8.53 (1.86)	7.91 (2.01)	8.56 (1.78)	7.93 (2.01)	8.46 (1.81)
Educational Achievement	-	8.75 (1.20)	8.99 (1.14)	8.81 (1.16)	8.99 (1.18)	8.73 (1.22)	9.03 (1.14)

Notes: M = mean (SD = standard deviation). α = Cronbach's alpha for scale scores collapsed across sex and zygosity. MZ = monozygotic; DZ = dizygotic; m = male; f = female; os = opposite sex; higher scores indicate greater difficulties (i.e. anxiety, conduct problems, hyperactivity, peer problems) or strengths (i.e. prosociality).

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Supplementary Material

Supplementary Table 1. Number of complete twin pairs across zygosity and sex for all study measures.

	Anxiety	Conduct	Hyper	Peers	Prosoc	Education
Age 4						
MZM	1165	1166	1166	1163	1166	-
MZF	1350	1350	1348	1348	1350	-
DZM	1193	1196	1195	1193	1196	-
DZF	1247	1247	1245	1245	1247	-
DZOS	2344	2350	2345	2345	2352	-
Age 12						
MZM	912	911	912	912	913	-
MZF	1130	1130	1130	1130	1132	-
DZM	844	845	845	845	847	-
DZF	968	968	968	968	973	-
DZOS	1742	1742	1741	1741	1744	-
Age 16						
MZM	-	722	720	-	721	1012
MZF	-	1029	1028	-	1028	1253
DZM	-	670	670	-	670	948
DZF	-	889	886	-	888	1059
DZOS	-	1517	1513	-	1514	1991

Note. MZ = monozygotic; DZ = dizygotic; M = male; F = female; OS = opposite –sex; conduct = conduct problems; hyper = hyperactivity; peers = peer problems; prosoc = prosociality; education = educational achievement

Supplementary Table 2. Phenotypic correlations between SDQ variables and educational achievement

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1. Anxiety$_{Age4}$	-												
2. Conduct$_{Age4}$	.26	-											
3. Hyper$_{Age4}$	.18	.42	-										
4. Peers$_{Age4}$	.29	.23	.14	-									
5. Prosoc$_{Age4}$	-.05	-.31	-.24	-.22	-								
6. Anxiety$_{Age12}$	.31	.18	.12	.17	-.04	-							
7. Conduct$_{Age12}$	.17	.40	.25	.14	-.14	.31	-						
8. Hyper$_{Age12}$	.14	.31	.44	.16	-.15	.26	.47	-					
9. Peers$_{Age12}$	.16	.18	.15	.24	-.08	.36	.31	.28	-				
10. Prosoc$_{Age12}$	-.06	-.16	-.13	-.18	.33	-.07	-.31	-.26	-.18	-			
11. Conduct$_{Age16}$	.11	.29	.19	.10	-.09	.20	.49	.33	.19	-.20	-		
12. Hyper$_{Age16}$	.14	.25	.32	.15	-.10	.20	.38	.55	.20	-.189	.53	-	
13. Prosoc$_{Age16}$	-.04	-.17	-.12	-.16	.27	-.09	-.27	-.22	-.15	.45	-.40	-.33	-
14. Education	-.06	-.19	-.23	-.09	.02	-.10	-.24	-.35	-.14	.06	-.27	-.41	.11

Note. Correlations are taken from one individual in each twin pair; conduct = conduct problems; hyper = hyperactivity; peers = peer problems; prosoc = prosociality; education = educational achievement; all coefficient's > .03 are $p < .01$; n_{range} = 3854-7382.

Supplementary Table 3. Cross-sectional twin analysis results for SDQ variables

Variable	A	C	E
Anxiety$_{Age4}$	**.58**	.00	**.42**
Conduct problems$_{Age4}$	**.64**	.00	**.36**
Hyperactivity$_{Age4}$	**.42**	.00	**.58**
Peer problems$_{Age4}$	**.67**	.01	**.31**
Prosociality$_{Age4}$	**.59**	.00	**.41**
Anxiety$_{Age12}$	**.53**	.05	**.41**
Conduct problems$_{Age12}$	**.59**	**.18**	.23
Hyperactivity$_{Age12}$	**.74**	.00	.25
Peer problems$_{Age12}$	**.72**	.00	.27
Prosociality$_{Age12}$	**.64**	.12	.24
Conduct problems$_{Age16}$	**.74**	.00	.27
Hyperactivity$_{Age16}$	**.74**	.00	.26
Prosociality$_{Age16}$	**.53**	**.32**	**.15**

Note. A = additive genetic effects; C = shared-environment effects; E = nonshared-environment effects; bolded = p < .05.

Supplementary Table 4. Correlations between SDQ variables and educational achievement for MZ twin pairs

|	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Anxiety$_{Age4}$	.56													
2. Conduct$_{Age4}$	.26	.64												
3. Hyper$_{Age4}$	.21	.33	.52											
4. Peers$_{Age4}$	.19	.21	.22	.65										
5. Prosoc$_{Age4}$	-.05	-.15	-.17	-.20	.59									
6. Anxiety$_{Age12}$	.26	.16	.10	.10	-.02	.59								
7. Conduct$_{Age12}$	.15	.36	.23	.12	-.12	.30	.76							
8. Hyper$_{Age12}$	.15	.30	.33	.17	-.14	.26	.45	.75						
9. Peers$_{Age12}$	.11	.21	.16	.17	-.07	.29	.27	.28	.70					
10. Prosoc$_{Age12}$	-.05	-.14	-.15	-.14	.27	-.13	-.28	-.29	-.20	.77				
11. Conduct$_{Age16}$	.11	.25	.19	.12	-.09	.17	.37	.32	.14	-.17	.71			
12. Hyper$_{Age16}$	.12	.22	.24	.14	-.12	.17	.36	.46	.18	-.19	.42	.73		
13. Prosoc$_{Age16}$	-.04	-.16	-.18	-.17	.25	-.10	-.30	-.30	-.15	.43	-.35	-.35	.85	
14. Education	-.07	-.20	-.20	-.07	.03	-.10	-.24	-.32	-.15	.10	-.23	-.35	.10	.89

Note. conduct = conduct problems; hyper = hyperactivity; peers = peer problems; prosoc = prosociality; education = educational achievement; all coefficient's > .07 are $p < .01$; n_{range} = 1419-2516.

Supplementary Table 5. Correlations between SDQ variables and educational achievement for DZ twin pairs

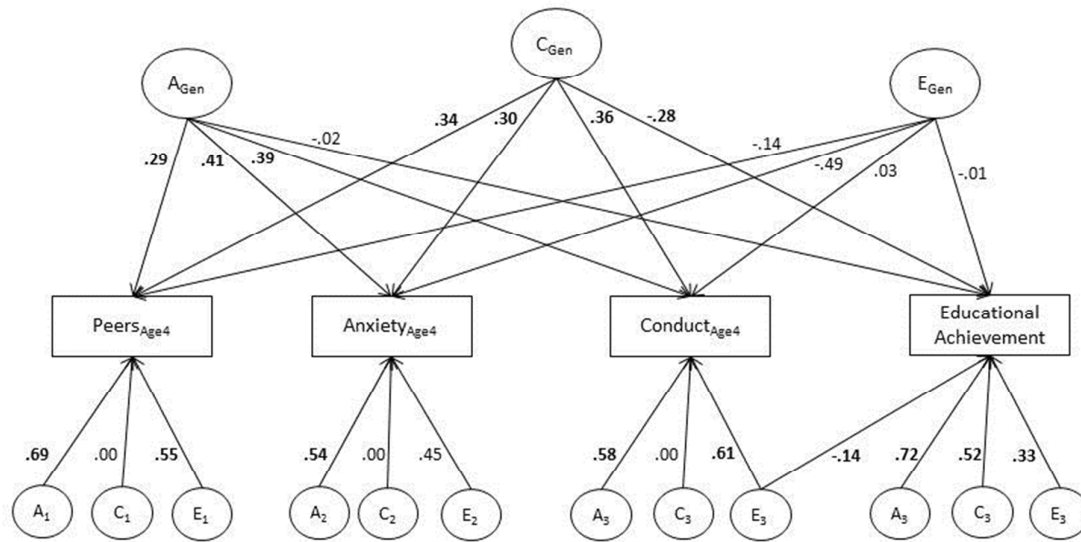|	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Anxiety$_{Age4}$	.31													
2. Conduct$_{Age4}$	.22	.34												
3. Hyper$_{Age4}$	.16	.16	-.05											
4. Peers$_{Age4}$	.16	.23	.17	.36										
5. Prosoc$_{Age4}$	-.04	-.06	-.07	-.12	.32									
6. Anxiety$_{Age12}$	.19	.15	.09	.13	-.05	.33								
7. Conduct$_{Age12}$	.16	.24	.12	.17	-.09	.22	.50							
8. Hyper$_{Age12}$	.13	.17	.05	.16	-.08	.22	.23	.23						
9. Peers$_{Age12}$	.16	.15	.07	.18	-.05	.20	.24	.18	.38					
10. Prosoc$_{Age12}$	-.06	-.12	-.12	-.12	.17	-.10	-.21	-.21	-.16	.46				
11. Conduct$_{Age16}$	.11	.14	.09	.10	-.03	.15	.20	.12	.13	-.11	.38			
12. Hyper$_{Age16}$	.09	.13	.06	.11	-.04	.14	.16	.10	.17	-.15	.23	.25		
13. Prosoc$_{Age16}$	-.05	-.09	-.07	-.11	.14	-.09	-.17	-.15	-.09	.22	-.20	-.23	.58	
14. Education	-.08	-.13	-.07	-.09	.01	-.08	-.18	-.13	-.13	.05	-.18	-.17	.10	.58

Note. conduct = conduct problems; hyper = hyperactivity; peers = peer problems; prosoc = prosociality; education = educational achievement; all coefficient's > .05 or < -.05 are $p < .01$; n_{range} = 2433-4795.

Supplementary Figure 1. Independent pathway model for conduct problems (age 4), hyperactivity (age 4), and educational achievement (age 16).
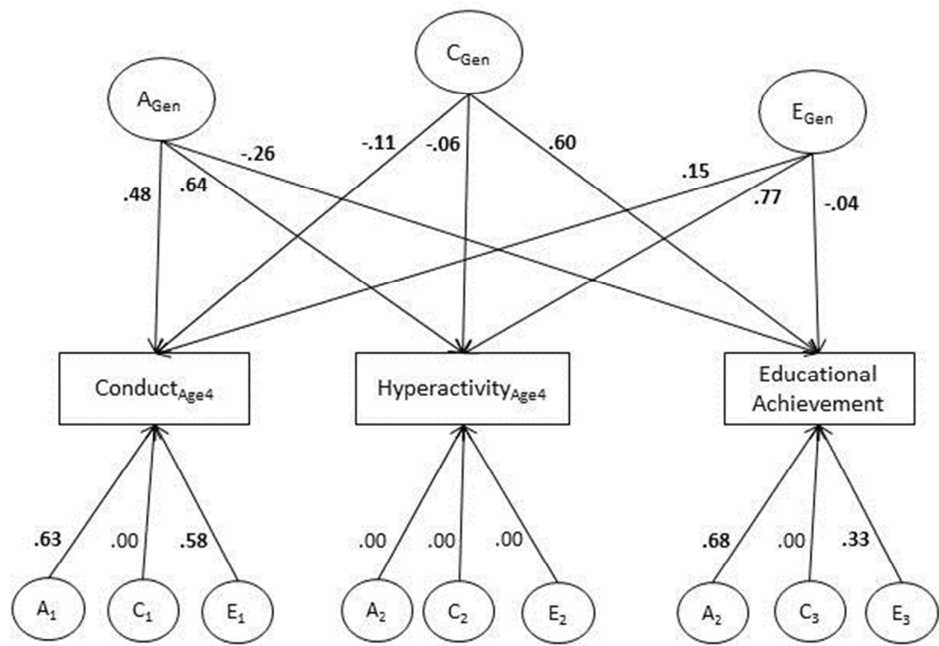
Note. A = additive genetic effects; C = shared-environment effects; E = nonshared-environment effects; gene = general factor; bolded = p < .05.

Supplementary Figure 2. Modified independent pathway model for peer problems (age 4), anxiety (age 4), conduct problems (age 4), and educational achievement (age 16).

Note. A = additive genetic effects; C = shared-environment effects; E = nonshared-environment effects; gene = general factor; bolded = p < .05.