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Hyperactive-impulsive symptom scores and oppositional behaviours reflect alternate manifestations of a single liability

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Abstract

Attention deficit hyperactivity disorder (ADHD) and oppositional behaviours frequently co-occur, We aimed to study the etiology of this overlap in a general population–based twin sample, assessing the symptom domains of hyperactivity-impulsivity and inattentiveness separately for their overlap with oppositionality. We further aimed to investigate whether rater bias may contribute to the overlap in previous data which used one rater only. Using parent and teacher ratings on hyperactivity-impulsivity, inattentiveness and oppositionality, and actigraph measurements of activity level, for 668 7-9-year-old twin pairs, oppositionality showed a higher overlap with hyperactivity-impulsivity (r=.95) than with inattentiveness (r=.52) and all etiological influences on hyperactivity-impulsivity were shared with those on oppositionality, indicated by a genetic correlation of .95 and a child-specific environmental correlation of .94. Actigraph data did not show an overlap with ratings of oppositionality. In middle childhood, symptoms of hyperactivity-impulsivity and oppositional behaviour may represent the same underlying liability, whereas the inattentive domain is more distinct.

Keywords

ADHD; oppositionality; twin study; actigraph; heritability; co morbidity

INTRODUCTION

An important clinical feature of attention deficit hyperactivity disorder (ADHD) is the high rate of comorbid behavioural problems, including oppositional defiant disorder (ODD) and conduct disorder (CD). Comorbidity is observed whether considering clinical samples, where 30-60% of children diagnosed with ADHD obtain also a diagnosis of CD (Abramowitz, Kosson, & Seidenberg, 2004), or symptoms of ADHD and ODD or CD in the general population (see Jensen, Martin, & Cantwell, 1997 for a review). In the recent collection of combined type ADHD cases by the International Multi-centre Genetics (IMAGE) project from clinical centres in eight European countries CD was found in 22.8% of cases (365/1602) and ODD in 58.8% (942/1602) (Asherson, personal communication). The British Child and Adolescent Mental Health Survey that used population survey data, found CD in 6.7% of ADHD cases and ODD in 10.1% (Ford, Goodman & Meltzer, 2003).

The strength of the association between ADHD and co-occurring oppositional behaviours has raised the question as to whether a real distinction can be made between the two behavioural domains (Biederman, Newcorn, & Sprich, 1991). Some authors suggest that

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ADHD and oppositionality may reflect a single underlying disorder with similar genetic and environmental risk factors, which differ only in terms of surface manifestations (Daugherty & Quay, 1991; Daugherty, Quay, & Ramos, 1993; Szatmari et al, 1990), while other data suggest separate but correlated behavioural disorders (Banaschewski et al., 2003; Jensen, 2001; Rubia et al., 2008). Longitudinal studies indicate that ADHD usually comes first and can lead to the development of oppositional behaviours, whereas oppositionality does not occur as a precursor of ADHD (Burns & Walsh, 2002; McArdle, O'Brien, & Kolvin, 1995; Taylor et al, 1996).

One approach used to delineate the aetiological relationship between ADHD and oppositionality is the use of twin study designs, which examine the extent to which the behaviours have shared or unique genetic and environmental influences. Previous twin studies of large general population samples indicate a shared genetic etiology between symptoms of ADHD and ODD, with much of the covariation between the two behaviours resulting from shared genetic effects (Nadder et al 2002). Similar results were found between ADHD and DSM-IV symptoms counts of CD in the general population (Young, Stallings, Corley, Krauter & Hewitt, 2000). However, behaviour-specific variance components are also reported for ODD symptoms (Dick et al, 2005; Nadder et al, 2002), with evidence pointing towards behaviour-specific environmental contributions to oppositionality but not to ADHD (Burt et al, 2001; Silberg et al., 1996; Thapar, Harrington, & McGuffin, 2001).

However, these data raise several questions. First, the use of one informant for assessment of both ADHD and oppositional behaviour symptoms in some studies may have inflated the estimated effect of genetic influences, as the ratings of each behaviour reflect aspects of the rater as well as of the child. For example, self ratings alone (Dick et al, 2005) or mother ratings alone (Thapar, Harrington, & McGuffin, 2001) have been used. Another study that combined mother ratings and child self-report of ADHD and DSM-III symptoms of ODD resulted in the covariation being attributed to environmental rather than genetic influences, an entirely different conclusion (Burt et al, 2005). A further study that used mother and teacher reports of ADHD and oppositionality found only low covariation cross-behaviour cross-informant, when compared to the covariation across behaviours rated by the same informant, although in both cases the covariation between the behaviours was attributable to genetic influences (Nadder et al, 2002).

A second issue is that previous studies have frequently combined hyperactivity-impulsivity and inattentiveness when examining the relationship between symptoms of ADHD and symptoms of ODD (Burt et al, 2001; Dick et al, 2005; Thapar, Harrington, & McGuffin, 2001) and CD (Young et al, 2000). Yet this may not be the best approach, since the two behavioural domains may reflect at least partly separate dimensions, with both overlapping and domain-specific genetic effects (Larsson, Lichtenstein, & Larsson, 2006; McLoughlin, et al, 2007). Some epidemiological evidence suggests a stronger phenotypic association between hyperactivity-impulsivity and oppositionality (Newcorn et al., 2001; Warner-Rogers et al, 2000), although a population twin study found similar correlations between oppositionality and mother-rated hyperactivity and mother-rated inattention at two separate time points (Nadder et al, 2002).

To address these questions further, this study investigated the phenotypic and etiological overlap between ADHD and oppositional behaviour in middle childhood, using a sample of 668 twin pairs between 7 and 9 years of age. Specifically, we aimed to: (1) use ratings from both parents and teachers to examine the overlap between ADHD and oppositionality; (2) examine whether rater bias contributes to the overlap between ADHD and oppositionality, when data are obtained from one rater only and (3) assess the symptom domains of

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hyperactivity-impulsivity and inattentiveness separately for their overlap with oppositionality.

Finally, our fourth aim was to use actigraph data to establish whether this aids the separation of symptoms of over-activity from those of oppositionality. Actigraphs measure movement in an objective and quantifiable way, which may help to separate over-activity from other aspects of externalising behaviour. In a previous study we found that actigraph data have good reliability (Wood, Kuntsi, Asherson, & Saudino, 2008) and show a similar degree of covariation with parent and teacher ratings of hyperactive-impulsive symptoms as the inter-rater agreement between parents and teachers on these behaviours (Saudino et al, 2004; Wood et al, 2008), making actigraph data a useful additional source of activity level measurement.

METHODS AND MATERIALS

Sample

Participants are members of the Study of Activity and Impulsivity Levels in children (SAIL), a general population sample of twins at age 7 to 9 years. The sample was recruited from a birth cohort study, the Twins' Early Development Study (TEDS; Trouton, Spinath, & Plomin, 2002), which had invited parents of all twins born in England and Wales during 1994-1996 to enroll. Despite attrition, the TEDS families continue to be fairly representative of the UK population with respect to parental occupation, education and ethnicity (Spinath & O'Connor, 2003). Zygosity has been determined using a standard zygosity questionnaire, which has been shown to have 95% accuracy when compared to zygosity status determined by genotype data (Price et al., 2000).

Families on the TEDS register were invited to take part if they fulfilled the following SAIL project inclusion criteria: twins' birthdates between 1st September 1995 and 31st December 1996; lived within feasible traveling distance of the Research Centre (return day trip); ethnic origin white European (to reduce population heterogeneity for molecular genetic studies); recent participation in TEDS, as indicated by return of questionnaires at either 4- or 7-year data collection point; no extreme pregnancy or perinatal difficulties (15 pairs excluded), specific medical syndromes, chromosomal anomalies (two pairs excluded) or epilepsy (one pair excluded); not participating in other current TEDS sub-studies (45 pairs excluded); and not on stimulant or other neuropsychiatric medications (two pairs excluded).

Of the 1,230 suitable families on the register whom we contacted, 672 families agreed to participate, reflecting a participation rate of 55%. Overall, the sample is as representative of the general population as is feasible for a study of this kind, and previous analyses on TEDS indicates that attrition in the TEDS sample has not been due to ADHD symptoms. For example, Saudino, Ronald & Plomin (2005) found that twins who participated at age 7 assessments were not significantly different in parent ratings of hyperactivity from lost twins at age 2 ($t=1.77$; $p=.08$). However slight bias towards higher parental occupational classification, compared to the original TEDS sample should be noted (39% of mothers and 52% of fathers in managerial or professional jobs, compared to 28% and 40%, respectively). Thirty individual children were subsequently excluded (sixteen children with IQs below 70, three children due to epilepsy, three children due to mild autism, two with obsessive-compulsive disorder, and one child due to each of the following: neurofibromatosis, hyperthyroidism, dyspraxia, severe autism, sickness on day of testing and on stimulant medication for ADHD).

This leaves a final sample of 1314 children. All participants were invited to a research centre for cognitive assessment see (Kuntsi et al., 2006), where Conners' rating scale data by the

parents was collected for the sample. The final sample consisted of 513 identical (monozygotic, MZ) twins (data for 255 complete twin pairs), 374 same-sex non-identical (dizygotic, DZ) twins (data for 184 complete twin pairs) and 427 opposite-sex DZ twins (207 complete twin pairs). The data for the remaining 22 'singleton' twins were also used for model fitting in the structural equation modeling (see Neale & Cardon, 1992). The mean age was 8.83 years (SD = 0.67), with nearly identical proportions of boys (49%) and girls (51%). Parents of all participants have given informed consent and the Institute of Psychiatry Ethical Committee approved the study.

Measures

Ratings of oppositionality, inattentiveness and hyperactivity-impulsivity— Parents were asked to complete the Long Version of the Conners' Parent Rating Scale (CPRS-R:L; Conners et al, 1998a) and teachers the Long Version of the Conners' Teacher Rating Scale (CTRS-R:L; Conners et al, 1998b). Ratings were completed by the primary caregiver, which for the majority was the mother. Teacher data were completed by the main class teacher for each child, previous analyses on the TEDS sample, from which the current subsample is drawn, indicates that the majority of twins are rated by the same teacher (Saudino, Ronald & Plomin, 2005). In a few cases, missing data in Conners' scales were pro-rated (i.e. a summary score based on the mean of individual questions on the rest of the subscale was used), if there was more than 75% completion for each subscale. Items were used from the 9-item DSM-IV inattentive subscale, the 9-item DSM-IV hyperactive-impulsive subscale and the 10- (parent) or 6- (teacher) item oppositional subscale.

Although the oppositional subscales largely include questions that mirror the DSM-IV criteria for ODD, there is one exception from the Parent Rating Scale (CPRS-R:L; Conners et al, 1998a); item no. 2 'Fights', which more directly related to symptoms of Conduct Disorder. Therefore we have avoiding using terms specific to ODD or CD and label the behaviour as 'oppositionality' after the name of the subscale, which here reflects symptoms of mainly ODD in the general population.

Actigraph measurements—Actigraph data were obtained for 486 families. In addition, data from 108 participants were lost due to mechanical failure and data from six participants were subsequently excluded due to difficulties during test sessions that inappropriately affected the data (e.g. playing with the actigraph). These data were considered 'missing at random' and those who lost actigraph data did not differ from those who did not in terms of either parent (t_{1296}=0.28; p=0.78) or teacher (t_{1152}=0.38; p=0.70) ratings of hyperactivity-impulsivity. The families visited the Research Centre for the actigraph assessments (for further details see Wood et al, 2007). Actigraph readings reflecting the cumulative intensity of movement were taken from the dominant leg and waist during two situations: a laboratory-based test session, when the twins were apart completing a short-form IQ test and several experimental tasks, and a 25-minute unstructured break when twins were together. The total length of the testing session, including break, was approximately 2.5 hours.

Previous analyses showed that over the whole session, including the laboratory-based testing and the break, the two actigraph measurements taken on the leg and waist were significantly correlated at r = 0.52 (p<0.001), and the same genetic influences underlay actigraph data in the two situations (Wood et al, 2007). Therefore a mean actigraph score was used, which represents the average cumulative frequency per minute, averaged across limbs and across situations, to give one actigraph measurement per child, over the whole session.

Analyses

The structural equation-modeling program Mx (Neale et al, 2006) was used to conduct the genetic analyses, and estimation of correlations. Models were fitted to age- and sex-regressed residual scores, transformed with the optimized minimal skew command 'lnskew0' within STATA version 9.2 (StataCorp, 2005) which uses a log transformation in conjunction with an optimized constant to reduce the group skew statistic to 0. Participants with incomplete data were included in the analyses as Mx provides a method for handling incomplete data by using raw maximum likelihood estimation, in which a likelihood statistic (−2LL) of the data for each observation is calculated. The fit of nested models is assessed by a likelihood ratio test, where the difference between −2LL of the full and restricted model is chi-square (χ^2) distributed with degrees-of-freedom (df) equal to the difference in estimated parameters of the two models. For comparing non-nested models, the AIC index (χ^2-2df) is used to indicate which model has more support (Williams & Holahan, 1994). A difference in AIC between two models of less than 2 suggests substantial evidence for both models; a difference between 3 and 7 indicates that the higher AIC model has considerably less support; a difference of more than 10 indicates that the higher AIC model is very unlikely (Wagenmakers & Farrell, 2004).

Saturated phenotypic models—In a saturated phenotypic model the data is fully described with the maximum number of free parameters and provides a baseline comparison for subsequent genetic models. This model can be constrained to obtain a smaller set of statistics in accordance with the assumptions of the genetic method, e.g. means and variances within traits and phenotypic correlations across traits are equated across twins in a pair and zygosity groups. This gives, for example, phenotypic correlations representative of the whole sample while taking into account the non-independence of the data (i.e. data of related subjects).

Univariate genetic models—Univariate genetic analyses use twin correlations for each trait, and on the basis that MZ twins share 100% of their segregating alleles, DZ twins 50% of additive genetic influences and 25% of non-additive genetic influences, partition the phenotypic variance of the measures into additive genetic (A), dominance (D) or shared environmental (C), and child-specific environmental (E) effects, including measurement error (Rijsdijk & Sham, 2002). In the presence of significant MZ and DZ variance differences, phenotypic interaction effects between siblings (or rater contrast effects) are modeled. Within the univariate model the presence of sex-specific influences on the phenotypes is tested. *Qualitative sex differences* are found where regardless of the magnitude of A, C and E influences underlying males and females, the nature of the influences differs (that is, different genes or different shared environment factors underlie the variance in the trait for males and females). These are tested in models where, in turn, the genetic correlation between male and female twins of DZ opposite sex twin pairs (r_GO) is fixed to .5 and the shared environment correlation between male and female twins of DZ opposite sex twin pairs (r_CO) is fixed to 1.00, as we would expect from same-sex pairs. As there was no evidence of qualitative sex differences underlying any phenotype, model fits testing for this are not presented (but are available from first author).

Variance inequality is found where unstandardised A, D/C and E estimates only differ (but standardized estimates are the same), due to variance differences in the trait distribution between males and females. *Quantitative sex differences* are found where the magnitude of A, C and E influences underlying a trait are significantly different for males and females. To test for these a sex differences model is run, which allows for both scalar (variance inequality) and quantitative differences between males and females. First, the non-scalar model is run where the variances are equated across males and females and compared to the

sex differences model, with a 1-df test of significance. Then the no quantitative differences model is run where the standardised A, C and E parameters are equated across males and females and the fit is compared to the sex differences model with a 2-df test of significance.

Given the increased power to detect small differences in variance components (e.g. the power to detect male / female differences in C is high where heritability for a trait is high; Neale, Roysamb & Jacobsen, 2006), and to correct for multiple testing issues, a p-value of . 01 was adopted. There were neither qualitative nor quantitative sex differences underlying the variance in traits. Although we observed a trend for quantitative sex differences for teacher ratings of oppositionality, parent ratings of hyperactivity-impulsivity and for actigraph data, the differences were small in magnitude (further details are available from first author). Therefore, to maximize power, in the multivariate analyses, no sex differences in variance components were modeled, but for all traits a scaling factor was allowed to account for variance differences between males and females (see Table II).

The univariate modeling is used to inform the multivariate parameter choice (such as the decision to include sex-specific variances), but due to the increased power of the multivariate models (Schmitz, Cherny, & Fulker, 1998), univariate parameter estimates are not presented.

Multivariate genetic models—To decompose the variance shared by raters into additive genetic (A), shared environmental (C) and child-specific environmental (E) variance (which includes possible measurement error), cross-rater cross-twin correlations are used. Genetic effects to the covariance are indicated when MZ cross-rater cross-twin correlations are higher than those of the DZ pairs, and C effects when they are equal.

Bivariate models for oppositionality, inattentiveness and hyperactivity-impulsivity rated by more than one rater

Cholesky model (Figure 1A): A triangular decomposition is used to decompose the variance in each rating into A, C and E influences. The extent to which the shared influences of A, C and E underlying the mothers' ratings also influence the teachers' ratings are estimated. However, as this would give precedence to the latent variables underlying the mothers' ratings, a correlated factor solution of the Cholesky model is presented. This is a mathematically equivalent solution of the triangular decomposition, where the variance in each rating is decomposed into A, C and E influences, and the correlations between variance components for each rater are estimated. This model provides a baseline comparison for subsequent models, as it makes no psychological assumptions regarding covariation between ratings of the same behaviour (Hewitt et al, 1992). The implication is that parents and teachers report on behaviours that are distinct from each other (but may be correlated); for example, because the parents observe situationally-specific behaviours or have different understandings of the behavioural descriptions.

Psychometric model (Figure 1B): The variance in each rating is decomposed into that which is shared between the ratings, i.e. the shared behavioural view, and that which is unique to each rating. For both the shared and unique aspect to each rating, A, C and E influences are estimated. The implication for this model is that parents and teachers are measuring behaviours that have unique aspects, arising out of the same possible reasons as above for the Cholesky, but also that a significant aspect of the behaviours is observed by both parents and teachers. To identify the model with bivariate data, both factor loadings are constrained to be 1 (Neale & Cardon, 1992). Although this constrained psychometric model estimates the same number of parameters as the Cholesky model when using bivariate data, and represents a constrained rotation of the Cholesky (Hewitt et al, 1992), the model can still

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fail due to additional implicit constraints, such as the phenotypic covariance between ratings not exceeding the variance of either rating (see Hewitt et al, 1992 for more details). Due to an equal number of degrees of freedom between the psychometric and Cholesky models, the AIC index is used to compare fit.

Rater bias model (Figure 1C): As in the psychometric model, the variance in each rating is composed into the shared behavioural view. In this model however, the remaining variance is attributed to rater bias and residual error, not represented as a unique (or situational) phenotype. Here the assumption is that parents and teachers rate one, shared and pervasive phenotype, plus error in the form of rater bias and residual error.

Multivariate models for three measures of behaviour, each rated by two raters, and a mechanical assessment of activity level (Figure 2)—Based on the bivariate modeling, the best-fitting model (psychometric / rater bias) for each behaviour is chosen, and the variance component correlations between shared behavioural views are estimated. All latent traits that represent a 'shared behavioural view' of behaviour are modeled with A, C and E influences, as we are again, unlikely to have the power to distinguish between A and D.. However, given univariate twion correltions, it should be noted that A refers to 'broad sense heritability' which may subsume some dominant genetic effects. Actigraph data, a mechanical assessment of activity level, is included with the underlying A, C and E influences, and the correlations between these and those of the shared behavioural view of each of the parent and teacher rated behaviours are estimated.

RESULTS

Principal components analyses on parent and teacher ratings of oppositionality, inattentiveness and hyperactivity-impulsivity

We first carried out a principal components analysis on the individual items that comprise all six subscales. This enabled us to investigate empirically, in the present sample, the extent to which the individual items loaded onto the subscales or, possibly, overlapped across the different subscales. Such possible item overlap would indicate that the subscales do not separate out the domains of behaviour (Burns, 2000). Six components were extracted (Eigenvalues of over 1) and factor loadings examined after an oblimin rotation. For all but one subscale the questions from each subscale loaded neatly onto one main factor, with no substantial item overlap. The only exception was question 10 on the CTRS-L:R which was part of the teacher-rated oppositional behaviour subscale. This loaded equally onto the latent teacher-rated oppositional behaviour latent factor, and onto the teacher-rated inattentive behaviour latent factor. A new summary score for the teacher-rated oppositional behaviour subscale was created without this item. It correlated highly with the original summary score ($r=.98$, $p<.001$) and yielded the same parameter estimates in model fitting analyses. Therefore results are only shown using the original teacher-rated oppositional behavior subscale from the CTRS:R-L to assess teacher ratings of oppositionality (further details are available upon request from the first author).

Phenotypic correlations

The phenotypic and twin correlations are presented in Table I.

Within-rater results—The domains of hyperactivity-impulsivity and inattentiveness showed a similar degree of overlap as reported in previous samples (McLoughlin et al, 2007; Sherman, Iacono, & McGue, 1997) for both parent ratings (r=.58 (95% confidence interval . 54 - .61)) and teacher ratings (r=.58 (.53-.61)). Both raters reported a significantly higher correlation, as indicated by non-overlapping 95% confidence intervals, between

hyperactivity-impulsivity and oppositionality (parents r=.55 (.51-.59); teachers r=.68 (.64-.71)), than between inattentiveness and oppositionality (parents r=.40 (.35 - .45); teachers r=.38 (.33 - .44)).

Between-rater results—For all behaviours inter-rater correlations were in the range of r=.21 to r=.46. Across-behaviour inter-rater correlations ranged from r=..12 to r=.21 and all were significant, as indicated by confidence intervals that do not include a zero.

Genetic analyses

Univariate models—For parent-rated inattentive behaviours, MZ correlations were significantly higher than twice the DZ correlations (Table I), which in the absence of significant MZ and DZ variance differences (p=.31) indicates dominant genetic influences on the traits. For this phenotype only the full ACE sex differences model was a significantly worse fit to the data (χ^2=34.70, df=17, p=.01; Table II), so an ADE model was fit to the data. This was a good fit (χ^2=17.56, df=17, p=.44; Table II). However, fitting an ADE model in multivariate analyses with other phenotypes that best fit an ACE model will decrease estimation of a potential genetic overlap. Since we don't have the power to distinguish between A and D in the first place, we focus on the broad-sense genetic effects by fitting ACE models in the multivariate analyses. This results in the same estimates for overall heritability (A+D) as in the ADE model, but the consequence is usually a drop in fit for the multivariate models (Table II).

Bivariate models—For all behaviours the psychometric model was not a worse fit, when AIC values were compared to the saturated biometric model, as indicated by differences of less than 2 (Table II). The high AIC value for inattentive behaviours is likely to represent the drop in fit resulting from not modeling dominance parameters. For all behaviours the rater bias model represented a drop in fit, as indicated by the higher χ^2 statistic and higher AIC (Table II). This indicates that parents and teachers are assessing unique, or situational phenotypes, not affected by consistent rater bias. Therefore parameters representing psychometric models were fitted to all behaviours in the multivariate model (Figure 2).

Multivariate Psychometric model (Figure 2)

Table III provides results from the full multivariate psychometric model. The 'shared' behavioural views between parents and teachers of all three of the child behaviours were positively correlated. The phenotypic correlation between hyperactivity-impulsivity and inattentiveness (r=.79), when assessed by two raters, was higher than the correlation when assessed by one rater (parents: r=.58 (.54 - .61); teachers r=.57 (.53-.61)). It was not possible to compute 95% confidence intervals around the parameters from the full multivariate psychometric model, due to the computational demands of the model, so it is not possible to comment whether the phenotypic correlation between hyperactivity-impulsivity and inattentiveness is significantly higher when rated by two raters, then when assessed by either parents or teachers alone. The overlap between oppositionality and inattentiveness (r=.59) was lower than that of oppositionality and hyperactivity-impulsivity (r=.94). Actigraph data showed very low correlations with all latent traits of behaviours assessed by two raters (r=.05 - .11). Given the low phenotypic correlation of actigraph data with questionnaire data modeled in this way, other parameters from the model are unlikely to be significant and so are not discussed further.

Genetic etiology within and across the shared view of behaviours—All latent factors showed heritabilities within the expected range, when compared to previous data from one rater (61 - 66%). None showed any large influence of shared environment (0 – 8%), with most of the remaining variance (29 - 60%) being attributable to child-specific

environmental influences, which include possible measurement error. Hyperactivity-impulsivity and inattentiveness showed a genetic correlation (r_G) of .83. To test for significant differences between two genetic correlations, a model in which the two genetic correlations of interest were equated was compared to a model in which they were free, with a 1-df test of significance. The r_G between hyperactivity-impulsivity and oppositionality (r_G =.94) was significantly higher than that of oppositionality and inattentiveness (r=.58; χ^2 =6.77, df=1, p=.01). Child-specific environment correlations were similarly significantly higher between hyperactivity-impulsivity and oppositionality (r=.98) than between inattentiveness and oppositionality (r=.57; χ^2 =15.41, df=1, p<.001).

For both measures of ADHD behaviours (inattentiveness and hyperactivity-impulsivity) the covariation with oppositionality was largely due to shared genes (61-73%), although there was a small role for child-specific environmental influences (which include any measurement error; 20-34%).

DISCUSSION

We used parent and teacher ratings of oppositionality, inattentiveness and hyperactivity-impulsivity, as well as actigraph data, from a general population twin sample aged 7-10 years to examine the overlap between ADHD and oppositionality. Extending previous findings on parent ratings of externalizing behaviours (Hewitt et al, 1992; van der Valk et al, 2001) to include teacher ratings, we found that a psychometric model fit the data well and a model, which specified a consistent rater bias effect, was a worse fit. These findings indicate that parents and teachers assess different but valid phenotypes relating children's oppositional, inattentive and hyperactive-impulsive behaviours, that do not reflect a consistent rater bias. Whether the individual ratings reflect different perceptions of the same behaviours or situationally-specific behaviours from the child cannot be determined within this study design and requires further investigation.

The analysis of hyperactive-impulsive and inattentive behaviours separately showed a higher overlap between hyperactivity-impulsivity and oppositionality (r=.95) than between inattentiveness and oppositionality (r=.56), as expected from previous studies which examined this question (Newcorn et al., 2001; Warner-Rogers et al, 2000). Nearly all of the etiological influences underlying ratings of hyperactivity-impulsivity and oppositionality were the same (r_G=.94, r_E=.98), but only approximately 60% of those underlying inattention and oppositionality were (r_G=.58, r_E=.57). This suggests that the debate over whether ADHD and oppositionality are separate or overlapping behaviours can be further clarified by considering the inattentive and the hyperactive-impulsive parts of the ADHD phenotype as separate domains, with some aspects of the inattentive behaviours being distinct from oppositionality, but the hyperactive-impulsive behaviours being largely indistinguishable from oppositionality. This suggests that the hyperactive-impulsive component of ADHD reflects a different phenotypic expression of the same underlying liability as oppositionality. Although of immediate interest to behaviour geneticists, a report commissioned by the National Institute for Health and Clinical Excellence (NICE) recently highlighted the importance of research such as this for clinical practice (NICE, 2008). It emphasised the need for research which aided understanding of the 'accurate differentiation of ADHD from co-occurring conditions to help refine diagnostic criteria, which currently "may lead to the overlooking of ADHD when it co-exists with another problem" (p.23).

Mechanical assessments of activity level from actigraph data did not show an overlap with oppositionality. This may reflect the case that actigraph data measures only physical activity level, whereas the questionnaire data were designed to assess more complex constructs of behavioural hyperactivity and impulsivity. Previous analyses on the current sample showed

that the domains of hyperactivity and impulsivity did not load onto separate factors in a principal component analysis (Wood, Rijsdijk, Saudino, Asherson, & Kuntsi, 2008) and so were assessed together. However, there is evidence that it is the impulsive domain and not the hyperactive domain, which drives the covariation between hyperactivity-impulsivity and oppositionality. This may be an alternative or additional explanation for the lack of overlap between actigraph data and ratings of oppositionality (Newcorn et al., 2001).

A limitation of the current analyses is that confidence intervals were too computationally intense to calculate, and so we were not able to establish which parameters are significant, although whether correlations differed significantly within a model was tested, as described above. Data on the percentage of twins rated by the same, and by different teachers were not available, and future analyses should examine the effect of this on, in particular, the rater bias model. Further, a larger sample would allow more complex analysis, such an investigation of sex-specific effects underlying the co-variation in phenotypes, and possible reasons underlying, or manifestations of, the shared liabilities (Neale & Kendler, 1995). As such, results should be interpreted in the light of small unaccounted for quantitative sex differences in the etiological influences underlying some phenotypes.

Actigraph data were collected in a laboratory set-up and although the genes underlying actigraph data in structured and free-play settings may be the same, environmental influences may differ (Wood et al, 2007). It may be that phenotypic and genetic correlations with ADHD are greater with free-play settings, such as activity level measured in the home, rather than laboratory-based situations (the latter of which may reflect activity in novel situations; Saudino & Asherson, unpublished data). It has yet to be explored how actigraph data collected in different situations overlap with oppositionality. The use of other objective measures of ADHD behaviours, such as cognitive task data, may also help disentangle the effects of item overlap from true covariation and aid the separation of hyperactivity, inattention and impulsivity. The separation of aspects of the ADHD phenotype from oppositionality may vary at different developmental stages. For example, one study suggested that in middle childhood oppositionality always co-occurs with hyperactivity, whereas in early adolescence oppositionality may appear in the absence of hyperactivity (McArdle, O'Brien, & Kolvin, 1995). The use of longitudinal data would address these further questions. Finally it is not explicitly clear how our results, ascertained on ADHD symptom scores and oppositional behaviours in a general population sample, would compare to those obtained with diagnostic information on ADHD, ODD and / or CP.

Our data indicate that rater effects do not artificially inflate the overlap between ADHD and oppositional behaviours. Further, the overlap is higher, if the hyperactive-impulsive domain is treated separately to the inattentive domain, than if the two domains are considered together. Our analyses indicate that the hyperactive-impulsive behaviours of the ADHD phenotype is shared etiologically and phenotypically with oppositional behaviours in the general population, whereas the inattentive domain is more distinct. Further research should address whether the pattern of these results, found in middle childhood, are reflected later on in life.

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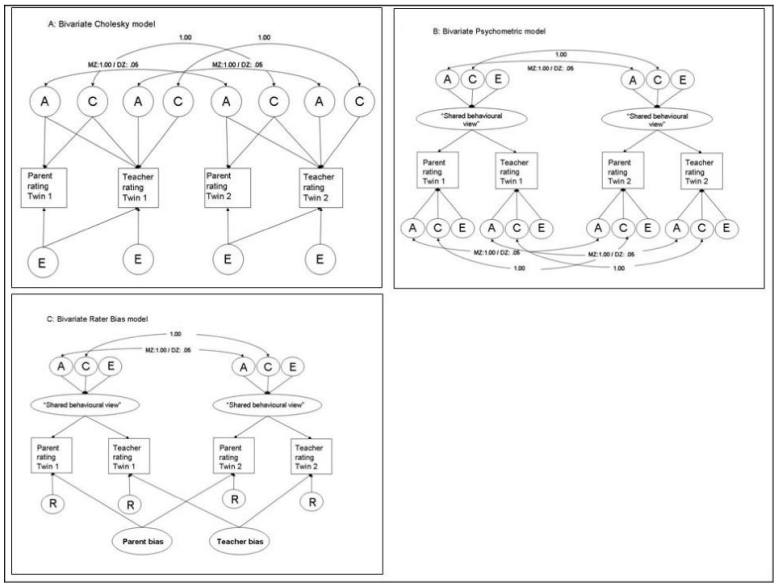

Figure 1.
Bivariate Cholesky, Psychometric and Rater Bias models

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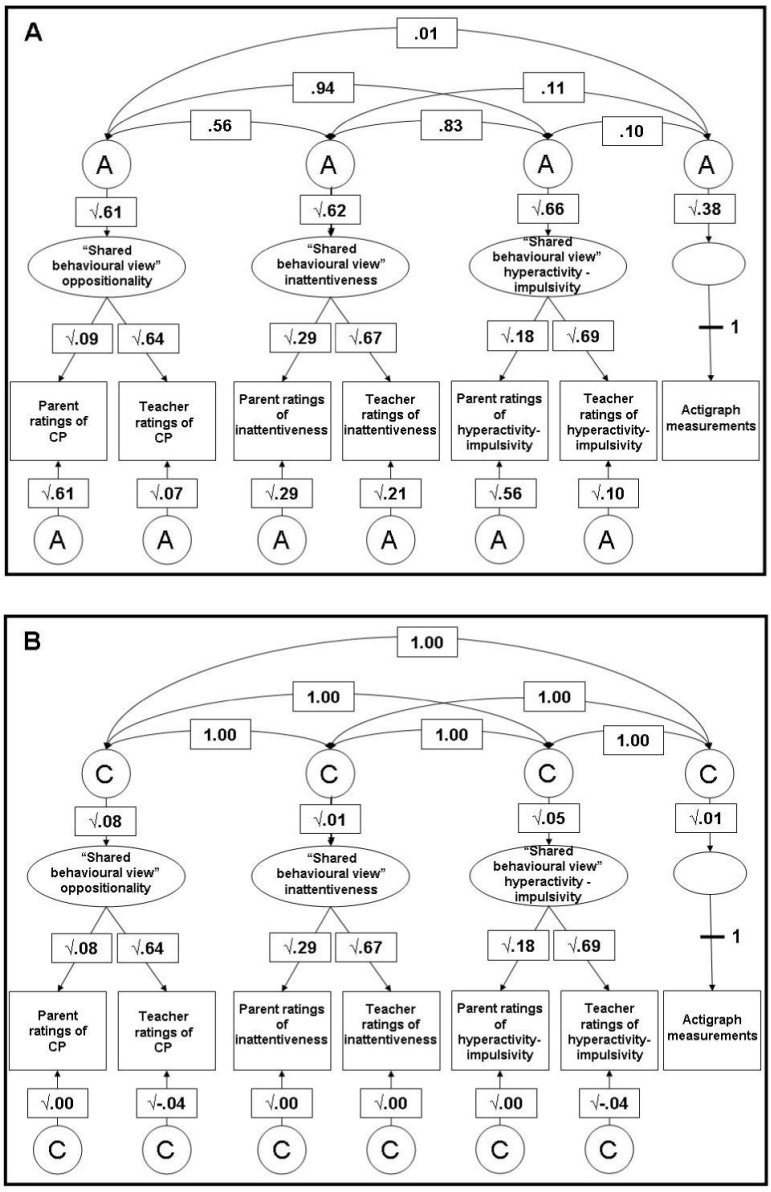

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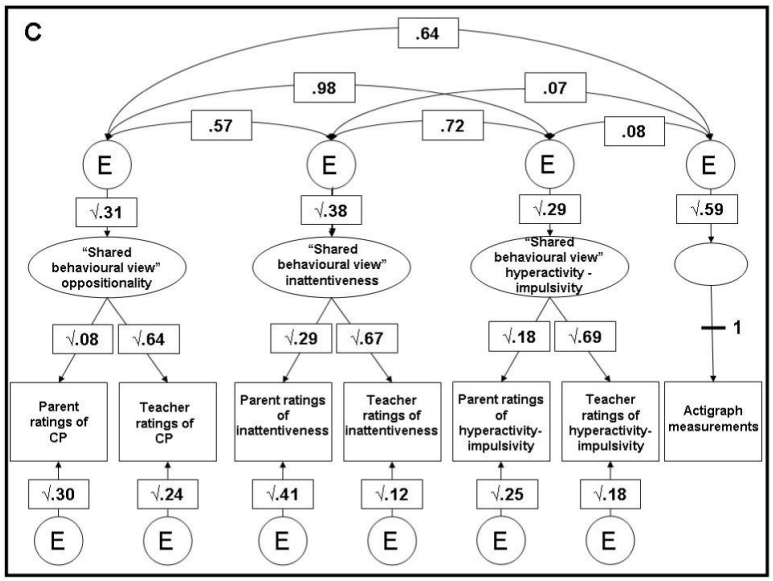

Figure 2.

Standardised parameter estimates from a full multivariate psychometric model examining the overlap between assessments of oppositionality, inattentiveness and hyperactivity-impulsivity shared by parents and teachers, and actigraph measurements of activity level. Note: Twin 1 only shown for simplicity. Latent factor loading on actigraph measurements constrained to 1. All parameter estimates are derived from the same model, split here for clarity. Panel A shows additive genetic parameter estimates plus factor loadings, panel B shows shared environment parameter estimates plus factor loadings, panel C shows child-specific parameter estimates plus factor loadings

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Table I

Phenotypic and within-pair correlations (95% confidence intervals in brackets) and means (standard deviations in brackets) for and across parent and teacher ratings of oppositionality, inattentiveness and hyperactivity-impulsivity and actigraph measurements from the constrained, saturated model

	Parent- rated oppositionality	**Teacher- rated oppositionality**	**Parent- rated inattentiveness**	**Teacher- rated inattentiveness**	**Parent- rated hyperactivity-impulsivity**	**Teacher- rated hyperactivity-impulsivity**	**Actigraph measurements**
Teacher -rated oppositionality	.21 (.15 - .27)						
Parent- rated inattentiveness	.40 (.34-.45)	.22 (.16-.27)					
Teacher- rated inattentiveness	.13 (.07 - .19)	.38 (.33 - .44)	.45 (.39 - .49)				
Parent- rated hyperactivity-impulsivity	.55 (.51-.59)	.27 (.21-.33)	.58 (.54 - .61)	.26 (.20 - .32)			
Teacher- rated hyperactivity-impulsivity	.21 (.16 - .27)	.68 (.64-.71)	.29 (.24 - .35)	.57 (.53 - .61)	.35 (.30 - .41)		
Actigraph measurements	.08 (.00 - .15)	.12 (.04-.20)	.14 (.07 - .21)	.17 (.09 - .24)	.24 (.17 - .31)	.16 (.09 - .23)	
MZ mean (SD)	**7.78(5.65)**	**1.19 (2.23)**	**5.56 (5.01)**	**4.56 (4.74)**	**6.11 (5.08)**	**2.83 (4.10)**	**337.45 (198.48)**
DZ mean (SD)	*8.18 (5.73)*	*1.42 (2.52)*	*6.43 (5.76)*	*5.48 (6.05)*	*6.17 (5.27)*	*2.50 (5.06)*	*350.81 (247.88)*

	Twin 1						
	Parent -rated oppositionality	**Teacher- rated oppositionality**	**Parent -rated inattentiveness**	**Teacher- rated inattentiveness**	**Parent -rated hyperactivity-impulsivity**	**Teacher- rated hyperactivity-impulsivity**	**Actigraph measurements**
MZ Twins *DZ twins*							
Twin 2							
Parent -rated oppositionality	**.70 (.64 - .75)**	*.30 (.21 - .39)*					
Teacher- rated oppositionality	**.18 (.10 - .25)**	*.03 (-.04 - .11)*	**.49 (.38 - .58)**	*.27 (.17-.36)*			
Parent -rated inattentiveness	**.38 (.32 - .44)**	*.29 (.23 - .36)*	**.13 (.05 - .21)**	*.04 (-.03 - .11)*	**.56 (.47 - .63)**	*.07 (-.02 - .17)*	

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	Twin 1													
	Parent -rated oppositionality		**Teacher- rated oppositionality**		**Parent -rated inattentiveness**		**Teacher- rated inattentiveness**		**Parent -rated hyperactivity-impulsivity**		**Teacher- rated hyperactivity-impulsivity**		**Actigraph measurements**	
MZ Twins /*DZ twins*														
Twin 2														
Teacher- rated inattentiveness	**.13 (.06 - .20)**	*.10 (.03 - .17)*	**.20 (.12 - .28)**	*.09 (.01 - .17)*	**.33 (.25 - .40)**	*.01 (-.07 - .09)*	**.63 (.54 - .70)**	*.22 (.12 - .31)*						
Parent -rated hyperactivity-impulsivity	**.52 (.47 - .57)**	*.29 (.21 - .36)*	**.23 (.15 - .30)**	*.06 (-.01 - .13)*	**.45 (.39 - .51)**	*.18 (.11 - .26)*	**.24 (.17 - .30)**	*.06 (-.01 - .13)*	**.75 (.69 - .79)**	*.21 (.12 - .30)*				
Teacher- rated hyperactivity-impulsivity	**.17 (.10 - .24)**	*.09 (.01 - .16)*	**.41 (.33 - .49)**	*.20 (.12 - .28)*	**.25 (.17 - .32)**	*.03 (-.04 - .10)*	**.38 (.30 - .45)**	*.16 (.08 - .23)*	**.29 (.22 - .35)**	*.09 (.01 - .16)*	**.59 (.50 - .67)**	*.27 (.17 - .36)*		
Actigraph measurements	**.12 (.03 - .20)**	*.05 (-.04 - .13)*	**.07 (.00 - .16)**	*.07 (-.02 - .16)*	**.13 (.04 - .21)**	*.05 (-.03 - .13)*	**.10 (.01 - .19)**	*.12 (.04 - .20)*	**.23 (.14 - .31)**	*.11 (.03 - . 19)*	**.14 (.05 - .23)**	*.09 (.01 - .18)*	**.73 (.65 - .79)**	*.57 (.48 - .65)*

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Table II

Model fits for genetic models examining the relationship between parent- and teacher ratings of oppositionality, inattentiveness and hyperactivity-impulsivity, and actigraph measurements of activity level

Model	−2LL	df	χ^2	df	p	$\Delta\chi^2$	Δdf	p	AIC	Comparison model
Univariate models										
Parent rated oppositionality										
1. Saturated phenotypic model	6668.34	1277	-	-	-	-	-	-	-	-
2. ACE (sex differences) [a]	6681.01	1294	12.67	17	.76	-	-	-	−21.33	1
3. ACE (no scalar) [b]	6686.01	1295	17.67	18	.48	4.88	1	.03	−18.33	1/2
4. ACE (no quantitative differences)[c]	6684.05	1296	15.71	19	.47	2.91	2	.23	−22.29	1/2
Teacher rated oppositionality										
5. Saturated phenotypic model	5273.41	1130	-	-	-	-	-	-	-	-
6. ACE (sex differences) [a]	5293.68	1147	20.27	17	.26	-	-	-	−13.73	5
7. ACE (no scalar) [b]	5516.59	1148	243.18	18	<.001	222.91	1	<.001	207.18	5/6
8. ACE (no quantitative differences)[c]	5300.36	1149	26.95	19	.11	6.67	2	.04	−11.05	5/6
Parent rated inattentiveness										
9. Saturated phenotypic model	6462.98	1278	-	-	-	-	-	-	-	-
10. ACE (sex differences) [a]	6497.68	1295	34.70	17	.01	-	-	-	0.70	9
11. ACE (no scalar) [b]	6550.03	1296	87.05	18	<.001	52.35	1	<.001	51.05	9/10
12. ACE (no quantitative differences)[c]	6503.91	1297	40.93	19	.002	6.23	2	.04	2.93	9/10
13. ADE (sex differences) [a]	6480.54	1295	17.56	17	.42					9
14. ADE (no scalar) [b]	6533.83	1296	70.85	18	<.001	53.30	1	<.001	34.85	9/13
15. ADE (no quantitative differences)[c]	6485.15	1297	22.17	19	.28	4.61	2	.10	−15.83	9/13
Teacher rated inattentiveness										
16. Saturated phenotypic model	5876.40	1134	-	-	-	-	-	-	-	-
17. ACE (sex differences) [a]	5893.57	1151	17.17	17	.44	-	-	-	−16.83	16
18. ACE (no scalar) [b]	6007.50	1152	131.10	18	<.001	113.93	1	<.001	95.10	16/17
19. ACE (no quantitative differences)[c]	5898.40	1153	22.00	19	.28	4.83	2	.09	−16.00	16/17
Parent rated hyperactivity-impulsivity										

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Model	−2LL	df	χ^2	df	p	$\Delta\chi^2$	Δdf	p	AIC	Comparison model
20. Saturated phenotypic model	6528.94	1278	-	-	-	-	-	-	-	-
21. ACE (sex differences) [a]	6558.74	1294	30.20	17	.02	-	-	-	−3.80	20
22. ACE (no scalar) [b]	6608.59	1296	79.65	18	<.001	49.85	1	<.001	43.65	20/21
23. ACE (no quantitative differences)[c]	6567.40	1297	38.46	19	.001	8.66	2	.01	.46	20/21
Teacher rated hyperactivity-impulsivity										
24. Saturated phenotypic model	5520.14	1134	-	-	-	-	-	-	-	-
25. ACE (sex differences) [a]	5552.08	1151	31.94	17	.02	-	-	-	−2.06	24
26. ACE (no scalar) [b]	5786.58	1152	266.44	18	<.001	234.50	1	<.001	230.44	24/25
27. ACE (no quantitative differences)[c]	5554.35	1153	34.21	19	.04	2.27	2	.32	−3.79	24/25
Actigraph measurements										
28. Saturated phenotypic model	4162.46	804	-	-	-	-	-	-	-	-
29. ACE (sex differences) [a]	4185.32	821	22.86	17	.15	-	-	-	−11.14	28
30. ACE (no scalar) [b]	4189.10	822	26.64	18	.14	3.78	1	.05	−9.36	28/29
31. ACE (no quantitative differences)[c]	4193.44	823	30.98	19	.04	8.13	2	.02	−7.02	28/29
Bivariate models										
Oppositionality										
32. Saturated phenotypic model	11876.28	2387	-	-	-	-	-	-	-	-
33. Biometric model	11941.72	2442	65.44	55	.16	-	-	-	−44.56	32
34. Psychometric model	11941.74	2442	65.46	55	.16	-	-	-	−44.58	32
35. Bias model	12048.17	2443	171.89	56	<.001	106.43	1	<.001	59.89	32/34
Inattentiveness										
36. Saturated phenotypic model	12048.09	2392	-	-	-	-	-	-	-	-
37. Biometric model	12173.65	2447	125.56	55	<.001	-	-	-	15.56	36
38. Psychometric model	12173.81	2447	125.72	55	<.001	-	-	-	15.72	36
39. Bias model	12234.23	2448	186.14	56	<.001	60.42	1	<.001	74.14	36/38
Hyperactivity-impulsivity										
40. Saturated phenotypic model	11904.46	2392	-	-	-	-	-	-	-	-
41. Biometric model	12003.37	2447	98.91	55	<.001	-	-	-	−11.09	40
42. Psychometric model	12003.43	2447	98.97	55	<.001	-	-	-	−11.15	40

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Model	−2LL	df	χ^2	df	p	$\Delta\chi^2$	Δdf	p	AIC	Comparison model
43. Bias model	12117.80	2447	213.34	56	<.001	114.37	1	<.001	101.34	40/42

[a]Sex differences model indicates a model where males and females are specified to have different variances and different standardised A, C/D and E estimates

[b]No scalar model indicates a model where males and females are specified the same variance and different standardised A, C/D and E estimates

[c]No quantitative differences indicates a model where males and females are specified to have different variances but the same standardised A, C/D and E estimates

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Table 3

Parameter estimates from the full multivariate psychometric model examining the overlap between the 'shared behavioural view'[d] from parent and teacher ratings of oppositionality, inattentiveness and hyperactivity-impulsivity, and actigraph measurements of activity level

	Shared behavioural view of oppositionality	Shared behavioural view of inattentiveness	Shared behavioural view of hyperactivity-impulsivity	Actigraph data
Phenotypic correlations				
Shared behavioural view of oppositionality	1.00			
Shared behavioural view of inattentiveness	.56	1.00		
Shared behavioural view of hyperactivity-impulsivity	.95	.79	1.00	
Actigraph data	.05	.09	.11	1.00
Etiological influences				
Genetic influences [a]				
Shared behavioural view of oppositionality	**.61**	.36 (61%)	.60 (73%)	.00 (0%)
Shared behavioural view of inattentiveness	.58	**.62**	.53 (67%)	.05 (56%)
Shared behavioural view of hyperactivity-impulsivity	.94	.83	**.66**	.05 (50%)
Actigraph data	.01	.11	.10	**.39**
Shared environment influences [b]				
Shared behavioural view of oppositionality	**.08**	.03 (5%)	.06 (7%)	.03 (60%).
Shared behavioural view of inattentiveness	1.00	**.01**	.02 (3%)	.01 (11%)
Shared behavioural view of hyperactivity-impulsivity	1.00	1.00	**.05**	.02 (20%)
Actigraph data	1.00	1.00	1.00	**.01**
Child specific environment influences [c]				
Shared behavioural view of oppositionality	**.31**	.20 (34%)	.17 (20%)	.02 (40%)
Shared behavioural view of inattentiveness	.57	**.38**	.24 (30%)	.03 (33%).
Shared behavioural view of hyperactivity-impulsivity	.98	.72	**.29**	.03 (30%)

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	Shared behavioural view of oppositionality	Shared behavioural view of inattentiveness	Shared behavioural view of hyperactivity-impulsivity	Actigraph data
Actigraph data	.04	.06	.08	**.60**

[a]The heritability (with 95% confidence intervals) of each latent factor (representing the shared behavioural view) is given in bold on the diagonal. The genetic correlations are given below the diagonal. The contribution of genetic factors to the phenotypic correlations is given above the diagonal, with the percentage of the phenotypic correlation that is due to genetic effects in brackets.

[b]The same three types of information are presented for shared environmental influences.

[c]The same three types of information are presented for child-specific environmental influences.

[d]Note: Shared behavioural view represents a factor score between parent and teacher ratings of the same behaviour.