Genetic and Cultural Transmission of Antisocial Behavior: An Extended Twin Parent Model

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Considerable evidence from twin and adoption studies indicates that both genetic and shared environmental factors play a substantial role in the liability to antisocial behavior. Although twin and adoption designs can resolve genetic and environmental influences, they do not provide information about assortative mating, parent–offspring transmission, or the contribution of these factors to trait variation. We examined the role of genetic and environmental factors for conduct disorder (CD) using a twin–parent design. This design allows the simultaneous estimation of additive genetic, shared and individual-specific environmental effects, as well as sex differences in the expression of genes and environment in the presence of assortative mating and combined genetic and cultural transmission. A retrospective measure of CD was obtained from twins and their parents or guardians in the Virginia Twin Study of Adolescent Behavior Development and its Young Adult Follow up sample. Both genetic and environmental factors play a significant role in the liability to CD. Major influences on individual differences appeared to be additive genetic (38%–40%) and unique environmental (39%–42%) effects, with smaller contributions from the shared environment (18%–23%), assortative mating (~2%), cultural transmission (~2%) and resulting genotype-environment covariance. This study showed significant heritability, which is slightly increased by assortative mating, and significant effects of primarily nonparental shared environment on CD.

In this article, we aim to accomplish four goals. First, we highlight some of the main findings published so far with respect to individual differences in internalizing and externalizing behavior from a range of perspectives. Second, we briefly introduce the genetic epidemiologic literature on antisocial behavior, with a special focus on longitudinal applications. Third, we describe the overall design, sample and measures in the Virginia Twin Study of Adolescent Behavior Development (VTSABD) and Young Adult Follow Up (YAFU) in detail as well as the measures on antisocial behavior used in the current analyses. Fourth, we illustrate some of the unique features of its design by extending the classical twin study with data collected from biological and nonbiological (referred to as step- for this article) parents to examine the role of genetic and environmental factors in the variation of antisocial behavior. The added information from parent–offspring correlations and spousal correlations will allow us to (i) disentangle genetic and cultural transmission, (ii) partition the environmental variance into influences shared with parents, with twin siblings and those specific to the individual, and (iii) test for and quantify the effects of assortative mating. This design thus provides an extended perspective to the traditional longitudinal study by studying the sources of continuity and change across generations. Although we only illustrate this using retrospective data in adolescence and one single measure, these type of analyses can be readily applied to include prospective longitudinal data from childhood to adolescence and young adulthood.

The VTSABD (Hewitt et al., 1997) and its YAFU were designed specifically to focus on the developmental trajectories of behavior and disorders from childhood to young adulthood and to study the interplay of genes and environment in shaping individual differences in internalizing and externalizing behaviors. The study used a rich assessment battery including dimensional and categorical measures, multiple raters, and environmental indices. Data were collected at up to five occasions with varying intervals

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from genetically related and unrelated individuals, aged 8 through 30.

Previous analyses of the data in the VTSABD have focused on baseline prevalence rates and univariate genetic analyses. Several publications have addressed the representativeness of the population-based Adolescent Behavior Development (ABD) sample for nosological and epidemiological studies. CBC-L externalizing behaviors were slightly but significantly elevated in the ABD twin sample compared with norms for nontwins and elevation for internalizing symptoms in older twins (Gau et al., 1992). Meyer et al. (1996) showed that families in the ABD sample demonstrate only slight differences in the distribution of socioeconomic status (SES) from the census tracts in which the targeted twins were located, there being a slight deficit of the very highest SES respondents. The impact of this bias on rates of psychopathology was slight. Simonoff et al. (1997) found that age- and sex-specific prevalence rates of the principal *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; DSM-III-R; American Psychiatric Association, 1987) diagnoses at Wave 1 were close to those from other epidemiological studies. Pickles et al. (2001) showed that prediction of depressive symptomatology at Wave 2 is affected only by Wave 1 symptoms and not impairment. For conduct and oppositional defiant disorders, prior impairment improved prediction of later symptomatology.

Eaves et al. (1997) presented cross-sectional model-fitting results, pooled across ages, for symptom counts and factorially derived scales for ratings by mothers, fathers and children on all aspects of behavior; then showed that genetic contributions were generally small to moderate, with some evidence of shared environmental influences on questionnaire ratings of conduct problems. Sibling contrast effects were found for parental ratings of attention-deficit/hyperactivity disorder (ADHD), but not for teacher ratings (Simonoff et al., 1998). Manifest anxiety ratings by twins and parents showed that children and parents rate genetically different aspects of the phenotype (Topolski et al., 1999). Reynolds et al. (1996) analyzed Wave 1 oral reading performance. Maes et al. (1999) reported cross-sectional analysis of the first wave of data relating to the lifetime and current use of alcohol, drugs and tobacco. The findings demonstrate that the contribution of genetic and environmental factors depends significantly on context and severity of use.

The VTSABD assesses a wide variety of putative indices of the family and individual environment and is therefore valuable for the study of nature-nurture interplay. Silberg at al. (1999) showed that the genetic variance in depression symptoms increases significantly during puberty in girls, as does the variance in life events. Furthermore, (1) genetic variance in female postpubertal depression is greater in twins exposed to higher 'doses' of adverse environments (G × E interaction, Silberg et al., 2001b); (2) depression is associated with adverse life events (G–E correlation, Silberg et al., 1999); and (3) prepubertal genetic differences in anxiety predict postpubertal genetic differences in depression (heterotypic continuity, Silberg et al., 2001a). Subsequently, Eaves et al. (2003) used Markov Chain Monte Carlo (MCMC) methods to analyze the joint roles of G × E and *r*G–E in the developmental transition from early anxiety to later depression, thus integrating Silberg et al.'s papers into a single analysis of the simultaneous interaction and correlation of random genetic and environmental factors and identified three separate pathways by which prepubertal differences in anxiety may produce later differences in depression. Analyses focused on the role of environment, G × E interaction and G–E correlation for disruptive behavior have shown significant associations of conduct disorder (CD) with shared environmental factors: parental alcoholism and presence versus absence of a stepfather (Foley, Pickles, et al., 2004); maternal smoking in pregnancy (Silberg et al., 2003); marital discord and family adaptability (Meyer et al., 2000); and differential treatment of individual twins (Carbonneau et al., 2002). Preliminary developmental analyses of antisocial behavior measures suggested that the shared environment is more important prior to adolescence and that genes play a greater role afterwards (Lyons et al., 1995) and that puberty is an important threshold for change in the expression of genetic factors on CD (Eaves et al., 2000). Thus, for depression, genetic differences create differences in sensitivity to the environment (G × E) while for anxiety and antisocial behavior, genes influence exposure to the environment (*r*G-E). Recent publications have explored the main effects and interaction of childhood adversity (parental neglect and exposure to violence between parents in the home) and genotype at the (X- linked) MAO-A locus in boys and girls from the VTSABD. Foley, Eaves, et al. (2004) detected a significant G × E interaction in boys that followed closely the pattern reported by Caspi et al. (2002). A parallel analysis in girls (Prom et al., 2006) shows a significant main effect of the locus, with greater risk in the 'low activity' homozygotes with weak evidence of G × E.

A range of VTSABD analyses have focused on patterns of comorbidity and heterogeneity of disorders. Multivariate analyses of mood disorders have shown a common underlying genetic basis to prepubertal overanxious disorder (Silberg et al., 2001a) and postpubertal juvenile depression in girls, while prepubertal depression may have a shared-environmental component that is indexed by separation anxiety. Rowe et al. (2002) explored the role of genes in comorbidity between symptoms of bulimia and other disorders. Analysis of the Wave 1 externalizing data (including ADHD and oppositional disorder [ODD]) demonstrated the need to integrate the concepts of etiological heterogeneity with those of comorbidity. Silberg, Meyer, et al. (1996) identified three latent classes in child and parent ratings of disruptive behaviors in male

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twins: a 'pure' CD class, distinguished from 'normals' by additive genetic and shared environmental effects; a comorbid CD/ADHD class, distinguished by additive and nonadditive genetic effects; and a multiply disordered class characterized by additive genetic differences. A cross-sectional analysis of the first wave of data (Silberg, Rutter, et al., 1996) showed developmental change in the pattern of heterogeneity/comorbidity in externalizing disorders, such that in younger male twins there was virtually complete genetic correlation between ADHD and conduct disturbance, while in older twins a further set of genetic effects specific to CD are expressed (Nadder et al., 2002). Eaves et al. (2000) showed significant genetic and environmental correlation between the components of ADHD: impulsivity; inattention and hyperactivity, but also detected significant genetic effects specific to the components suggesting that ADHD is genetically heterogeneous. There was marked consistency of genetic effects on CD and ODD across sexes, but the sexes were heterogeneous with respect to genetic effects on ADHD. Nadder et al. (2001) detected rater- and measure-specific genetic effects on ADHD. Simonoff et al. (1998) fitted a multivariate 'ACE' model to four a priori subtypes of disruptive behavior: property violations; status violations; oppositional behavior; and aggression. They concluded that child self-reports showed high genetic correlations between subtypes, whereas maternal ratings showed a high degree of specificity across subtypes. Consistency between behavioral ratings by children, parents and teachers was low and parents attributed greater similarity to the symptoms of their twin children than can be explained by similarity in self-reports (Simonoff et al., 1995). Children's self-reports were less highly correlated between twins than parent or teacher reports (Eaves et al., 1997) but self-reports seem to show similar long-term consistency. In the case of ADHD, parents appear to contrast twins with one another, whereas teachers do not (Simonoff et al., 1998). Eaves et al. (2000) noted significant 'genetic overlap' between child and parent ratings of CD and ODD, but also rater-specific genetic effects that imply raters are sensitive to genetically distinct aspects of a child's behavior, or that they experience behavior in different contexts that interact with gene expression. The rater's own psychopathology may affect patters of rater inconsistency (Foley et al., 2005).

The longitudinal nature of the VTSABD study has permitted the analysis of continuity and change of the relative roles of genes and the family environment during development. The shared environment is more important prior to adolescence and genes play a greater role afterwards for both depression (Silberg et al., 1999, 2001a, 2001b) and lifetime versus current use of alcohol (Maes et al., 1999). In the short-term and mid-term (1–2 years), there is little evidence to decide whether parent–offspring interaction influences child psychopathology more than child psychopathology influences parent–offspring interaction (Carbonneau et al., 2002, Foley, Pickles, et al., 2004). Puberty is an important threshold for change in the expression of genetic factors on anxiety/depression (Eaves et al., 2003), ADHD/CD (Silberg, Rutter, et al., 1996) and obesity (Meyer et al., 1997). Recently with the availability of up to five waves of measurement necessary to track development into young adulthood, Silberg et al. (submitted) examined the genetic and environmental causes of continuity or change in antisocial behavior in males from childhood to young adulthood, which revealed important developmental differences in the etiology of antisocial behavior. Incorporating both mother and child reported assessments of CD symptoms on twins between ages 8 and 17 and antisocial behavior of the twins as young adults revealed the following four features: (1) a single genetic factor influencing antisocial behavior beginning at age 10 through young adulthood ('life-course persistent'); (2) a shared environmental effect beginning in adolescence ('adolescent-onset'), (3) a transient genetic effect at puberty, and (4) a specific genetic influence on adult antisocial behavior. Overall, these etiological findings coincide with Moffitt's developmental theory of antisocial behavior (Moffit, 1993). The genetic effect at puberty at age 12 to 15 is also consistent with a genetically mediated influence on the timing of puberty effecting the expression of genetic differences in antisocial outcomes. Tracy et al. (submitted) focused on sex differences in the genetic architecture of antisocial behavior in young adults and its genetic and environmental relationship to retrospective assessment of conduct disorder prior to age 18. They showed that both genetic and shared environmental factors contribute to variability of antisocial behavior in adolescence. However, shared environmental factors were no longer significant in young adulthood, supporting the hypothesis that in addition to unique environmental factors, 'adolescent-limited' antisocial behaviors are at least partly mediated by common environmental factors while 'life-course persistent' antisocial behaviors are influenced primarily by genetic effects. While genetic effects were stronger in males than in females, the shared environment had a greater impact on juvenile antisocial behavior in females than in males.

The results from analyses of the VTSABD and YAFU are remarkably consistent with considerable evidence, primarily from twin and adoption studies which shows that both genetic, shared and specific environmental factors play a substantial role in antisocial behavior. A meta-analysis of studies focused on antisocial behavior (Rhee & Waldman, 2002) concluded that genetic factors accounted for about 32% of the variance, with smaller but significant proportions of variance explained by dominance (9%) and shared environmental factors (16%). The unique environmental variance, including measurement error, was estimated at 43%. Their analysis included adolescent and adult samples, males and females, prospective and retrospective assessments of behavior, and both self-report and reports by other raters. A few publications

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Table 1
VTSABD/YAFU Sample Sizes by Age

Age (year)	8	9	10	11	12	13	14	15	16	17	18+	Total
VTSABD												
Wave 1	119	203	158	167	165	165	128	137	139	31		1412
Wave 2		79	126	154	105	124	156	93	107	107		1051
Wave 3					42	87	133	102	106	158		628
Wave 4							26	50	69	48		193
Total	119	282	284	321	312	376	443	382	421	344		3284
YAFU												
Age at Wave 1	85	165	134	133	147	144	112	125	129	28	1202	1202
% of Wave 1	0.71	0.81	0.85	0.80	0.89	0.87	0.88	0.91	0.93	0.90		0.85

have addressed the question of continuity in genetic and environmental effects between childhood and adulthood. Lyons et al. (1995) identified genetic, common and unique environmental factors that influenced both juvenile (before age 15) and adult (before age 15) behaviors, with additional unique environmental effects specific to adults in 3326 twin pairs from the Vietnam Era Twin Registry. Genetic factors had a larger impact in adulthood, while shared environmental factors were more important in childhood. Jacobson et al. (2002) identified quantitative differences in genetic effects between two retrospective measures of antisocial behavior in childhood (before age 15) and adolescence (age 15–18) with genetic factors having a larger impact in adolescence and shared environmental factors being more significant in childhood in 6806 male and female twins from the Virginia Twin Registry. However, no such difference was observed between adolescence and adulthood. Focusing specifically on young adulthood in males, Malone et al. (2004) found additive genetic and unique environmental effects to be the only factors contributing to antisocial behavior at three time points (ages 17, 20 and 24) in 289 twin pairs from the Minnesota Twin Family Study.

Although these prior studies have answered several important questions regarding the nature and nurture of antisocial behavior and its developmental trajectory, they did not address issues of transmission of behavior from parents to children, or the role of genes and environment on the influences across generations. Such questions require a longitudinal design that spans at least two generations or a twin design augmented with data from parents or other extended twin designs.

Materials and Methods

Virginia Twin Study of Adolescent Behavioral Development (VTSABD) and Young Adult Follow-Up (YAFU)

The VTSABD is the first population-based, multiwave, cohort–sequential twin study of adolescent psychopathology and its risk factors. The design includes Caucasian families of male and female monozygotic (MZ) and dizygotic (DZ) twins and their parents. Its goals are to assess genetic and environmental factors in developmental change, to identify many of the major familial psychosocial risk factors, and to characterize their correlation and interaction with genetic risk. Twins were initially assessed at ages 8 to 16. The YAFU has assessed the sample at a median follow-up age of 21 years. Details of sample ascertainment and assessment of socioeconomic bias were reported by Meyer et al. (1996).

In the VTSABD, adolescent male and female twins aged 8 through 16 were ascertained by statewide recruitment through Virginia schools. Of the 1894 Virginia families that were initially eligible for study, 1412 Caucasian families (74.5%) participated in the first wave of data collection (2775 individuals twins comprising 1384 complete pairs). Twins under age 18 and currently enrolled in high school were followed every 18 months up to 3 times. One thousand and fifty-one out of the 1302 families that continued to meet the age and Virginia residence requirements of the study completed a second home interview (80% participation rate), and 628 of the 777 eligible twins families (81%) participated in a third wave of assessment; 193 families also completed Wave 4. Table 1 summarizes the numbers of pairs assessed in each year of life and each assessment wave. The aggregate data provide the equivalent of 4486 pairs of twins assessed at ages distributed between 8 and young adulthood. All assessments were done by face-to-face interview in the twins' home. A pair of interviewers each assessed one twin and one parent in each family.

At age 18 or older, all twins who participated in the first wave of the VTSABD were targeted for a young adult assessment. To date (8/06), 1185/1412 (84%) of pairs have been followed up in the YAFU. Of the 2,692 individuals eligible for participation, 2291 have completed the telephone interview and 186 remain to be interviewed. We have been unable to contact 97, and 118 have refused to participate. Twenty-four per cent of the YAFU subjects participated in only the first wave of the VTSABD, 32% participated in two waves, 31% in three waves, and 13% in all four waves. Ages of the

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Table 2

VTSABD/YAFU Sample Size by Zygosity and Sex of Twins and by Type of Relative/Informant

	Zygosity and sex of twins							Type of relative/informant			
Sample	MZ male	MZ female	DZ male	DZ female	DZ MF	Unknown	Total pairs	Indiv twins	Mother	Father	Teacher
VTSABD adolescents	313	408	183	189	295	24	1412	2824	1365	1105	1369
YAFU young adults	272	349	155	169	240	17	1185	2289	—	—	—

Note: Indiv = Individual

twins during participation in the YAFU ranged from 18 to 30 with a mean age of 21.4 years. Greater than 75% have had some level of college education and about 50% of the subjects were currently in school at the time of interview.

Table 2 gives the structure of the samples broken down by zygosity and sex of twin pairs, and the number of individuals twins, relatives and/or informants. VTSABD data comprise 6282 face-to-face assessments of juveniles across four waves and 2289 assessments of young adult twins. The twins' parents (N = 2470) also completed psychiatric assessments during the home interview, comprising 97% of mothers (N = 1365) and 78% of fathers (N = 1105). Of the mothers, 1119 were the biological mothers of the twins, and 59 were adoptive mothers, stepmothers or female guardians. 946 biological fathers and 224 nonbiological fathers participated. Parents as well as teachers also acted as informants about the adolescent twins' behavior. Teacher assessments of both twins on at least one occasion are available for 97% of the families (N = 1369). Zygosity assignment of twins was based on DNA when available, and otherwise on an algorithm combining standard questions gathered from the parents about the twins' similarity, and photographs of the twins rated independently by multiple raters. A detailed description can be found elsewhere (Eaves et al., 1997).

Measures

The VTSABD data comprise psychiatric assessment of parents and offspring as children and young adults, together with the principal psychosocial and environmental risk factors. Table 3 details the instruments used, their mode and wave of assessment, and rater(s). Prospective ratings were secured about each child and home by direct assessment of the child and by reports of parents and teachers. The core juvenile assessment comprised face-to-face interviews with each twin and both parents using the Child and Adolescent Psychiatric Assessment (CAPA, Angold et al., 1995) adapted for use with twins and their parents. The CAPA has forms for interviewing parents about their children (CAPA-P) and for the children themselves (CAPA-C); it yields over 300 coded pages of symptom data relevant to the common areas of childhood and adolescent disorder nuanced with onset, frequency, duration, incapacity, treatment, and context for each clinical domain. The core assessment of psychopathology in the twin pairs as young adults and their parents is in the M and S-sections that are based on the Structured Clinical Interview of DSM (SCID, Spitzer et al., 1990). Twins and parents also filled out self-report questionnaires (SRQ) which contained the following scales: What I Think and Feel (Manifest Anxiety, MA; Reynolds & Richmond, 1978), Mood and Feelings Questionnaire (MFQ, Costello & Angold, 1988), Behavior and Activities Checklist (BAQ, adapted from Olweus, 1989), Fears and Phobias (FSSC, adapted from Ollandick et al. 1989), Life Events Checklist (LEQ, adapted from Johnson and McCutcheon 1980), Twin Index of Rearing Environment (TIRE, adapted from Sibling Inventory for Differential Experience, SIDE, Daniels & Plomin, 1985), Family Adaptability and Togetherness Scale (FAT, Olsson et al., 1979), EASI Temperament Scale (EASI, Buss & Plomin, 1975). The Junior Eysenck Personality Questionnaire (Juvenile, JEPQ, Eysenck & Eysenck, 1975) was added in Wave 3; young adult twins filled out the Eysenck Personality Questionnaire (Adult Short form, Eysenck & Eysenck, 1975). Parents filled out questions about Health, Habits and Behavior (HHB), the Rutter 'A' Scale (RA, Rutter et al., 1970), and the Dyadic Adjustment Scale (DAS, Spanier, 1976). Teachers filled out the Rutter 'B' Scale (Teacher, (RB, Rutter et al., 1970), the Conners Teacher Rating Scale (CO, Conners) and the (Child Behavior Checklist (CBC, Achenbach). Prior to Wave 1 of the VTSABD, parents filled out the CBC, which was repeated in Wave 4 by parents and twins. These questionnaires cover personality measures, internalizing and externalizing constructs. The behavioral assessment was augmented with cognitive measures (Slosson Oral Reading Test (Slosson, 1990) and Raven's Standard Progressive Matrices (Raven, 1956) in Wave 3) and physical measures (height, weight) and questions about the twins' pregnancy and health problems. Environmental assessment includes the Home Environment Interview (based on Robins et al., 1985). Census block/tract data from 1990 US Census are also available.

In this article we use data on antisocial behavior (AB) to illustrate features of the data set and the types of analyses that may be conducted. AB was measured both prospectively in the twins between the ages of 8 and 16, and retrospectively in the young adult twins after they reached the age of 18 and in the twins' parents. Here we use the Conduct Disorder (CD) data obtained as part of the semi-structured interview for the

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Table 3

Measures Used in the VTSABD/YAFU with their Mode and Wave of Assessment and Rater(s)

			VTSABD				
Abbreviation	Content	Mode	Wave 1	Wave 2	Wave 3	Wave 4	YAFU
Behavioral							
CAPA	Behavior and psychopathology (family, peers, school, anxiety, depression, suicide, eating, puberty, hyperactivity, oppositional and conduct, tobacco, alcohol, drugs, incapacity)	I	TMoFa	TMoFa	T(MoFa)	T(MoFa)	
M, L, L2, M2	Adult psychopathology (depression, anxiety, panic, phobia, alcohol, drugs)	I	MoFa^{s} [M]	MoFa^{s} [M]	MoFa^{s} [L] (M2)*	MoFa^{s} [L2]	T^
S, S2, S3, ASP	Adult and childhood antisocial behavior	I	MoFa^{s} [S]	MoFa^{s} [S]	MoFa^{s} [S2] (ASP.A)*	MoFa^{s} [S3]	T
ADDR	Retrospective attention-deficit	I			MoFa^{s}		
SRQT	MA, MFQ, BAQ, FSSC, LEQ, SIDE, FAT^{s}, EASI, JEPQ$^{\&}$	Q	T	T	T	T	
SRQP	HHB, RA, MA, MFQ, BAQ, FSSC, LEQ, SIDE, FAT^{s}, EASI, DAS^{s}	Q	MoFa	MoFa	MoFa	MoFa	
SRQS	RB, CO, CBC	Q	Te	Te	Te	Te	
CBC	Internalizing and externalizing behavior	Q	P#			TP	
EPQ	Personality						T
Cognitive							
SORT	Reading	I	T	T	T	T	
SPM	Nonverbal reasoning	I			T		
Physical							
A	Twins' pregnancy, birth, similarity	I	MoFa	MoFa*	MoFa*	MoFa*	
AA, AA.1	Medical/health problems	I			MoFa [AA]	MoFa [AA.1]	
Measures	Height, weight		T	T	T	T	
DNA							
Environmental							
CL/CL3	Religion, attendance, REL^{Y}	I	MoFa	MoFa	MoFa*	MoFa*	T
P/Q	Household socioeconomic status	I		P	P*	P*	
S, S2, S3	Employment, relationships	I	MoFa [S]	MoFa [S]	MoFa [S2]	MoFa [S3]	
E	Home environment	I			TP		
R	Peer relations	I			T	T	
Census	Demographic indices						

Note: I: interview (face to face); Q: questionnaire; T: twins self-report, Mo: mother, Fa: father, Te: Teacher, P: Parent, (MoFa) mother or father rating, ^{s} assessment of parents of twins, [] section used, * if not done in Wave 1 or 2, ^telephone interview, $^{\&}$Wave 3 and 4 only, #mailed prior to Wave 1, ^{Y}YAFU only

CAPA: Child and Adolescent Psychiatric Assessment (Angold et al., 1995); M, L, M2, L2, S, S2, S3, ASP, ADDR, A, AA, AA.1, CL, CL3, P, Q, E, R: sections of interview with twins/parents; SRQ: Self-Report Questionnaire of T: twin, P: parent, S: school teacher; CBC: Child Behavior Checklist (Achenbach, 1988); JEPQ: Junior Eysenck Personality Questionnaire (Juvenile), EPQ: Eysenck Personality Questionnaire (Adult Short form; Eysenck & Eysenck, 1975); MA: Manifest Anxiety/What I Think and Feel (Reynolds & Richmond, 1978), MFQ: Mood and Feelings Questionnaire (Costello & Angold, 1988), BAQ: Behavior and Activities Questionnaire (adapted from Olweus, 1989), FSSC: Fears and Phobias (adapted from Ollandick et al., 1989), LEQ: Life Events Checklist (adapted from Johnson & McCutcheon, 1980), TIRE: Twin Index of Rearing Environment (adapted from SIDE; Daniels & Plomin, 1985), FAT: Family Adaptability and Togetherness Scale (Olsson et al., 1979), EASI: EASI Temperament Scale (Buss & Plomin, 1975), HHB: Health, Habits & Opinions, RA: Rutter 'A' Scale (Rutter et al., 1970), DAS: Dyadic Adjustment Scale (Spanier, 1976), RB: Rutter B Scale (Teacher, see Rutter et al., 1970), CO: Conners Teacher Ratings Scale (Conners CK); SORT: Slosson Oral Reading Test (Slosson, 1990), SPM: Raven's Standard Progressive Matrices (Raven, 1956); REL: Young Adult Religious Practices Interview, Home Environment Interview (based on Robins et al., 1985).

evaluation of adult Antisocial Personality Disorder (ASPD). The answers were incorporated into a scoring algorithm designed to correspond to DSM-III-R criteria for CD, and were used to generate a sum score of the number of symptoms present. DSM-III-R criteria were chosen for the basis of these measurement algorithms so that informative comparisons could be made with results from previously reported analyses using the VTSABD data. Parental data were obtained during any of the waves of the home interview, as part of the section about employment, relationships, adult and childhood behavior. Section S (including 11 out of 12 items, and excluding 'stolen with confrontation of victim') was used in Wave 1 or Wave 2 if not done in Wave 1. In Waves 3 and 4, parents were given a short version of the section (S2, 10 out of 12 items, excluding 'stolen with confrontation of victim' and 'forced sexual activity') if they had completed section S in Wave 1 or 2. Otherwise, they were given a full antisocial personality (ASP) section including all 12 CD items. This latter version was also administered to the young adult assessment of the twins. In all cases, the presence of the

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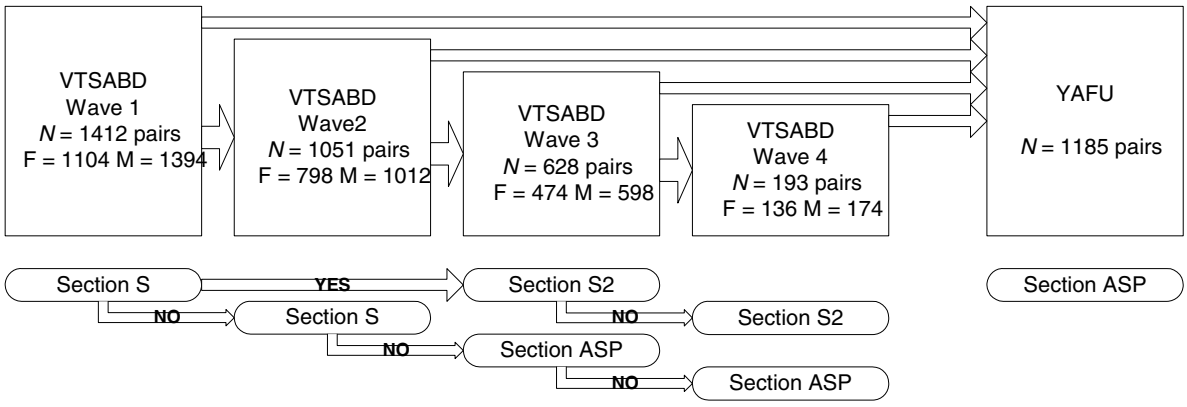

Figure 1

Design of the Virginia Twin Study of Adolescent Behavioral Development (VTSABD) and the Young Adult Follow Up (YAFU), with specific reference to the assessment of antisocial behavior.

Note: F: number of fathers; M: number of mothers; S: S-section in Waves 1 or 2 for parents or guardians; S2: short S-section in Waves 3 or 4 for parents or guardians; ASP: full S-section in Waves 3 or 4 for parents or guardians if not done in Waves 1 or 2, and for YAFU young adults.

items prior to age 18 was assessed and a diagnosis of CD was assigned if three or more symptoms were present. Missing values were imputed if less than 25% of the items were not missing. Figure 1 provides a graphical representation of the design of the study and the measures of antisocial behavior in parents and young adult twins used at each of the assessments.

Statistical Methods

Structural equation modeling of the data was undertaken using methods described in Eaves et al. (1999), which assesses the contributions of genetic effects in the presence of effects including vertical cultural inheritance, phenotypic assortative mating, shared and within-family environment. While this model was described for the extended twin kinship design (ET model), the core parameters can be estimated with the twin–parent design. Data from twins and their parents permit quantification of the contributions of additive genetic, dominance or shared environmental, and specific environmental factors as is the case for the classical twin study. In addition, it is possible (i) to estimate the degree of assortment (or to what extent the data deviate from the assumption of random mating), and (ii) the degree to which parents phenotypes influence their offspring (termed vertical cultural transmission). This second component effectively partitions the shared environment into parental versus nonparental sources. Although alternative models exist, the most often used model includes phenotypic assortment and phenotypic cultural transmission (Fulker, 1988; Heath & Eaves, 1985; Neale & Fulker, 1984). Phenotypic assortment occurs when mate selection is based at least partly on the trait being studied, and generates a correlation between the observed phenotypes of spouses. Also, the impact of assortment on the contribution of genetic and shared environmental factors can be estimated (Neale & Cardon, 1992). Furthermore, the contribution of the genetic and environmental factors may depend on sex, both in their magnitude and nature.

To date, this model has been applied to data from nuclear twin families, and also from adoption designs (Baker et al., 1983). In the VTSABD, data were obtained from the parents/guardians who were most familiar with the adolescent twins. Therefore, a relatively large number of nonbiological fathers, and a smaller number of nonbiological mothers participated in the study. We therefore extended the traditional twin–parent model to allow for both biological and nonbiological parents, resulting in four different types of families. Given the relatively small percentage of families with a biological father and nonbiological mother (1.5%) or with two nonbiological parents (3.5%), we limited the current analyses to families with either (i) both biological parents; or (ii) a biological mother and nonbiological father. In addition to the assumption of equilibrium of variances across generations, three additional assumptions are made: (1) equal variance for biological and nonbiological parents (while allowing for different means); (2) equal assortment between two biological parents and between a biological and nonbiological parent; and (3) equal cultural transmission from a biological or nonbiological parent. When genetic factors are operating, the expected parent–offspring covariance will be smaller between children with their nonbiological parent than with their biological parents. Although the genetic and shared environmental covariance will be the same, genotype–environment covariance (resulting from the combined presence of genetic and cultural transmission) will be greater in twins with both biological parents versus those with one biological and one nonbiological parent. Figure 2a presents a path diagram of the extended twin parent model (ETP model), which was implemented in the statistical modeling package Mx (Neale et al., 2006, see Maes et al.,

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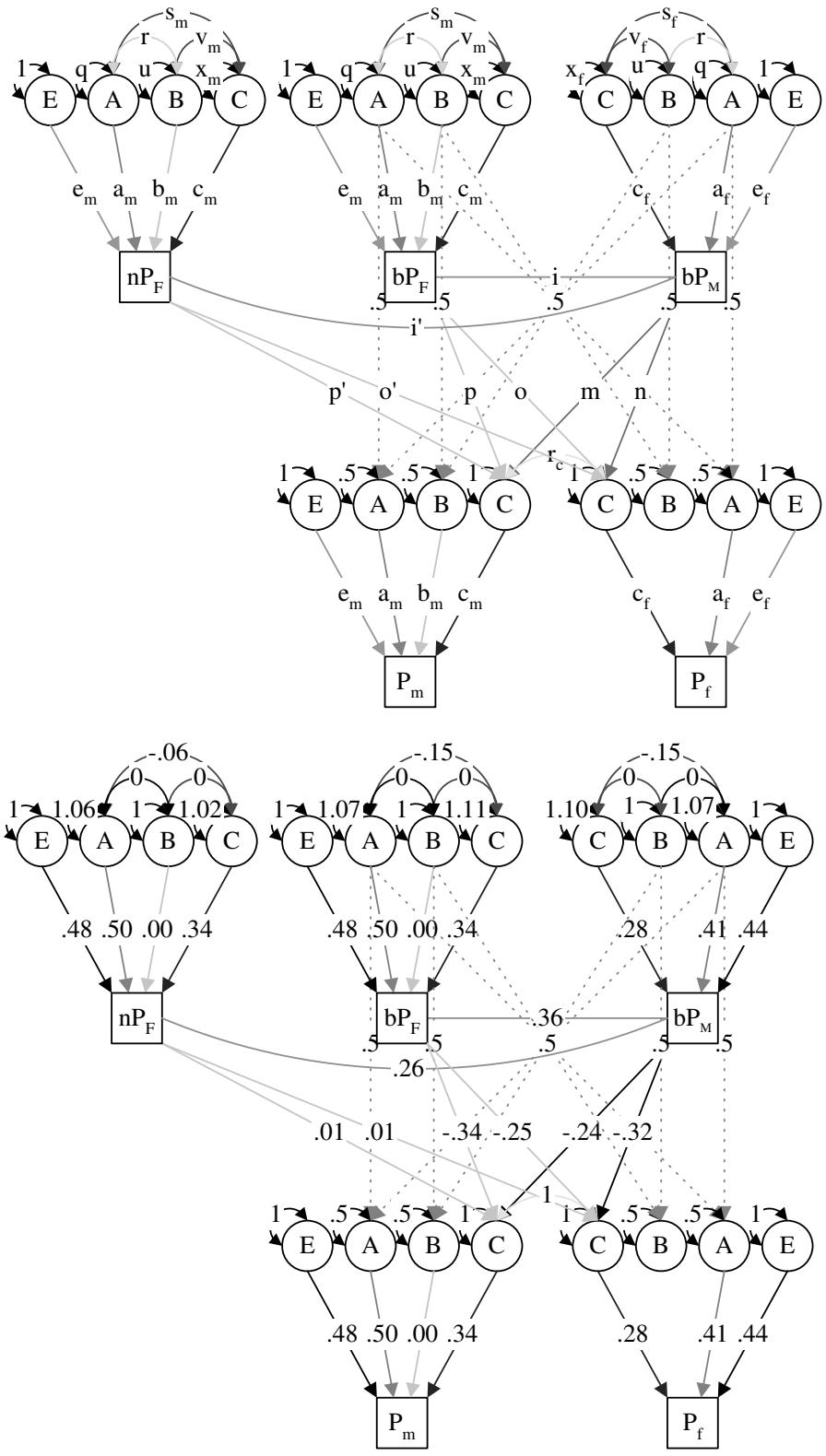

Figure 2a and 2b

Full twin–parent resemblance model for opposite-sex DZ twins (P_m, male twin; P_f, female twin) and their parents (either both biological parents, bP_F, biological father and bP_M, biological mother; or biological mother and nP_F nonbiological father).

Note: Path coefficients are the same in both generations, and gene–gene and gene–environment correlations occur in both generations. Coefficients include additive genetic [*a, b*], shared environmental [*c*] and unique environmental [*e*] sources of variance; subscripts m and f for males and females [e.g., a_m and a_f]; phenotypic cultural transmission from the parent's phenotype to the offspring's shared environment, which may differ by sex of parent and child [m, n, o ,p]; genotype–environment covariance between gender-common and male-specific genetic factors and shared environmental factors [s, v]; phenotypic assortment [i]. Correlation between gender-common and male-specific genetic factors [*r*]. Variances of latent factors [q, u, x] are constrained to be equal across generations. Estimates of path coefficients are shown in Figure 2b.

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Table 4

Prevalence Rates — *N* cases (per cent) — for Conduct Disorder and Antisocial Personality Disorder in Young Adult Twins and Parents of Twins, and Their Tetrachoric Correlation (*r*)

	N	Bio male	Bio female	BM male	BM female	All male	All female	Total	ASP	*r* CD-ASP
Young adults	2244					301 (30)	153 (13)	454 (20)	139 (6)	.44
All parents	2533	220 (22)	107 (8)	46 (28)	6 (13)	266 (23)	113 (8)	379 (15)	89 (4)	.45
Parents S (w1/w2)	2475	213 (22)	107 (8)	41 (30)	4 (10)	254 (23)	111 (8)	365 (15)	84 (3)	.46
Parents S2 (w3/w4)	949	77 (21)	38 (7)	4 (17)	0 (0)	81 (20)	38 (7)	119 (13)	20 (2)	.47
Parents ASP	58	7 (35)	0 (0)	7 (28)	2 (33)	14 (31)	2 (15)	16 (28)	4 (7)	.31

Note: Bio: twin families with both biological parents; BM: twin families with biological mother and nonbiological father;

S: S-section of interview on adult and childhood antisocial behavior used in Wave 1 and Wave 2;

S2: S-section of interview on adult and childhood antisocial behavior used in Wave 3 and Wave 4;

ASP: ASP-section of interview on adult and childhood antisocial behavior used in Wave 3 if no S-section available from Wave 1 or 2

CD: Conduct disorder

ASP: Antisocial personality disorder

1999 for description of the Mx script) and fitted to the raw ordinal data to obtain maximum likelihood estimates of the model parameters. We now describe these parameters in detail with reference to the path diagram. The variance of the phenotype in twins and parents (P_m, male twin; P_f, female twin; bP_F, biological father; bP_M, biological mother; nP_F, nonbiological father) is partitioned in additive genetic [*a,b*], shared environmental [*c*] and unique environmental [*e*] sources of variance. These can vary in magnitude between males and females [e.g., a_m and a_f]. Furthermore, to allow a different set of genes in males and females, we modeled them as gender-common [*a*] and male-specific [*b*] additive genetic factors, where the latter would contribute to the phenotypic variances of males only. Note that the paths from the genetic factors in the parental generation to those in the offspring generation are fixed to .5, including the paths from the mother's male-specific genes to her male and female offspring. In addition to the genetic transmission from parents to offspring, parents may also influence their children through nongenetic pathways. In the current specification, these influences are modeled as phenotypic cultural transmission from the parent's phenotype to the offspring's shared environment, and they are allowed to differ by sex of parent and child [m, n, o, p]. Note that we can test whether these environmental transmission paths are equal for biological and nonbiological fathers. Besides parental environmental influences, other aspects of the environment may generate similarity between twins or siblings, which are modeled as residual covariances, and referred to as nonparental shared environment [r_c]. The combined presence of genetic and cultural transmission generates genotype–environment covariance, which may exist between shared environmental factors and both gender-common and male-specific genetic factors [s, v]. Finally, a design including spousal information can also assess the degree of assortment, here modeled as phenotypic assortment [i]. We can also test the equality of assortment between the biological parents and between a biological and a nonbiological parent. As a consequence of assortment, sources of variance may become correlated, for example, gender-common and male-specific genetic factors [*r*].

Results

Response Frequencies

Twenty per cent of young adult twins endorsed more than three conduct items retrospectively, resulting in a diagnosis of conduct disorder (Table 4). Rates are significantly higher in males (30%) than females (13%). The prevalence of conduct disorder — when asked retrospectively — is somewhat lower in parents (15%), with fathers about three times more likely to have a diagnosis of conduct disorder than mothers. As the parental data resulted from the combination of slightly different instruments administered at different times, we also report the prevalence rates separately by instrument. Rates were somewhat lower at the second assessment of CD (S2) than at the first (S) but showed a similar pattern of sex differences. Parents who had not participated in the first two waves of the study showed markedly higher rates of CD in both males and females. This is partly due to the greater proportion of nonbiological parents in this group, whose rates for CD are consistently higher than those of biological parents. We assessed the reliability of CD using polychoric correlations, both at the level of symptom counts (.73) and diagnosis (.72). The reliability was slightly higher in males (.71) than in females (.62). Rates for adult antisocial behavior (ASP) are about a third of CD rates, with tetrachoric correlations over time ranging from .31 to .47, indicating that only a subset of individuals with CD continue to show antisocial behavior as adults, and that not all individuals with ASP presented with CD in adolescence. Note that for these analyses we did not include the presence of CD before age 15 as a prerequisite for a diagnosis of ASP according to DSM-III-R. Raw CD symptom count shows marked skewness. To improve the metric properties of the scale,

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Table 5

Sample Sizes, Mean and Variance for Conduct Disorder (After Square Root Transformation and Regression of Sex, Age and Their Interaction)

	Both biological parents				Biological mother + nonbiological father			
	Twin 1	Twin 2	Father	Mother	Twin 1	Twin 2	Father	Mother
N MZM	213	210	194	222	31	32	22	35
N DZM	118	116	110	122	23	24	12	26
N MZF	284	260	236	270	62	63	37	63
N DZF	124	129	115	130	20	21	7	23
N DZO	177	182	171	185	34	33	16	36
	Male twin	Female twin	Dad	Mom	Male twin	Female twin	Dad	Mom
Mean	–0.011	–0.044	–0.010	–0.017	0.158	0.185	0.168	0.135
Variance	0.581	0.456	0.593	0.401	0.568	0.477	0.522	0.423

Note: MZM: monozygotic male twins; DZM: dizygotic male twins; MZF: monozygotic female twins; DZF: dizygotic female twins; DZO: dizygotic opposite-sex twins

the count data were square root transformed and the effects of sex, age and their interaction regressed out within each generation.

Maximum Likelihood Estimation of Correlations

Table 5 presents sample sizes by zygosity and family type, as well as means and variances for twins and parents by sex and family type. We fitted a baseline model with free parameters for all means, variances and covariances. Means could be equated for male twins across both members of a twin pair and across zygosity within nuclear families and within step families without significant loss of fit. This was also true for all female twins, all fathers and all mothers. However, the CD means for twins and parents in nuclear families were significantly lower than those in twin families with a biological mother and nonbiological father. Similar tests for the equality of variances across order of the twins and zygosity were nonsignificant both within and across family type. Tests for equality of parent–offspring and spousal correlations across twin order and zygosity within family type, that is, for example, equality of all father–son pairs, including father–male Twin 1, father–male Twin 2 in both MZ and DZ pairs, were also nonsignificant. Maximum likelihood estimates of the correlations by type of relative (twin, parent–offspring, spousal) and family are presented in Table 6. The twin correlations were consistent with the contribution of both additive genetic and shared environmental influences to antisocial behavior in addition to specific environmental influences. They were slightly but not significantly higher in step families than in nuclear families. Parent–offspring correlations were considerably lower than DZ twin correlations. Nonbiological father–offspring correlations were negative but not significant. Mother–offspring correlations did not differ significantly by family type . The spousal correlation was significant in nuclear families but not in step families.

Maximum Likelihood Estimation of Genetic and Environmental Contributions

A twin–parent model estimating the contributions of additive genetic, shared and specific environmental factors was fitted to conduct disorder measured retrospectively in twins and either their biological parents or their biological mother and nonbiological father. This model includes assortative mating and cultural transmission and allows tests for sex differences in all the effects. Given the inclusion of nonbiological fathers, we allowed the assortment and paternal cultural transmission paths to be different in the two types of families. Path coefficients are presented in Figure 2b; variance components, both unstandardized and standardized, and their confidence intervals (CIs), are presented in Table 7. Genetic factors and unshared environmental factors were the major sources of variance in antisocial behavior. Although shared

Table 6

Maximum Likelihood Estimates of Twin, Parent–Offspring and Spouse Correlations for Retrospective Reports of Conduct Disorder

	Twin correlations					Parent–offspring correlations				Spouse
	MZM	DZM	MZF	DZF	DZO	FS	FD	MS	MD	MF
Bio	.590	.351	.557	.361	.399	.123	.151	.169	.108	.175
BM	.681	.345	.623	.416	.438	-.035	-.015	.051	.211	.123
All	.598	.356	.570	.366	.409	.107	.139	.149	.124	.170

Note: MZM: monozygotic male twins; DZM: dizygotic male twins; MZF: monozygotic female twins; DZF: dizygotic female twins; DZO: dizygotic opposite-sex twins; FS: father–son; FD: father–daughter; MS: mother–son; MD: mother–daughter; MF: mother–father; Bio: twin families with both biological parents; BM: twin families with biological mother and nonbiological father.

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environmental factors significantly contribute to individual differences in antisocial behavior, when split into parental and nonparental sources, only the nonparental factors remain significant. Cultural transmission accounted for only 2% of the variance in both male and female offspring. Genotype–environment covariance resulting from the combined presence of genetic and cultural transmission was negative, given the negative causal paths from parents to offspring, but also nonsignificant. Although the genetic consequences of assortment only explained 3% of the variance, their effects were significant. Estimates of assortment and paternal cultural transmission were lower in step families compared to nuclear families, resulting in less genotype-environment covariance and larger estimated total variance. However, these parameters were not significantly different from those of nuclear families. When aggregated across sources, additive genetic factors accounted for 38% (95% CI 16%–55%) of the variance in antisocial behavior in males and 40% (CI 17%–56%) in females. The contribution of shared environmental factors was 23% (CI 11%–41%) in males and 18% (CI 8–35%) in females, and genotype-environment covariance was estimated at negative 6%–7% (CI males –15%–2%, females –18%–1%). Specific environmental factors explained the remainder of the variance, 39% (CI 30%–48%) and 42% (CI 33%–51%) in males and females respectively. Note that for the twin–parent analyses, the standardized estimates were obtained by dividing each of the variance components by the total variance minus the genotype-environment covariance.

We evaluated sex differences in (unstandardized) variance components by fitting a series of submodels. The contributions of cultural transmission did not differ according to the sex of the parent or the sex of the child. Although the total shared environmental variance or the additive genetic variance could be equated across sexes, both could not be equated simultaneously without significant loss of fit. The unique environmental variance components also differed significantly by sex.

Table 7

Standardized Variance Components from the Twin–Parent Model for Antisocial Behavior

	Variance components		Standardized variance components	
	Male	Female	Male	Female
Assortative mating	.018	.012	.031 .002 to .067	.028 006 to .061
	.016	*.010*	*.026* *.005 to .059*	*.024* *.005 to .056*
Common additive genetic	.258	.171	.445 .061 to .681	.398 .152 to .620
Male-specific genetic	.000	—	.000 .000 to .313	—
Unique environment	.232	.192	.400 .335 to .479	.448 .387 to .519
Shared environment	.113	.081	.194 .043 to .381	.189 .045 to .367
Cultural transmission	.012	.008	.020 .000 to .082	.018 .000 to .076
	.003	*.004*	*.005* *.000 to .046*	*.008* *.000 to .050*
Genotype–environment correlation	–.052	–.035	–.090 –.247 to .011	–.082 –.228 to .011
	–.019	*–.017*	*–.031* *–.103 to .174*	*–.039* *–.144 to .054*
Total Variance	.581	.429		
	.603	*.441*		

Note: Estimates in italics for families with nonbiological father

Discussion

Results of our analyses extend previous evidence on the contribution of genetic and environmental factors on antisocial behavior. Both additive genetic and shared environmental factors appear to contribute significantly to the variance of CD in males and females. In addition to data collected from the classical twin design, the twin–parent design allows us to identify the effects of assortative mating and cultural transmission. Both appear to explain a very modest proportion of the total variance in CD, and only the consequences of assortment were statistically significant. There is greater power to detect the effects of assortment, which directly derive from the spousal correlation, than there is to detect cultural transmission, which is inferred from the relative magnitude of parent–offspring and twin correlations.

At face value, it appears that adding the parental data to the twin design does not provide much additional information. However, this may depend of the phenotype of interest. If the assumptions of the twin design are (mostly) met, that is, no assortative mating, no genotype–environment covariance (which is theoretically possible but would imply that biological parents who provide the genetic make-up of their children do not have any environmental influence on their offspring for a phenotype that is at least in part genetic), and if it is not the case that both shared environment and dominance contribute to variability of the phenotype (which cannot be simultaneously estimated with data from the classical twin study), then one would not expect the inclusion of phenotypic data of the parents to alter the partitioning of the variance. In all other instances, however, the addition of the parents provides at least a check of some of the assumptions or allows for the simultaneous estimation of shared environment and genetical dominance assuming no cultural transmission.

Table 8 compares the unstandardized and standardized variance components from the twin–parent analysis with those obtained from fitting traditional models using twin data only. The estimates for the

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Table 8

Comparison of Variance Components Estimates for Conduct Disorder Obtained from the Twin–Parent Analyses with Those from the Twin Data Alone

	Unstandardized variance components				Standardized variance components			
	Twins and parents		Twins		Twins and parents		Twins	
	Male	Female	Male	Female	Male	Female	Male	Female
Total variance	58	43	58	47				
Additive genetic	23 9 to 37	19 7 to 29	20 7 to 31	14 4 to 23	38 16 to 55	40 17 to 56	34 13 to 52	29 8 to 47
Shared environment	14 7 to 24	8 4 to 15	15 6 to 27	14 6 to 23	23 11 to 41	18 8 to 35	26 10 to 44	29 12 to 47
Unique environment	24 20 to 28	19 17 to 22	23 20 to 28	20 17 to 23	39 30 to 48	42 33 to 51	40 33 to 48	42 36 to 50
Genotype–environment covariance	–3 –10 to 1	–3 –9 to 1			–6 –15 to 2	–7 –18 to 1		

unique environmental component were almost identical in the twin–parent and twin-only analyses, as would be expected. The slightly wider confidence intervals in the twin–parent analyses may reflect a larger number of parameters estimated in that model. The additive genetic variance component is greater in the twin–parent analysis than in the twin analysis, but includes the consequences of assortative mating. Conversely, the shared environmental component (including the effects of cultural transmission) is smaller in the twin–parent analysis. This comparison supports the notion that the estimates obtained from a classical twin study are biased by assortment, such that the additive genetic component is underestimated and the shared environmental component overestimated. The negative estimate for the genotype–environment covariance, which further biases the estimate of the shared environmental component, could reflect the presence of dominance or age by genotype interaction.

The standardized estimates for additive genetic, shared environmental and unique environmental contributions to variation in antisocial behavior from this analysis are consistent with those from a meta-analysis of 51 twin and adoption studies (Rhee & Waldman, 2002), when considering the sum of the additive and dominance effects. However, the shared environmental component appears to be slightly greater in our sample than in the meta-analysis. We are aware of only one other study that includes twins and their parents, but it focused on general versus disorder-specific externalizing disorders rather than CD specifically (Hicks et al., 2004). That study did not explicitly model and estimate genetic and environmental variance components; instead it estimated correlations between different types of relatives and calculated two kinds of heritability estimates based on the twin correlations and the parent–offspring correlations.

The current analyses augment the literature on the genetics of antisocial behavior by refining our estimates of the contributions of additive genetic, shared environmental and unique environmental factors to the variability in CD. However, these analyses need to be combined with the prospective longitudinal data in twins and the data on ASP in the young adults and parents, to provide a comprehensive understanding about the role of genes and environment in the developmental trajectory of antisocial behavior. Our results are consistent with those of many twin studies of CD in adolescence, whether assessed concurrently or retrospectively, in finding a significant effect of the shared family environment. The parent–offspring data augment this finding, and affect assessment of its clinical significance in several ways. First, allowing for any genetic consequences of assortative mating accounts for very little of the apparent shared environmental effect. Indeed, the parent–offspring correlations, though genetic, are relatively small and contribute little to the prediction of juvenile behavior. No less important, perhaps, is the implication that how parents behaved as adolescents themselves has little long-term impact on how they function as parents later in life. This finding is consistent with the view that a substantial part of adolescent misconduct is short-term, adolescence-limited and without pervasive influence on subsequent life course. Indeed, the apparent negative passive genotype-environment correlation, though theoretically possible, is psychologically unlikely and suggests that many genetic effects on adolescent CD are not only transient developmentally but also transient culturally. That is, genetic effects on behavior in one generation do not translate readily to genetic effects on the same behavior assessed in the next.

Limitations

The results of these analyses of the VTSABD data should be considered in the light of five potential limitations. First, the results may not be generalizable to the general population, as participation was limited to Caucasian twin families. Second, we used retrospective assessment of CD, which for the young adult twins corresponds to a 3- to 15-year recall and for their parents a 15- to 45-year recall. The accuracy

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with which one recalls adolescent behavior may vary with the time interval. However, the advantage of the current analysis is that the exact same measure was used for the parents and the offspring that is the optimal situation for parent–offspring designs. These models are based on the assumption that sources of variance as well as their magnitude are equal across generations. This assumption seems reasonable when the phenotype is comparable, as in the current example where we were asking parents and young adults retrospectively about a behavior during the same period of the life span. However, the instrument used to assess a particular behavior may differ across children, adolescents or adults. Potential failure of the assumption of equal phenotypic variances and variance components in parents and offspring might be addressed by adding genetically informative data from the two generations involved, or by adding longitudinal data that span the period from childhood/adolescence to adulthood. Such data are available in the VTSABD/YAFU, and future analyses will make use of these data. Third, while we included families in which a biological mother and nonbiological father was interviewed, the uneven split of families with two versus one biological parent(s) balances the power and precision of the estimates in favor of the nuclear families. Fourth, because of the high level of skewness in the symptom count for CD, we used a square root transformation of the data which were treated as continuous and normally distributed. Further analyses will include an ordinal treatment of the data. Finally, we fitted only twin–parent models which modeled phenotypic assortment and phenotypic cultural transmission, as they seem most plausible for CD. Other mechanisms of assortment and transmission, or a model including nonadditive genetic effects could be specified.

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