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Sex differences in the genetic and environmental influences on the development of antisocial behavior

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Abstract
The present study uses a population-based sample of 6,806 adult twins from same-sex and opposite-sex twin pairs to examine sex differences in the underlying genetic and environmental architecture of the development of antisocial behavior (AB). Retrospective reports of AB during three different developmental periods were obtained: prior to age 15 years (childhood), age 15–17 years (adolescent), and age 18 years and older (adult). Structural equation modeling analyses revealed that there was no evidence for sex-specific genetic or sex-specific shared family environmental influences on the development of AB; that is, the types of genetic and environmental influence were similar for males and females. For both sexes, a model that allowed for genetic influences on adolescent and adult AB that were not shared with childhood AB fit better than a model with a single genetic factor. In contrast, shared environmental influences on adolescent and adult AB overlapped entirely with shared environmental influences on childhood AB. Genetic factors played a larger role in variation in childhood AB among females, whereas shared environmental factors played a larger role among males. However, heritability of AB increased from childhood to adolescence and adulthood for both sexes, and the magnitude of genetic and environmental influences on adolescent and adult AB was approximately equal across sex. We speculate that sex differences in timing of puberty may account for the earlier presence of genetic effects among females.

One of the more intriguing results from research on genetic and environmental influences on antisocial behavior (AB) is the divergence of findings between studies of childhood and adolescent AB (e.g., conduct disorder [CD] and delinquency) and studies of adult AB (e.g., antisocial personality disorder and criminality). Research using children and adolescents typically find that both shared rearing environments and genetic factors are a significant source of family resemblance for AB (Edelbrock, Rende, Plomin, & Thompson, 1995; Eley, Lichtenstein, & Stevenson, 1999; Rowe, 1986). In contrast, studies of adult populations often find that genetic factors are the sole source of familiality (Cadoret, 1974; Cloninger & Gottesman, 1987; Crowe, 1974; DiLalla & Gottesman, 1989; Mednick, Gabrielli, & Hutchings, 1984). A meta-analysis of 24 twin and adoption studies of aggression revealed that age of subjects was a significant predictor of both heritability and shared envi-

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ronmental influences (Miles & Carey, 1997). Specifically, genetic and shared environmental influences contributed about equally to variation in aggression among studies of children and adolescents, whereas, among studies of adults, the average heritability was substantial, accounting for upwards of 50% of variation, and no shared environmental influences were detected.

Developmental Changes in Genetic Influence on Antisocial Behavior

However, the drawing of conclusions about developmental changes in the relative influence of genetic and shared environmental factors on AB from cross-study comparisons is problematic, because studies use different samples and different measures of AB. These differences in methodologies may introduce systematic biases. In addition, studies that focus on only one time point (i.e., childhood, adolescence, or adulthood) cannot determine the source of potential increases in genetic influence. For example, heritability of AB may increase in adulthood because of the presence of new genetic influences on adult AB that are not shared with child and adolescent AB. Alternatively, the same set of genetic factors may influence AB at all time points, but the magnitude of the genetic influence may increase with age. Finally, genetic influences might remain constant across time while environmental influences decrease in importance. This too would account for an increase in heritability. Ideally, prospective, longitudinal, genetically informative studies of AB are needed to disentangle these effects. Although several longitudinal twin and adoption studies are in progress (Hewitt et al., 1997; Plomin & DeFries, 1983), none currently have data from both childhood and adulthood. Thus, a second strategy is to collect retrospective reports of child and adult AB using the same adult sample.

At least two prior studies have used this method. A study of 32 monozygotic twin pairs reared apart reported heritability estimates of .41 and .28 for childhood and adult antisocial personality disorder (APD), respectively (Grove, Eckert, Heston, Bouchard, Segal, & Lykken, 1990). However, the 95% confidence intervals surrounding these estimates were large, and results from this small and rather unusual sample were not replicated in a second, large-scale study that used retrospective reports of AB from a sample of 3,226 male twin pairs from the Vietnam Twin Registry (Lyons, True, Eisen, Goldberg, Meyer, Faraone, Eaves, & Tsuang, 1995). This second study supported the hypothesis of increasing genetic effects on AB. Genetic influences on juvenile antisocial traits were quite modest, explaining only 7% of the variance, compared to 43% of variance explained by genetic factors for adult antisocial traits. Conversely, shared environmental influences explained 31% of the variation in juvenile antisocial traits, but only 5% of the variation in adult antisocial traits.

In the Lyons et al. (1995) study, the correlation between juvenile and adult antisocial traits was .44, and genetic and shared environmental influences each accounted for approximately one-third of this correlation. This study also found that the same set of genetic and shared environmental factors accounted for variation in both juvenile and adult antisocial traits; that is, there were no genetic or shared environmental influences specific to either juvenile or adult antisocial traits. Thus, the greater heritability estimate for adult antisocial traits was due to an increase in the magnitude of the genetic influence on adult antisocial traits, rather than to the presence of new genetic influences.

However, this study had two important limitations: first, only two points in time were used with a cutoff point of age 15, so the measure of "adult" antisocial traits included behaviors during middle and late adolescence, as well as adult behaviors. Because most adolescent twin siblings live together until age 18, combining behavior from ages 15 to 17 with the behavior after age 18 may have overstated the continuity of shared environmental factors between juvenile and adult AB. Likewise, combining adolescent and adult behaviors may have obscured any new genetic influence that occurs after age 18. Second, because the Vietnam Twin Registry was restricted to male twins, sex differences could

not be examined. It is possible that the genetic and environmental architecture underlying the development of AB differs for males and females. There is substantial evidence for sex differences in mean levels of both childhood CD and adult ASP (Cohen, Cohen, Kasen, Velez, Hartmark, Johnson, Rojas, Brook, & Streuning, 1993; Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, Wittchen, & Kendler, 1994; Robins & Reiger, 1991; Rutter, Giller, & Hagell, 1998; Simonoff, Pickles, Meyer, Silberg, Maes, Loeber, Rutter, Hewitt, & Eaves, 1997), but it is less clear whether sex differences in mean level are associated with sex differences in the sources of individual differences in AB.

Sex Differences

To date, only a few studies have examined sex differences in genetic and environmental influences on AB. One of the first studies of behavioral deviance using an unselected sample of 13-year-old twins found stronger evidence for genetic influences among males than among females (Graham & Stevenson, 1985). A more recent large-scale study using two separate samples of adolescent twins found that the heritability of nonaggressive delinquent behavior was higher among females than among males, although no sex differences emerged for aggressive delinquent behavior (Eley et al., 1999). Still other twin studies have failed to find evidence for significant sex differences in the heritability of AB (e.g., Slutske, Heath, Dinwiddie, Madden, Bucholz, Dunne, Statham, & Martin, 1997).

Results from adoption studies are similarly conflicting. A recent adoption study found that CD among adopted males was predicted by adoptive family environment alone, while biological background and gene–environment interactions predicted CD among females, suggesting greater genetic influence on CD among females (Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998). However, results from earlier adoption studies suggested that the same genetic factors predicted antisocial behavior in both males and females (e.g., Baker, Mack, Moffitt, & Mednick, 1989; Cadoret & Cain, 1980; Sigvardsson, Cloninger, Bohman, & Knorring, 1982) Thus, evidence for sex differences in the heritability of AB is equivocal. In addition, no published study has investigated sex differences in the underlying genetic and environmental influences on the development of antisocial behavior over time.

The Present Study

The present study uses data on behavior from three different developmental periods (prior to age 15, between the ages of 15 and 17, and age 18 and older) to examine four primary questions concerning sex differences in the genetic and environmental architecture underlying the development of AB. First we test whether the genetic and shared environmental influences that impact AB are the same for males and females (a *qualitative* sex difference). For example, it has been suggested that the greater prevalence of AB among males may be due to genes related to sex-specific hormones such as testosterone. If so, genetic factors influencing AB should overlap only partially, if at all, for males and females, because these genes would be expressed only among males. Similarly, we can test whether the shared environmental factors that influence AB are the same for males and females. Second, we examine whether genetic and environmental factors exert similar magnitudes of influence on AB for males and females (a *quantitative* sex difference). For example, if genes activated at puberty are an important influence on AB, genetic influences on early adolescent AB might be stronger for females than for males, given the earlier age of puberty experienced by females. Third, we investigate the underlying structure of genetic and shared environmental influences on the development of AB. Specifically, we test whether a single genetic factor, and/or a single shared environmental factor, can account for the variation in AB at all three time points and, conversely, whether genetic influences on antisocial behavior at the three time points are completely independent. Finally, we examine whether the same underlying genetic and environmental structure can account for continuity and change in AB for both males and females.

Previous analyses using the same-sex female twins from the present study found that the heritability of CD symptoms up to age 18 was .41, and shared environmental influences did not significantly differ from zero (Goldstein, Prescott, & Kendler, 2001). In contrast, a paper using a two-wave measurement model to assess genetic and environmental influences on CD among the same-sex male twins in the present study reported that genes and shared environments both explained approximately one third of the variation in CD, and both estimates were significantly greater than zero (Jacobson, Prescott, & Kendler, 2000). However, it should be noted that these reports were independent analyses of the same-sex pairs only, and thus cannot answer the question of sex differences directly.

Results from the present study may have implications for developmental taxonomies of antisocial behavior such as those proposed by Moffitt (1993) and DiLalla and Gottesman (1989). Longitudinal research has shown that although the majority of individuals diagnosed with adult APD met criteria for CD in childhood and adolescence, most individuals diagnosed with CD do not go on to become antisocial adults (Robins, 1978). This unidirectional effect, coupled with the finding that delinquent behavior in adolescence is a nearly universal phenomenon (e.g., West & Farrington, 1973) has led a number of researchers to propose the existence of two major types of delinquents: "transitory" or "adolescent-limited" (AL) delinquents, whose antisocial behavior is limited to the adolescent years, versus "continuous" or "life-course-persistent" (LCP) delinquents, whose antisocial behavior begins at a younger age and continues from adolescence into adulthood (DiLalla & Gottesman, 1989; Moffit, 1993). In addition to showing different patterns of AB across the life span, these two groups of individuals are further surmised to have different genetic and environmental etiologies (Rutter, MacDonald, Le Couteur, Harrington, Bolton, & Bailey, 1990; Wilson & Herrnstein, 1985).

In particular, the antisocial behavior of LCP individuals is thought to be influenced, in part, by relatively stable cognitive and personality characteristics, many of which are at least partly heritable (Plomin & McClearn, 1993; Rowe, 1994). To the extent that these characteristics also influence the development of AB, one might predict that a common set of genes would influence AB at all three time points. Thus, models constraining genetic influences on antisocial behavior to be independent across time periods should not fit the data well. In contrast to LCP antisocial behavior, AL delinquency is considered to be normative, particularly among males, and may be most strongly influenced by factors such as peer group composition, parental discipline and monitoring, and structural factors at the family, school, and community level (Moffitt, 1993). This would suggest that environmental influences, both shared and nonshared, would be more important for variation in child and adolescent AB than for adult AB.

However, it should be noted that the design of the present study does not directly test hypotheses concerning developmental taxonomies. Predictions pertaining to different genetic and environmental etiologies of LCP and AL individuals require either person-centered analyses that focus on group differences or growth curve analyses. Thus, although results from this study might be consistent with predictions based on developmental typologies, they do not test them directly. Instead, the present study focuses on a different developmental issue, namely, estimating the timing and relative importance of genetic and environmental influences on the development of AB and testing whether these influences are similar for males and females.

Methods

Sample and procedure

Data are from two longitudinal studies of psychiatric disorders in adult twins: a four-wave longitudinal study of female–female twins (the FF study; Kendler, Neale, Kessler, Heath, & Eaves, 1992) and an ongoing three-wave study of male–male and male–female twins (the MMMF study; Kendler & Prescott, 1999). Twins were ascertained via the Virginia Twin Registry (VTR, now part of the Mid-Atlantic Twin Registry). The VTR was

formed by a systematic search of all Virginia birth certificates since 1918. Twins were eligible for participation if one or both twins could be successfully matched to state records, they were Caucasian, and they were born between 1934 (FF) or 1940 (MMMF) and 1974. Inclusion in the FF study also required that both twins in a pair return a mailed questionnaire, whereas MMMF participants were first recruited by a telephone interview. Initial response rates were 64% (FF) and 73% (MMMF). Both studies were approved by the local Institutional Review Board, and subjects were informed about the goals of the study and provided verbal consent prior to phone interviews and written consent prior to in-person interviews and collection of DNA samples.

Data for the present study come from self-report questionnaires (SRQ) that were part of the wave 4 (FF) or wave 2 (MMMF) data collection. The FF study includes 2,164 twins originally interviewed at the first wave of data collection in 1988–1989 and 275 twins who were ascertained and studied subsequently. Of these 2,439 twins, 1,934 (79%) were successfully reinterviewed via telephone at the fourth wave of data collection in 1996–1997. Wave 4 participants were also sent an SRQ and were asked to fill it out and mail it back later. We received SRQs from 1,497 (77%) twins interviewed at wave 4. The majority of SRQs (85%) were returned within 3 months of the wave 4 interview. A minority of SRQs (1%) were answered orally.

The MMMF study includes 6,847 twins originally interviewed in 1993–1996. Eighty-three percent (N = 5,651) completed a second wave interview in 1994–1998, and 5,326 (94%) of those interviewed at wave 2 also completed an SRQ. The majority (80%) of wave 2 interviews were conducted face-to-face, with the SRQ filled out during the middle of the interview. An additional 1.8% completed the SRQ prior to the wave 2 interview ([M] = 8.6 days, SD = 19.7), and 18.2% returned it after the wave 2 interview (M = 3.53 months, SD = 8.9). In approximately one-quarter of cases, the SRQ was answered orally, most often because the wave 2 interview was given over the phone.

In total, we received SRQs from 6,823 individual twins. Eight twins were eliminated because of missing information concerning zygosity, and 9 twins were eliminated because of missing data for all three measures of antisocial behavior. The remaining 6,806 twins included the following: 2,580 twin pairs in which both twins had data on antisocial behavior (346 monozygotic female–female [MZF] pairs, 212 dizygotic female–female [DZF] pairs, 635 monozygotic male–male [MZM] pairs, 432 dizygotic male–male [DZM] pairs, and 955 dizygotic male–female [DZOS] pairs), 8 sets of triplets (creating 1 DZF, 9 MZM, 4 DZM, and 10 DZOS pairs), and 1,622 twins whose cotwins did not have valid data on antisocial behavior (206 MZF, 169 DZF, 299 MZM, 322 DZM, and 626 DZOS). Twins had an average of 13.5 (SD = 2.7, males) and 14.0 (SD = 2.5, females) years of education at the time of the SRQ. Female twins were slightly younger than male twins (M_{female} = 36.7, SD = 8.7; M_{male} = 37.1, SD = 9.1), F (1, 6,792) = 6.5, $p < .05$, and MZ twins were younger than DZ twins (M_{MZ} = 36.1, SD = 8.9; M_{DZ} = 37.4 SD = 8.9), F (1, 6,792) = 36.2, $p < .001$. However, the interaction between sex and zygosity for age was not significant, F (1, 6,792) = 0.02, $p > .50$, indicating that the difference in age between MZ and DZ twins was similar for males and females. Thus, our results concerning sex differences in estimates of heritability and shared environmental influences should not be biased by the slight age differences between males and females.

Measures

Zygosity. Zygosity of same-sex twin pairs who both participated at the initial assessment was determined by a combination of twins' responses to standard questions regarding twin similarity, photographs, and DNA typing. Assignment of zygosity for twins whose same-sex cotwins did not cooperate at wave 1 was done using a discriminant function analysis of items regarding physical similarity and twin self-report of zygosity, with DNA-typed twins as the comparison group.

Child and adolescent antisocial behavior. Items concerning antisocial behavior were identical in the FF and MMMF studies. Three measures of antisocial behavior (AB) were used: AB prior to age 15 years (child), AB age 15–17 years (adolescent), and AB age 18 years and older (adult). For AB prior to age 15 years, twins were asked to report how often they had engaged in 11 specific antisocial behaviors, corresponding to 11 of the 13 symptoms used to establish conduct disorder in the DSM-III-R (American Psychiatric Association [APA], 1987; the DSM-III-R item regarding forced sex was eliminated, given its low prevalence in other samples and potential offensiveness, and two of the DSM-III-R items regarding theft behavior were combined into a single item). Responses ranged from 0 (*never*) to 3 (*6 or more times* [or *often*]). Nine of the 11 items were repeated for AB age 15–17 years. (Two of the items, frequency with which the respondent lied and started fights, were only asked for the period prior to age 15 years.) A computer algorithm was applied to the frequencies for each item to indicate whether a given symptom was present (1) or absent (0). The algorithm was designed to match the wording of the DSM-III-R criterion as closely as possible. Summary scores of symptom counts were then calculated, and there was a possible range of 0–11 for AB prior to age 15 years and 0–9 for AB ages 15–17 years.

Adult antisocial behavior. The SRQ also included 17 items relating to 9 of the 10 symptoms for adult antisocial personality disorder (ASPD; APA, 1987), such as frequency of being irresponsible at work, frequency of arrest, and frequency of fighting. (Questions relating to the 10th adult symptom, failure to establish a monogamous relationship for at least 1 year, were not included.) All items were asked for the period age 18 years and older, and the scale for each item ranged from 0 (*never*) to 3 (*often*). For adult AB, a computer algorithm was used that combined these 17 items into the 9 possible symptoms. A given symptom was coded as present (1) if respondents met criteria for one or more of the behaviors that represented that particular symptom and absent (0) if none of the criteria were met. Symptoms were then summed to create a continuous measure, with a possible range of 0–9.

For all three measures of AB, if a respondent had missing data for a particular item, then he or she was given a score of 0 for that particular item. Twins with 50% or more items missing for a particular scale were given scores of missing for that scale (0.91% for child AB, 1.03% for adolescent AB, and 0.31% for adult AB). Ninety-five percent of the sample (N = 6,464) did not have missing data for any of the items. Only four individuals had more than two missing items for any of the three scales.

Table 1 presents the proportion of twins with each symptom count at each of the three age points, separately by zygosity and sex. The prevalence of AB at all three time points was similar across zygosity, although a greater proportion of males than females reported one or more AB symptoms. Given the small proportion of cases with more than five childhood AB symptoms, more than four adolescent symptoms, and more that six adult symptoms, categories were combined so that the number of symptoms ranged from 0 to ≥5 (childhood), 0 to ≥4 (adolescent), and 0 to ≥6 (adult); all variables were treated as ordinal in the structural equation modeling; and thresholds corresponding to each category were estimated.

Statistical analysis. Structural equation modeling analyses were conducted using the statistical package Mx (Neale, 1999). Models were fit to correlation matrices (shown in Appendix A) created from the raw data.[1] The full sex-limitation trivariate Cholesky model (Neale & Cardon, 1992) is shown in Figure 1. The diagram is shown for DZOS twin pairs, with the parameters for male twins on the left (designated by the subscript m), and those for female twins on the right (designated by the subscript f). The model allows for 3 underly-

1. Models were fit to correlation matrices, rather than to covariance matrices, because the variables were ordinal, and it is assumed that ordinal variables have variance = 1.0.

Table 1. *Prevalence of AB symptoms*

	Childhood AB[a]						Adolescent AB[b]						Adult AB[c]					
	Females			Males			Females			Males			Females			Males		
	MZ	DZ	DZOS	MZ	DZ	DZOS	MZ	DZ	DZOS	MZ	DZ	DZOS	MZ	DZ	DZOS	MZ	DZ	DZOS
N	878	580	1,317	1,569	1,183	1,217	880	578	1,318	1,565	1,180	1,215	898	593	1,320	1,574	1,185	1,215
0	81.9	81.7	82.7	51.7	53.1	53.6	84.9	83.6	81.0	58.8	57.5	54.8	65.9	61.6	66.7	49.0	47.9	46.3
1	11.7	12.1	12.7	26.3	26.1	24.1	12.2	11.6	14.3	25.2	26.0	26.8	21.8	22.1	19.4	23.3	22.1	23.0
2	4.9	4.7	2.7	11.7	11.2	12.1	2.3	3.5	3.6	10.2	10.0	11.0	6.9	9.3	7.3	13.8	13.1	13.1
3	0.9	1.0	1.3	6.0	5.7	6.2	0.6	0.9	0.6	3.5	4.0	4.8	3.1	4.4	3.2	6.4	7.3	8.9
4	0.6	0.2	0.5	2.8	1.9	2.3	0.1	0.5	0.5	1.5	1.9	1.9	0.8	1.0	1.6	3.4	4.9	3.5
5	—	0.2	0.1	0.8	1.5	1.3	—	—	0.1	0.2	0.4	0.4	0.8	1.3	1.3	2.4	3.0	3.0
6	—	0.2	—	0.3	0.3	0.3	—	—	—	0.4	0.2	0.2	0.6	0.2	0.3	1.0	1.0	1.7
7	—	—	0.1	0.3	0.1	—	—	—	—	0.1	—	—	0.1	0.2	0.2	0.5	0.4	0.3
8	—	—	—	0.1	—	0.1	—	—	—	0.1	—	—	—	—	—	0.1	0.3	0.2
9	—	—	—	0.1	—	—	—	—	—	—	0.1	—	—	—	—	—	—	—

Note: MZ, monozygotic twin; DZ, dizygotic twin from same-sex twin pair; DZOS, dizygotic twin from opposite-sex twin pair.
[a]Defined as behavior prior to age 15 years.
[b]Defined as behavior at ages 15–17 years.
[c]Defined as behavior at age 18 years and older.

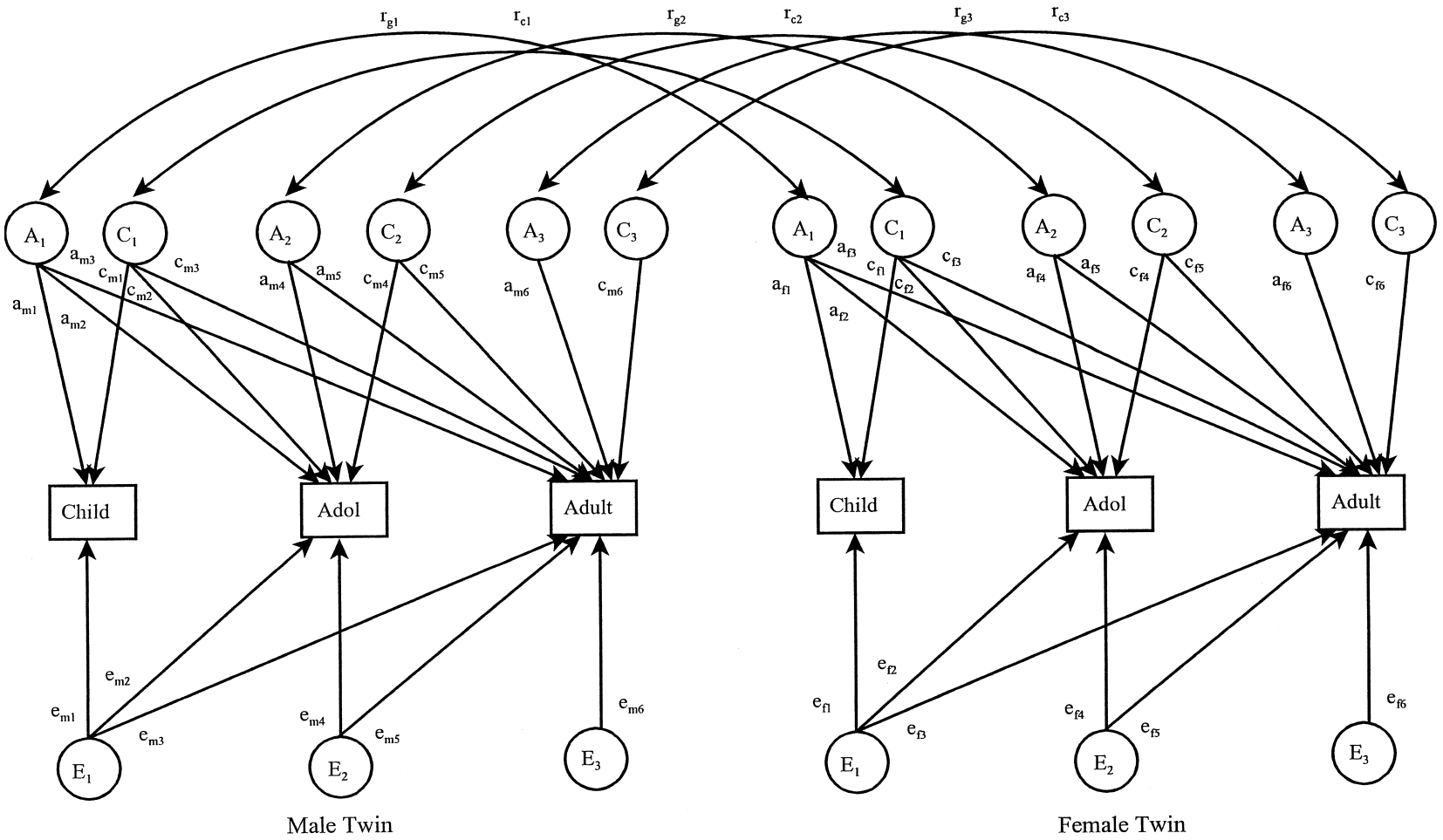

Figure 1. The trivariate Cholesky model. The parameters for female twins are shown on the right; those for male twins are shown on the left. Child, AB before age 15 years; Adol, AB age 15–17 years; Adult, AB 18 years and older; A, additive genetic influences; C, shared environmental influences; E, nonshared environmental influences.

ing genetic (A_1, A_2, A_3), shared environmental (C_1, C_2, C_3), and nonshared environmental factors (E_1, E_2, E_3) that represent influences that appear during childhood, adolescence, and adulthood, respectively.[2] In this full model, earlier influences are assumed to persist into later stages, although this is a testable assumption.

Variation in scores at a given time point is calculated as the sum of the squared parameters that point to that particular time point. For example, variation in childhood AB is $a_1^2 + c_1^2 + e_1^2$, and the heritability of childhood AB is simply a_1^2. For adolescent and adult AB however, variation arises both from influences specific to that time point as well from influences that are shared with previous time points. For example, the total variation in adult AB is $a_3^2 + a_5^2 + a_6^2 + c_3^2 + c_5^2 + c_6^2 + e_3^2 + e_5^2 + e_6^2$, and the heritability of adult AB is $a_3^2 + a_5^2 + a_6^2$. Thus, the heritability of adult AB can be decomposed into new genetic influences specific to adult AB (a_6^2), genetic influences that are common to adolescent but not childhood AB (a_5^2), and genetic influences that are common to AB at all three time points (a_3^2). Total shared environmental influences are calculated by $c_3^2 + c_5^2 + c_6^2$ and can be similarly decomposed. The hypothesis that a single set of genetic factors influences variation in behavior at all three time points is tested by fitting a nested submodel that constrains the paths a_4, a_5, and a_6 to zero, thereby eliminating any genetic influence on variation in adolescent or adult AB that is not shared with the genetic influence on child AB. Likewise, the hypothesis that there is a single set of shared environmental factors that influence AB is tested by a submodel constraining the paths c_4, c_5, and c_6 to be zero. In contrast, the hypothesis that genetic influences on AB are completely independent across time is tested by constraining the paths a_2, a_3, and a_4 to zero.

Among same-sex twin pairs, each of the three shared environment factors is correlated 1.0 across twins (r_{c1}–r_{c3} in Figure 1), regardless of zygosity. Each of the three genetic factors is correlated 1.0 for MZ twins and 0.5 for DZ twins (r_{g1}–r_{g3} in Figure 1) because identical twins share 100% of their genes, and fraternal twins, like nontwin siblings, share 50% of their segregating genes (on average). Nonshared environmental influences, by definition, are not correlated across twins. The model shown in Figure 1 is sex limited in two ways. First, the model estimates each of the individual parameters separately for males and females. Thus, the absolute magnitude of genetic and environmental influences on AB can vary across sex (a quantitative sex difference).[3] Second, this model can estimate the degree to which genetic influences are shared across males and females (a qualitative sex difference) by allowing each of the r_g coefficients to vary from 0.5 among DZOS twins. Similarly, the degree to which shared environmental influences are the same for males and females is tested or by allowing each of the r_c coefficients to vary from 1.0 among DZOS twins. Because of constraints imposed by the twin design, qualitative sex differences in genetic and shared environmental influences cannot be tested simultaneously.

The absolute fit of the model shown in Figure 1 is obtained by comparing the likelihood of this model to the likelihood of a model that fits the raw data perfectly (i.e., a saturated model), using the likelihood ratio test statistic (LRC), which is calculated as twice the difference in log-likelihoods (Neale & Cardon, 1992). The LRC is distributed as a chi-square value and is an indication of model fit, and a nonsignificant LRC indicates that the model fits the raw data well. Similarly, the relative fit of nested submodels can be obtained by calculating the LRC from the difference in log-likelihoods between the full model shown in Figure 1 and the particular submodel. When two competing, nonnested submodels both have nonsignificant LRCs, Akaike's Informa-

2. All latent factors were constrained to have a variance of unity.

3. Because of the inclusion of DZOS twin pairs, the parameters specific to each of the three time points (i.e., those with the subscripts 1, 4, and 6) were constrained to be nonnegative to avoid a situation in which parameters were estimated as negative for one sex but positive for the other. It should be noted that this constraint still allows genetic and environmental influences on covariation between time points to be negative in one sex and nonnegative in the other.

tion Criteria (AIC; Akaike, 1987) can be used. The AIC indicates the balance of goodness of fit and parsimony (Williams & Holahan, 1994), and models with more negative AIC values are preferred.

Results

Reliability

A subset of twins ($N = 127$ from the FF study; $N = 172$ from the MMMF study) completed a second SRQ an average of 28 days after the initial SRQ. The intraclass correlations for AB prior to age 15, age 15–17, and age 18 years and older were .77, .71, and .67, respectively, among the FF sample and .72, .71, and .69, respectively, among the MMMF sample. The age of the twins used for reliability varied from 21 to 57. Therefore, we were able to examine whether age affected short-term reliability by taking the absolute difference between the number of symptoms obtained from the original SRQ and the number of symptoms obtained from the reliability SRQ and regressing this difference score onto age. This was done for each of the three variables, separately by gender. Age did not significantly predict any of the absolute difference scores for either males or females (t range = −1.22–0.20; all $p > .20$).

Within-person correlations

Within-person polychoric correlations were calculated using SAS v.8.01. The correlation between childhood and adolescent AB was .56 for males and .51 for females. Correlations between childhood and adult AB were .39 (males) and .42 (females), and the correlations between adolescent and adult AB were .56 and .49 for males and females, respectively. The fact that the correlations between adolescent and adult AB were nearly identical to the correlations between child and adolescent AB demonstrates the cross-time validity of our measures of AB. Specifically, if measures of adult AB were tapping a different construct than that measured by child and adolescent AB, the correlations between adolescent and adult AB, which use different scales to assess AB, should be lower in magnitude than the correlations between child and adolescent AB, which use the same items.

Twin correlations

Inspection of the twin correlations presented in Appendix A can provide some expectation of what results from the structural equation modeling analyses may be.[4] For example, if MZ correlations are greater than DZ correlations, genetic influences are suggested. To the extent that DZ correlations are greater than one-half the MZ correlation, shared environmental influences are suggested. Finally, a comparison of the same-sex and opposite-sex twin correlations can give some indication of the likelihood of significant sex differences.

Examination of the correlations suggests a few general patterns. First, DZOS correlations are not substantially lower than the same-sex DZ correlations, suggesting that there are no qualitative differences in genetic or shared environmental influences on AB. To take the most extreme example, if the genetic and environmental influences on AB were completely different for males and females, correlations among DZOS twins would be zero. Instead, correlations among DZOS twins range from approximately .10 to .25. However, there is some suggestion of quantitative sex differences (i.e., sex differences in the magnitudes of genetic and environmental influences on AB). Most notably, for child AB, the MZF correlation (.39) is substantially

4. Formal structural equation modeling programs such as Mx are preferred to a visual inspection of correlations for three primary reasons. First, standard errors on correlations can vary widely across zygosity group, making determinations of statistically significant differences difficult. Mx takes into account sample size when estimating parameters and calculates confidence intervals around the parameters. Relatedly, Mx provides fit statistics that indicate the goodness of fit of each model and allows for specific hypothesis testing. Finally, in multivariate models, both cross-twin, within-trait (e.g., Child_1 with Child_2) and cross-twin, cross-trait (e.g., Child_1 with Adol_2) correlations are taken into account simultaneously when estimating parameters. Thus, simple visual comparisons of pairs of correlations can sometimes yield misleading results.

greater than the DZF correlation (.09), indicating strong genetic influence and no shared environmental influence among females. In contrast, the MZM correlation (.34) is only slightly greater than the DZM correlation (.30), suggesting that the primary sources of familial resemblance for child AB among males are shared environmental influences.

Second, especially among male twins, MZ correlations increase from childhood to adulthood, while the DZ twin correlations are similar across developmental periods. This suggests that the heritability of AB increases with age. Finally, a comparison of MZ and DZ cross-twin, *cross-trait* correlations (e.g., Child_1 with Adol_2) shows that MZ cross-twin, cross-trait correlations are uniformly higher than their DZ counterparts, indicating some overlap of genetic influence.

Trivariate Cholesky analyses

Prior to beginning our primary analyses, we examined whether thresholds were significantly different across Twin 1 and Twin 2 (same-sex pairs only), across zygosity, or across sex. Based on the LRC statistic, neither equating thresholds across Twin 1 and Twin 2 among same-sex pairs nor equating thresholds across zygosity within sex resulted in a significant deterioration in fit (LRC = 42.85, df = 60, p = .95; LRC = 110.63, df = 120, p = .72, respectively). In contrast, equating thresholds across sex resulted in a highly significant deterioration in fit (LRC = 982.05, df = 135, $p < .001$), indicating that the prevalence of AB varied significantly across sex. Therefore, thresholds were constrained to be equal within genders but were allowed to vary across sex for all subsequent analyses.

Results from the primary model-fitting analyses are presented in Table 2. Model 1 is the full trivariate Cholesky shown in Figure 1, with parameters allowed to vary across sex, but with r_g = 0.5 and r_c = 1.0 among the DZOS twins. This model fit the data very well (p = .82). Next we ran three models estimating each of the three r_g parameters in DZOS twins (data not shown). For all three models, the r_g was estimated at close to 0.50, and none of the models was a significant improvement in fit compared to Model 1 (LRCs < 1.0, df = 1, all $p > .50$). We ran a similar series of models estimating the r_c among DZOS twins, and again, none of the models offered a significant improvement in fit compared to Model 1 (LRC < 1.00, df = 1, all $p > .50$, results not shown). Thus, there was no evidence for *qualitative* sex differences in genetic and environmental influences on the development of AB. In contrast, Model 2, which tested the hypothesis that there were no *quantitative* sex differences, did fit the data significantly more poorly than Model 1 (LRC = 33.91, df = 15, $p < .003$), indicating that the magnitude of the genetic and environmental influences on the development of AB varied significantly across males and females. Thus, parameters were allowed to vary across males and females in all subsequent models.

Models 3–6 tested whether the development of AB could be explained by a single set of genetic factors (Models 3 and 5) and/or by a single set of shared environmental factors (Models 4 and 6). These analyses were first conducted separately by sex. Among males, the hypothesis that there was a single set of genetic factors that influenced variation in AB at all three time points could be rejected (Model 3) because the fit of this model was significantly worse than Model 1 (LRC = 8.63, df = 3, $p < .05$). In contrast, the model that allowed for a single set of shared environmental influences on AB among males (Model 4) did not fit the data significantly more poorly than Model 1 (LRC = 3.86, df = 3, p = .28). Among females, neither the model with a single genetic factor (Model 5) nor the model with a single shared environmental factor (Model 6) fit the data significantly more poorly than Model 1 (LRC = 6.07, df = 3, p = .11, Model 5; LRC = 4.50, df = 3, p = .21, Model 6). However, a model simultaneously testing for a single set of genetic and a single set of shared environmental factors (Model 7) did fit the data significantly more poorly (LRC = 28.18, df = 6, $p < .001$), indicating that there was some familial influence on adolescent and/or adult AB among females that was not shared with the familial influence on childhood AB. Model 6 had a more negative AIC value, indicating that the model with a

Table 2. *Model-fitting results*

	Model	Absolute Model Fit			Relative Model Fit			
		−2 LL	*df*	*p* Value[a]	LRC[b]	*df*	*p* Value	AIC
1	Full model	40,297.24	20,277	.82	—	—	—	−181.82
2	All parameters: males = females	40,331.15	20,292	.44	33.91	15	.003	−177.91
3	Single genetic factor (males)	40,305.87	20,280	.72	8.63	3	.03	−179.19
4	Single shared environmental factor (males)	40,301.10	20,280	.81	3.86	3	.28	−183.96
5	Single genetic factor (females)	40,303.31	20,280	.77	6.07	3	.11	−181.75
6	Single shared environmental factor (females)	40,301.74	20,280	.80	4.50	3	.21	−183.32
7	Single genetic factor, single shared environmental factor (females)	40,325.42	20,283	.37	28.18	6	.001	−165.64
8	**Single shared environmental factor (both sexes)**	**40,302.25**	**20,283**	**.83**	**5.01**	**6**	**.54**	**−188.81**

Note: LRC, Likelihood ratio chi-square; AIC, Akaike's Information Criterion. The best-fitting model is indicated in bold.
[a]The significance with the model is based on a comparison with the saturated model. The fit of the saturated model was −2 LL = 40,149.06, *df* = 20,112.
[b]The LRC is obtained from a comparison with the full model (Model 1).

single shared environmental factor among females was a better fit than the model of a single set of genetic factors among females. Thus, the next model (Model 8) tested whether a single set of shared environmental influences could be used to explain the development of AB for both males and females simultaneously. This model fit the data as well as Model 1 (LRC = 5.01, $df = 6$, $p = .54$), and based on the AIC criteria it was the best-fitting, most parsimonious model.

Finally, we tested whether we could constrain the genetic influences on AB to be independent (i.e., uncorrelated) across time periods. Compared with Model 8, eliminating genetic influence on covariation across time resulted in a significant deterioration in fit for both males (LRC = 18.30, $df = 3$, $p < .001$) and females (LRC = 9.20, $df = 3$, $p < .03$; results not shown), indicating that genetic factors did account for at least some of the stability of AB from childhood to adolescence to adulthood.

Figure 2 presents the standardized parameter estimates from Model 8. Parameters for males are shown on the left; those for females are shown on the right. Table 3 presents the heritability and estimates of shared and nonshared environmental influences at each of the three time points based on the parameters shown in Figure 2. As can be seen in Table 3, heritability estimates for both sexes were substantially lower for childhood AB than for adolescent or adult AB (.06 vs. .41 and .40 for males; .29 vs. .50 and .42 for females). In addition, the heritability estimate for childhood AB was greater for females (.29 [95% confidence interval {CI} = .10; .34]) than for males (.06 [95% CI = .00; .24]). In contrast, the estimate of shared environmental influence on childhood AB was greater among males (.28 [95% CI = .09; .38]) than among females (.09 [95% CI = .02; .26]). Nonshared environmental influences on AB ranged from .43 to .66, indicating that approximately one-half of the variation in AB at each of the three time points was explained by nonshared environmental factors. This was true for both sexes.

To test the significance of the sex differences in the heritability and shared environmental influences on childhood AB, we ran a series of post hoc analyses equating the heritabilities and shared environmental estimates at each time point across sex. In this case, a significant LRC in comparison with Model 8 indicates that estimates cannot be equated across sex. The heritability of childhood AB was significantly different for males and females (LRC = 4.09, $df = 1$, $p < .05$), although heritabilities for adolescent and adult AB were not (LRC = 0.71, $df = 1$, $p = .40$ for adolescents; LRC = 0.15, $df = 1$, $p = .70$ for adults). Shared environmental estimates differed significantly across sex for both child and adult AB (LRC = 4.27, $df = 1$, $p < .05$; LRC = 6.16, $df = 1$, $p < .01$, respectively), but not for adolescent AB (LRC = 0.03, $df = 1$, $p = .82$). Thus, post hoc analyses confirmed that genetic influences were in fact stronger among females than among males for childhood AB and that shared environmental influences on childhood AB were stronger for males than for females.

Equal environments

The equal environments (EE) assumption in twin studies is that MZ and DZ twins are equally correlated in their exposure to environmental influences that impact the behavior or trait in question. If this assumption is violated, higher correlations among MZ twins may be due to environmental factors, rather than genetic factors, and heritability may be overstated. To examine whether the higher heritability of childhood AB among females may be due to violations of the EE assumption, we used multiple regression to examine whether similarity of childhood environment predicted within-pair differences in childhood AB once zygosity was controlled for. The EE variable was a composite of four standard questions asking "how often while growing up . . . ": " . . . did you share a room," " . . . did you have the same classroom at school," " . . . did you have the same friends," and " . . . did you dress alike." These questions were asked during the wave 1 interview (FF) or wave 2 (MMMF) interview. Possible composite scores ranged from 4 to 16, with higher scores indicating less equal environments. Scores were averaged across twins to create a

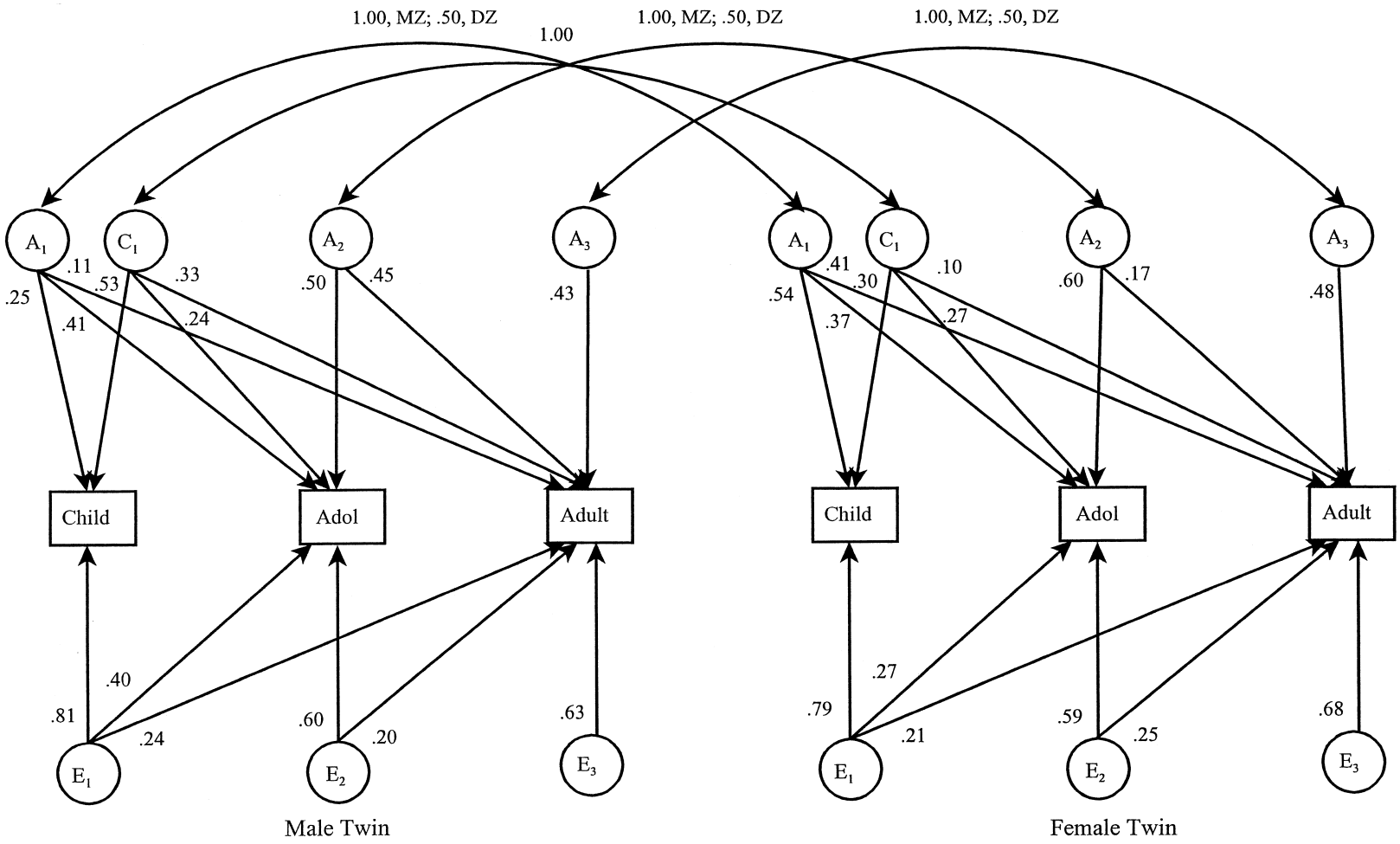

Figure 2. The standardized parameter estimates from the best-fitting trivariate model. The parameters for the female twins are shown on the right; those for male twins on the left. Child, AB before age 15 years; Adol, AB age 15–17 years; Adult, AB 18 years and older; A, additive genetic influences; C, shared environmental influences; E, nonshared environmental influences.

Table 3. *Estimates of genetic and environmental influences from best model*

	h^2		c^2		e^2	
	Males	Females	Males	Females	Males	Females
Child	.06	.29	.28	.09	.66	.62
95% CI	.00; .24	.10; .34	.09; .38	.02; .26	.62; .72	.61; .75
Adolescent	.41	.50	.06	.07	.53	.43
95% CI	.31; .53	.34; .55	.01; .15	.00; .19	.46; .59	.30; .58
Adult	.40	.42	.11	.01	.49	.57
95% CI	.28; .52	.33; .50	.05; .21	.00; .65	.46; .53	.56; .66

Note: h^2, heritability; c^2, estimate of shared environmental influence; e^2, estimate of nonshared environmental influence.

single score, and analyses were restricted to same-sex twin pairs with complete data on both the EE variable and childhood AB (*N* pairs = 298 MZF, 199 DZF, 642 MZM, 433 DZM).

MZ twins did report more similar childhood environments than DZ twins (M_{MZ} = 7.70, *SD* = 1.8; M_{DZ} = 8.75 *SD* = 1.8), F (1, 1,571) = 112.91, $p < .001$, and females reported more similar childhood environments than males (M_{female} = 7.88, *SD* = 1.9; M_{male} = 8.23, *SD* =1.9), F (1, 1,571) = 11.92, $p < .001$. However, the interaction between zygosity and sex was not significant, F (1, 1,571) = 0.25, p = .62, indicating that the difference between MZ and DZ twins was similar for males and females. Moreover, when the within-pair, absolute difference score for childhood AB was regressed onto the EE variable and zygosity, EE did not predict twin pair differences for either females (t = 0.75, p = .45) or males (t = 0.47, p = .64). Thus, the higher heritability of childhood AB among females was not due to violations of the EE assumption among females.

Discussion

Our results demonstrate both similarities and differences across sex in the genetic and environmental architecture underlying the development of antisocial behavior (AB) from childhood to adulthood. For both sexes, this study supports two primary hypotheses: (a) genetic factors increase in relative importance from childhood to adolescence and adulthood, and (b) shared environmental influences on AB are most important during childhood. Among males, heritability increased from .06 to approximately .40; for females, heritability increased from .28 to .42–.50. Among males, shared environmental influences accounted for over one-quarter of the variation in childhood AB but only about 10% of the variation in adolescent and adult AB. Shared environmental influences among females were weak overall, explaining less that 10% of the variance in childhood and adolescence and less than 1% of the variation in adult AB. This pattern of increasing heritability and decreasing shared environmental influences supports the conclusions drawn from previous cross-sectional studies of juvenile and adult AB (e.g., Cadoret, 1974; Cloninger & Gottesman, 1987; Crowe, 1974; DiLalla & Gottesman, 1989; Edelbrock et al., 1995; Eley et al., 1999; Mednick et al., 1984; Rowe, 1986) and is consistent with results both from a meta-analysis (Miles & Carey, 1997) and a prior retrospective study of adult male twins (Lyons et al., 1995).

Although the sample contained nearly 1,000 DZOS twin pairs, we could find no evidence that the specific genes and shared environments that influence AB were qualitatively different across sex. This is consistent with evidence that the psychosocial risks associated with problem behavior are qualitatively similar for males and females (Rowe, Vazsonyi, & Flannery, 1994). In addition, our results indicate that there are similarities across sex in the underlying structure of genetic and

environmental influences on the development of AB. For both males and females, the best-fitting model allowed for unique genetic influences on adolescent and adult AB, in addition to those that persist from child AB, and for a single set of shared environmental influences. These results are partly consistent with the prior study of male twins from the Vietnam Registry, which found that *both* the genetic and shared environmental factors that influenced adult AB overlapped completely with those factors that influenced juvenile AB (Lyons et al., 1995).

Genetic and environmental influences on the development of antisocial behavior

In our study, the finding of unique genetic influences on adolescent AB that are *not* shared with child AB may reflect the influence of genetically influenced biological processes that are first activated at puberty. For example, there is evidence from animal and human studies that hormone levels, such as testosterone, are related to aggression among males (Albert, Jonik, Watson, Gorzalka, & Walsh, 1990; Brooks & Reddon, 1996; Dabbs & Morris, 1990; Olweus, Mattson, Schalling, & Löw, 1988; Wagner, Beuving, & Hutchinson, 1979), although it should be noted that studies of hormonal effects on aggression among humans are inconsistent (see Archer, 1991; Jacobson & Rowe, 2000, for review). A second explanation is that by middle to late adolescence, adolescents have greater latitude in selecting environments, such as peer groups, that are more consistent with their genetically influenced characteristics (Scarr & McCartney, 1983). Because there is evidence that peer selection in adolescence is heritable (Rowe, 1989), the new genetic influence on adolescent AB may be related to these genetic influences on peer selection.

The explanation for the unique genetic influences on *adult* AB is somewhat less certain. There is evidence, both in our sample and others, that some antisocial adults do not report childhood or adolescent AB. Although these individuals are not discussed in Moffitt's (1993) typology, DiLalla and Gottesman (1989) have suggested that these "late bloomers" may have an even higher heritability of AB than the LCP delinquents. Thus, the new genetic influence on adult AB may be related to this "late bloomer" effect. In addition, genetic influences on many phenotypes turn on and off throughout the life span, and to date, little is known about the mechanisms that may responsible for age-related genetic effects.

Finally, our results may be supportive of the concept of LCP antisocial behavior because a single set of genetic factors did influence the development of AB across time, and a model suggesting completely independent genetic factors was rejected. Thus, there is evidence that certain genetically influenced characteristics are related to antisocial behavior during both adolescence and adulthood. Possible characteristics include both physiological factors and personality characteristics, such as impulsivity and sensation seeking, both of which are to some degree heritable (Zuckerman, 1994). Nevertheless, it should be reiterated that the present study cannot directly address hypotheses driven from developmental theories such as those expounded by Moffitt (1993) and DiLalla and Gottesman (1989) because this study focused on changes in genetic and environmental influences over time, not on how genetic and environmental factors may vary across different typologies of antisocial individuals. Such questions are of considerable interest, however, and work is currently under way to test these hypotheses using more appropriate statistical methods.

Sex differences in the development of antisocial behavior

Although the present study suggests that the underlying structure of genetic and environmental influences on AB is similar across sex and that genetic and environmental factors that influence AB are not qualitatively different among males and females, there was evidence for sex differences in the magnitudes of genetic and environmental influences on the development of AB (i.e., a quantitative sex difference). Based on post hoc analyses, the primary sex difference was in the magnitude

of genetic and environmental influences on child AB. Specifically, the heritability of child AB was significantly greater for females (.29) than for males (.06). Conversely, shared environmental factors accounted for 28% of the variation in child AB among males, but only 9% of the variation among females.

This result is consistent with a recent study using two separate, large-scale twin samples that found higher heritabilities and lower estimates of shared environmental influences on adolescent delinquent behavior for females (Eley et al., 1999). The finding that shared environmental factors may be more important for males than for females is also consistent with the hypothesis that although the types of psychosocial and environmental risk factors for adolescent problem behavior are similar across sex, males may have greater vulnerability to these factors (Rutter et al., 1998). Further, male twins may be more likely to commit antisocial activities jointly during childhood and early adolescence, which might also account for an additional source of shared environment (Rowe, 1983). This sex difference may extend to adult AB as well because our study showed significantly higher estimates of shared environmental influences on adult AB among males. However, it should be restated that shared environmental influences on adult AB were relatively weak for both sexes.

What is perhaps most intriguing about these results is that the sex differences, particularly the sex differences in heritability estimates, diminish with age, as depicted in Figure 3. The pattern of decreasing sex differences in heritability estimates suggests that the genetic factors that influence AB throughout the life course simply become penetrant at a later age among males than among females. To clarify this phenomenon, Figure 3 has apportioned each heritability estimate into the proportion due to genetic factors arising during childhood, adolescence, and adulthood. As discussed previously, the heritability of child AB is due solely to genetic influences present during childhood, and these influences were stronger for females than for males; hence, the higher heritability of child AB among females than among males. By adolescence, childhood genetic factors contributed about equally to heritability across sex, although the magnitude of unique genetic influences arising during adolescence was somewhat greater among females, again accounting for the slightly higher heritability estimate. By adulthood, however, total heritability estimates were similar for males and females. Moreover, the proportion of the heritability due to unique genetic factors arising during adulthood was approximately equal across sex. The striking sex difference was in the magnitude of childhood and adolescent genetic influences on adult AB.

For females, childhood genetic factors played a larger role in the heritability of adult AB than did genetic factors arising during adolescence. For males, the converse was true: genetic factors first present during adolescence played a larger role in the heritability of adult AB than did childhood genetic factors (see Figure 3). These results indicate that genetic influences on AB appear earlier among females than among males and have greater impact on the continuity of antisocial behavior into adulthood. This finding is consistent with the above hypothesis that certain genetic influences on antisocial behavior are first activated at puberty because females reach puberty earlier, on average, than males (Crockett & Petersen, 1987; Tanner, 1968).

Finally, the present study also found evidence for significant sex differences in the mean level of AB. Although thresholds for AB did not vary across zygosity, constraining thresholds to be equal across male and female twins resulted in a highly significant deterioration in fit. This is consistent with the nearly universal finding that being male is one of the strongest predictors of antisocial behavior (Cohen et al., 1993; Kessler et al., 1994; Robins & Reiger, 1991; Rutter et al., 1998; Simonoff et al., 1997).

Strengths and limitations

The present study has a number of different strengths, such as the use of data from both males and females from a large, population-based sample of twins. Moreover, the inclu-

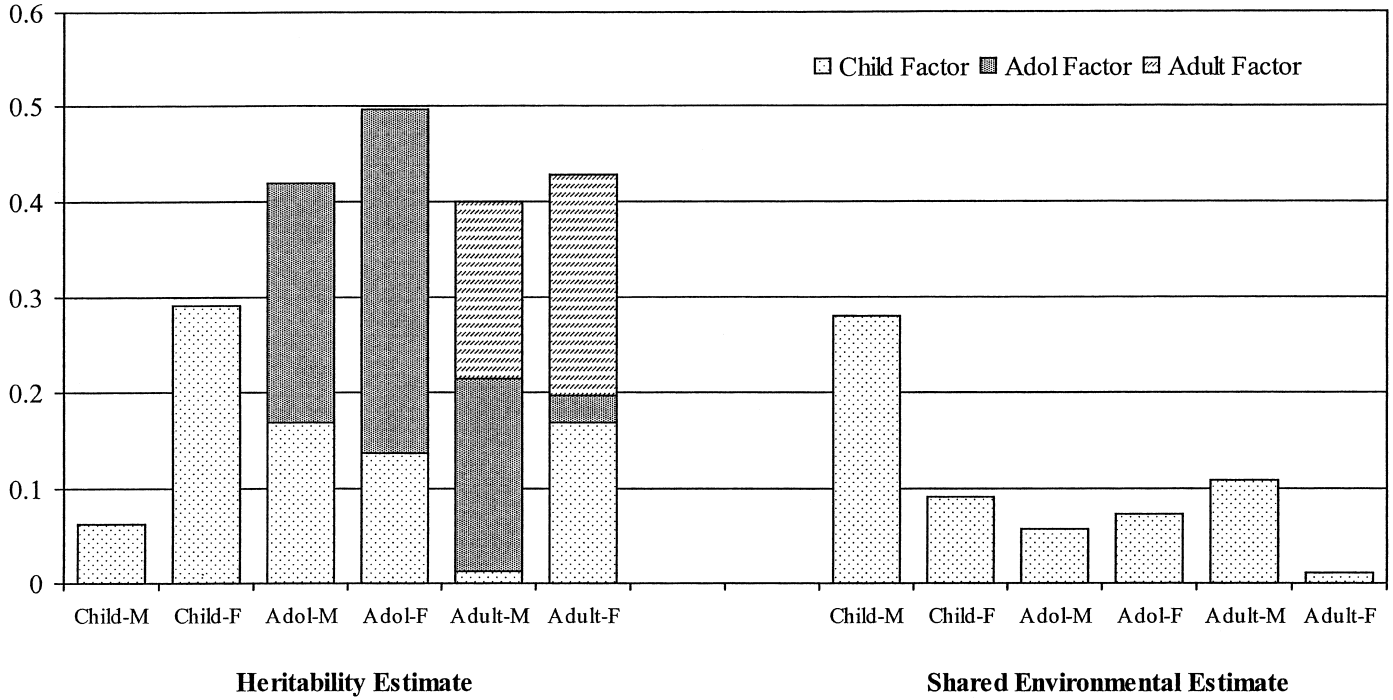

Figure 3. The estimates of heritability and shared environmental influences. M, male; F, female; Child, AB before 15 years; Adol, AB age 15–17 years; Adult, AB 18 years and older. The heritability at each age has been apportioned into those genetic influences first present during childhood, those first present during adolescence, and those that first appear during adulthood. All shared environmental influence comes from environmental factors first present during childhood.

sion of opposite-sex twins allowed us to differentiate between qualitative and quantitative sex differences in genetic and environmental influences on AB. A second strength is the use of self-report questionnaires to assess AB. Evidence suggests that individuals are more willing to report negative behaviors in self-report questionnaires than in structured face-to-face interviews (Siemiatycki, 1979), and individuals can also be seen as more valid reporters of their own behavior, as compared to parent or teacher reports (Rutter et al., 1998). Finally, the present study was able to use the same sample to assess antisocial behavior during three different time periods, suggesting that the higher heritability of adult AB typically found in cross-sectional research is not likely to be due to differences in methodologies or sample composition.

One limitation, however, is the use of retrospective reports from adult twins to assess childhood and adolescent AB. Although retrospective reports have some advantages, such as the fact that all individuals have passed through the age of risk so developmental differences in rates of CD cannot bias results, a prospective study of adolescents found that self-reports of delinquent activities up to age 13 years that are assessed at age 13 correlated only weakly with similar reports obtained when subjects were 18 years old (Henry, Moffitt, Caspi, Langley, & Silva, 1994). Thus, factors relating to recall may be biasing estimates of heritability and shared environmental influences. For instance, it is likely that genetic factors influence accuracy of recall. Thus, the heritability of AB may be partly due to genetic influences on memory, rather than to genetic influences on antisocial behavior per se. However, if genetic influences on memory were confounded with genetic influences on antisocial behavior, one might expect that heritability would increase with length of time since the behavior. Yet the present study indicates that heritability is higher for more recent events (i.e., adult AB), which is opposite of that prediction. In addition, our estimates of heritability at each age are consistent with estimates obtained from other samples using concurrent measures of adolescent and adult AB (e.g., Eley et al., 1999), further suggesting that retrospective reporting is not biasing our results. Finally, it is unclear how biases due to the use of retrospective reports could account for the sex differences in estimates of heritability found here because the average age difference between males and females in this study was less than 6 months.

A second limitation relating to the use of retrospective reports is that although we were able to classify behavior into three distinct periods (prior to age 15, between ages 15 and 17, and age 18 years and older), we did not have information as to the precise ages in which individuals engaged in antisocial behavior (we do have information concerning age of initiation of AB in the first wave of the MMMF study, but parallel information is lacking for females). In the study of reliability of retrospective reporting mentioned above, Henry et al. (1994) suggested that individuals likely remember a delinquent event truthfully, but may have more difficulty identifying the precise *age* in which the event occurred. This may make the distinction between childhood and adolescent AB in the present study somewhat blurred. As with above, however, it is unclear how this possible bias might manifest itself as the differential pattern of sex differences seen here. Nevertheless, prospective longitudinal studies that sample twins on a more systematic basis will be better able to determine more exactly the developmental periods in which the etiology of AB differs across sex and the ages in which genetic influences increase. Longitudinal studies beginning with preadolescents might be particularly helpful for testing the hypothesis that the observed sex difference in the timing of genetic influences found in the present study is due to sex differences in the timing of puberty.

A third limitation may be the broad age range of the sample, because twins in the present study ranged in age from 20 to 62 years. Thus, some of the younger twins may still be at risk for the development of symptoms relating to adult AB. Age may also influence accuracy of recall. However, we did not find evidence of aging effects on short-term reliability among either males or females, and a previous analysis of long-term

reliability among the same-sex male twins in this sample found that, if anything, older twins were more reliable reporters of their antisocial behavior than younger twins (Jacobson, Prescott, & Kendler, 2000). On the other hand, there is some evidence among the same-sex male twins that the magnitude of shared environmental influences on variation in juvenile AB has increased with more recent cohorts (Jacobson, Prescott, Neale, & Kendler, 2000), and cohort differences in mean levels of antisocial behavior for both males and females are commonly observed (Bureau of Justice Statistics, 1999; Robins, 1998). However, it is difficult to conceive how any potential cohort effects might account for the *differential* sex differences in heritability estimates of AB across childhood, adolescence, and adulthood. Nevertheless, prospective longitudinal studies following a single birth cohort of twins are sorely needed.

A final limitation is that results are based on a population of Caucasian twins born in Virginia. Thus, results may not generalize to samples from different cultures or in different ethnic groups. Despite these limitations, this is one of the first published studies to examine genetic and environmental influences on the development of antisocial behavior within the same sample, and it is the first study, to our knowledge, to look at sex differences. A better understanding of how these genetic and environmental factors vary in timing and importance across sex may shed light on similarities and dissimilarities of etiologic factors important in the development of antisocial behavior.

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Appendix

Table A.1. *Observed twin correlations*

	Child_1	Adol_1	Adult_1	Child_2	Adol_2	Adult_2
Female–female MZ twins						
Child_1	1.0000					
Adol_1	0.4537	1.0000				
Adult_1	0.2791	0.4134	1.0000			
Child_2	0.3939	0.2394	0.2226	1.0000		
Adol_2	0.2664	0.5331	0.1865	0.5212	1.0000	
Adult_2	0.2518	0.3025	0.3609	0.4664	0.5424	1.0000
Female–female DZ twins						
Child_1	1.0000					
Adol_1	0.3472	1.0000				
Adult_1	0.3947	0.4104	1.0000			
Child_2	0.0905	0.3708	0.0728	1.0000		
Adol_2	0.0875	0.2704	0.1582	0.5599	1.0000	
Adult_2	0.1959	0.0700	0.3447	0.3545	0.4973	1.0000
Male–male MZ twins						
Child_1	1.0000					
Adol_1	0.5812	1.0000				
Adult_1	0.4706	0.5822	1.0000			
Child_2	0.3363	0.2116	0.1808	1.0000		
Adol_2	0.2730	0.4642	0.3420	0.5780	1.0000	
Adult_2	0.2583	0.3699	0.4981	0.3557	0.5657	1.0000
Male–male DZ twins						
Child_1	1.0000					
Adol_1	0.5717	1.0000				
Adult_1	0.4427	0.5965	1.0000			
Child_2	0.3020	0.1968	0.1428	1.0000		
Adol_2	0.1715	0.2809	0.1564	0.5676	1.0000	
Adult_2	0.2369	0.2498	0.3451	0.4076	0.5308	1.0000
Male–female DZ twins						
Child_1	1.0000					
Adol_1	0.5249	1.0000				
Adult_1	0.3593	0.5685	1.0000			
Child_2	0.2431	0.1922	0.1016	1.0000		
Adol_2	0.1901	0.3123	0.2498	0.5282	1.0000	
Adult_2	0.1037	0.1809	0.2143	0.4568	0.5052	1.0000

Note: MZ, monozygotic; DZ, dizygotic.