

Sex Differences, Assortative Mating, and Cultural Transmission Effects on Adolescent Delinquency: A Twin Family Study

Jeanette Taylor, Matt McGue, and William G. Iacono

University of Minnesota, Minneapolis, U.S.A.

The twin study literature contains inconsistencies regarding both the estimates of genetic, shared, and nonshared environmental influence on delinquency and sex differences in the etiological influences on delinquency. Additionally, little is known about assortative mating and cultural transmission effects on delinquency. The present study was aimed at examining the etiology of delinquency in adolescence and examining assortative mating and transmission effects. To this end, we applied the social homogamy structural equation model to self-reported delinquency data from 486 families with 17-year-old male and female twins. We expected stronger shared environmental influences on delinquency among girls than boys and stronger environmental than genetic effects on adolescent delinquency in general. The results indicated nonsignificant sex differences in the estimates of genetic and environmental influences on delinquency. The best-fitting model suggested that 18%, 56%, and 26% of the variance in delinquency among both boys and girls is associated with additive genetic, nonshared environmental, and shared environmental factors, respectively. This finding was consistent with our expectation that environmental factors play an important role in adolescent delinquency. Parental cultural effects accounted for a small part of the transmission of delinquency within families while assortative mating was, in part, accounted for by cultural and social background factors. Future work should focus on identifying important environmental factors related to delinquency.

Keywords: Delinquency, adolescent, genetic, environment, twin family method.

Abbreviations: DBI: Delinquent Behavior Inventory; DZ: dizygotic; MTFS: Minnesota Twin Family Study; MZ: monozygotic.

The topics of delinquency and antisocial behavior have occupied researchers in psychology and sociology for several decades. Recent developmental theories suggest various pathways to delinquency in adolescence including maladaptive interactions between the child and the parents (e.g., Patterson, 1986) and mimicking the antisocial behavior of peers who have taken up adult-like behaviors such as smoking, drinking, and staying out late (Moffitt, 1993). Research, however, has not clarified the nature of the causal variables that contribute to the development of delinquency. It is possible to examine the relative importance of broad classes of causal influences (e.g., unique environmental experiences) on a trait that could inform the focus of future research on specific causal influences (e.g., peer pressure). The aim of this investigation is on the etiological influences that contribute to individual differences in delinquency in adolescence—a problem affecting as many as 80% of teenagers (Moffitt, 1993). For ease of exposition, the terms delinquency and delinquent behavior will be used broadly to refer to antisocial behavior and conduct problems of childhood or adolescence.

We can begin to understand the etiology of a behavior

or trait by examining the extent to which individual differences in the behavior or trait are associated with variation in additive genetic, shared environmental, and nonshared environmental influences. Additive genetic influence (A) is attributed to the genetic similarity among family members; shared environmental influence (C) is attributed to environmental influences common to members of a family (e.g., parenting style); and nonshared environmental influence (E) is attributed to environmental factors or experiences that have unique effects on members of a family (e.g., life events, peer interactions, differential parental treatment). Genetic and shared environmental influences contribute to phenotypic similarity among family members whereas nonshared environmental influences contribute to phenotypic differences among family members on a given trait. The relative contribution of these three influences on individual differences in behavior can be assessed by examining biologically related individuals such as first-degree relatives or monozygotic (MZ) and dizygotic (DZ) twins.

With few exceptions, reports on twin studies of delinquency from the past two decades are in good agreement in suggesting that genetic and nonshared environmental factors are part of the etiological picture for this kind of behavior. However, simple statements about the influence of genes and environment to individual differences in delinquency are difficult to formulate given the generally poor agreement among reports

Requests for reprints to: Matt McGue, Department of Psychology, University of Minnesota, 75 East River Road, Minneapolis, MN 55455, U.S.A.
(E-mail: mmcgue@tfs.psych.umn.edu).

regarding the relative magnitude of the influence of additive genetic and environmental factors on delinquency. For example, one can find support for the position that genetic effects on delinquency are modest at best in the presence of shared environmental effects (e.g., Lyons et al., 1995; Silberg et al., 1994) as easily as one can support the position that genetic effects account for the lion's share of the variance in delinquency (e.g., Rowe, 1983; Slutske et al., 1997). Certain methodological issues may be contributing to some of the inconsistencies in the literature. For example, shared environmental influence estimates tend to be of somewhat smaller magnitude among studies using self-reports (e.g., Taylor, McGue, Iacono, & Lykken, in press) than among those based on parent-reported data (Edelbrock, Rende, Plomin, & Thompson, 1995; Eley, 1997; Eley, Lichtenstein, & Stevenson, 1999; Silberg et al., 1994, 1996; see Eaves et al., 1997 for some exceptions). The literature is inconsistent even when limited to an examination of self-reports: genetic influence estimates are somewhat higher from samples aged 13–18 (Rowe, 1983, 1986; Taylor et al., in press) than from those with preadolescents in the sample (Eaves et al., 1997).

The literature also contains inconsistencies regarding sex differences in the relative contributions of genetic and environmental factors on delinquency. Surprisingly, all but one report in the literature included girls (or women) in their sample (Lyons et al., 1995, used an all-male sample). Part of the problem in clarifying this issue is that not all investigators reported tests of sex differences. For example, Rowe (1983, 1986) reported only sex-specific models that showed similar parameter estimates for boys and girls whereas Silberg et al. (1996) reported sex-specific models showing high genetic influence for boys and high shared environmental influence for girls but no indication of the significance of the sex differences. Grove et al. (1990) and Edelbrock et al. (1995) reported similar estimates of genetic effects from sex-corrected data. The remainder of studies are about equally split, with some reporting a good fit of a combined sex model despite substantially different sex-specific model results (Eaves et al., 1997; Silberg et al., 1994 [adolescent subsample]; Slutske et al., 1997; Taylor et al., in press) and others reporting a poor fit of a combined sex model (Eley et al., 1999; Silberg et al., 1994 [child subsample]). Studies with larger samples (e.g., over 500 twin pairs) fairly consistently suggest that sex differences in the relative magnitudes of genetic and environmental effects are not significant. Thus, issues of statistical power may be contributing to the inconsistencies in the literature regarding sex differences in the etiological influences on delinquency.

Additional studies of adolescent boys and girls are needed if the literature is to provide unambiguous information regarding sex differences and the relative magnitude of genetic and environmental influences on delinquency. Studies of older adolescent twins are lacking in the literature but would be particularly useful given that older adolescents have passed through much of the risk period for delinquency and are therefore less likely to contribute censored observations to a data set. Additionally, studies using self-reports are surprisingly lacking in the literature. Self-reports have some advantages over data from other sources: namely, delinquent acts often go undetected by outside observers (e.g., police, parents, teachers), which make self-reports an attractive approach to many delinquency researchers.

As such, studies using self-reports from older adolescent twins would make a needed contribution to the literature. Finally, studies that provide new information about the etiology of delinquency would make the greatest contribution to the literature.

To date, the majority of research on individual differences in delinquency has been limited to nontwin sibling studies and traditional twin studies. Twin studies have the advantage over nontwin sibling studies of allowing for the separate estimation of genetic and shared environmental influences through the comparison of MZ twins (who share 100% of their genes) to DZ twins (who share, on average, 50% of their genes). Although increasing sample size is one way to increase the statistical power in a twin study, the detection of subtle effects (such as shared environmental influence in the presence of genetic influence) is facilitated by the use of a design that increases the amount of genetically informative data to be modeled (Heath, Kendler, Eaves, & Markell, 1985). The twin family design gains power over the twin study method by including data on the twins and their biological parents (e.g., Eaves, Last, Young, & Martin, 1978). The added power derived from twin family data could help clarify the magnitude of genetic and environmental effects associated with individual differences in delinquency among boys and girls.

Family data are also useful for examining assortative mating (the tendency for spouses to be similar on a particular trait) and transmission effects, and very little is known about these types of effects on delinquency. Parents share genes with their biological children but they also provide an environment shared by the family, and each of these sources of influence potentially contribute to the phenotypic similarity among family members. Social homogamy refers to the notion that people prefer mates who come from a similar geographic location, similar culture, and similar social class as their own (Rao, Morton, & Yee, 1976). Thus, it is assumed under the social homogamy twin family model that we select mates based on shared social characteristics. Under this model, the similarity between spouses on a given trait (i.e., assortative mating) is assumed to result from the association of the trait with individual differences in social background factors. That is, if our cultural beliefs, where we live, and our level of economic resources are related to how we behave and how we function psychologically, then people who select mates with similar cultural beliefs, geographic location, and SES to their own will likely be similar on traits and behaviors associated with those factors. Under the social homogamy model, we further assume that parent-offspring phenotypic similarity results from the transmission of genetic factors and/or shared environmental factors that are either (a) common to both generations (e.g., social class, geographic location) or (b) unique to the offspring generation (e.g., parenting style). Finally, the social homogamy twin family model requires the assumption of system equilibrium—that is, the variable under study and the relative contributions of A, C, and E to individual differences in that variable are assumed to be similar across generations. The social homogamy model is thought to be well suited to data on variables such as delinquency where sizable shared environmental effects may be expected (Neale & Cardon, 1992; Rao et al., 1976).

The present investigation focused on an examination of etiological influences on delinquency among older adolescents. As part of an earlier study, we analyzed data

from same-sex twins in their late teens reporting on 36 mild to serious delinquent behaviors (Taylor et al., in press). In that study, we employed univariate models to estimate the magnitude of genetic and environmental influences on delinquency among twins who had passed through much of the risk period for the onset of delinquency. The present study extends our previous work and the work of others through the inclusion of data from the twins' biological parents, which will provide an increase in power to partial out genetic and shared environmental effects and will offer some initial insight into the effects of assortative mating and cultural transmission on delinquency within families. Based on our previous work and other reports in the literature, we expected that shared environmental influences would account for more of the variance in delinquency among girls than boys. In line with recent developmental theories of delinquency, we expected greater estimates of environmental (as compared to genetic) influence on adolescent delinquency in general.

Method

Male and female 16- to 18-year-old ($M = 17$; $SD = 0.65$ for females, 0.45 for males) MZ and DZ reared-together (same-sex) twins and their biological parents participating in the Minnesota Twin Family Study (MTFS) served as subjects. The MTFS is a community-based longitudinal study of the etiology of substance use and related disorders among twins and their families. Twin pairs were identified through Minnesota state birth records for the years 1972 through 1977 (male adolescent cohort) and 1975 through 1979 (female adolescent cohort). Families were excluded from participating if the twins were adopted, if the family lived farther than a day's drive from Minneapolis, or if the twins had a physical or intellectual disability that precluded their completion of the day-long intake assessment. Consistent with the demographics of Minnesota during the birth years sampled, nearly all of the twins (98%) were Caucasian.

Zygosity of the twins was established using the following three methods: (1) parents completed a zygosity questionnaire regarding the twins' physical similarity and the frequency with which people confused them, (2) staff rated the zygosity of the twins based on various points of physical similarity (e.g., eye color and ear shape), and (3) zygosity was determined using an algorithm based on ponderal index, cephalic index, and fingerprint ridge count. Any discrepancy in the three methods was resolved through a serological examination of 12 blood group antigens and protein polymorphisms. The first-born twin was always designated twin A; the other was designated twin B.

All families in the cohort were screened for inclusion in this analysis. Fifteen per cent of families with female twins and 12% of families with male twins participating in the MTFS were excluded from the present analysis due to unavailability of the delinquency data. Specifically, 30 families with female twins and 16 families with male twins were excluded because we did not have data on both twins; 12 families with female twins and 15 families with male twins were excluded because we did not have data on at least 1 biological parent. The biometrical analyses were conducted using data on 1803 individuals from 345 four-member families (data on both twins and both biological parents) and 141 three-member families (data on both twins and one biological parent). Three-member families did not necessarily constitute single-parent families; rather this label simply identifies families with valid data on only one biological parent. The female sample included 191 four-member families (125 MZ; 66 DZ) and 80 three-member families (55 MZ; 25 DZ). The male sample included 154 four-member families (100 MZ; 54 DZ) and 61 three-member families (43 MZ; 18 DZ).

Measure

As part of the MTFS psychological assessment, twins and their parents were administered a self-ratings booklet that contained a 36-item delinquency scale, which we have named the Delinquent Behavior Inventory or DBI (Gibson, 1967). The DBI is a checklist of minor (e.g., riding a bike recklessly; skipping school), moderate (e.g., shoplifting; destroying property), and more serious (e.g., using a weapon in a fight; struggling with a police officer) delinquent behaviors. Although it is made up of a variety of delinquent behaviors that could be classified as aggressive versus nonaggressive or overt versus covert, the DBI has a unitary factor structure and high internal consistency reliability ($\alpha = .96$ for boys and $.97$ for girls). As such, it is analyzed as a single composite scale. The DBI correlates moderately with other indices of antisocial behavior assessed in our adolescent participants such as symptoms of antisocial personality disorder ($r = .46$ and $.51$ for boys and girls, respectively) reported during a structured diagnostic interview. Participants were instructed to consider each item on the DBI if it occurred during adolescence or while "growing up." Although many of the DBI items constitute arrestable offenses, they refer to behaviors most likely committed in or before adolescence and are not likely to be endorsed as adult behaviors.

The DBI was scored dichotomously with 1 point given for each endorsed item and no points given for nonendorsed items. In the male study, which began several years before the female study, the DBI response format was dichotomous (yes vs. no). In the female study, the response format was trichotomous (no; yes, once; yes, more than once). For this analysis, female family participant answers were dichotomized (i.e., no vs. yes, once and yes, more than once) in an attempt to make the male and female family data comparable. The DBI score was simply the sum of the responses (maximum score of 36) with high scores indicating a greater variety of delinquent behaviors committed in or before adolescence.

Analyses

Structural equation modeling using maximum-likelihood estimation was done with the Mx software program (version 1.38, Neale, 1997; users of LISREL are referred to Neale & Cardon, 1992, for a discussion of this model using that program). Figure 1 illustrates the social homogamy transmission model (adapted for Mx from Neale & Cardon, 1992) that was fitted separately to the male and female family data and to all family data in a fully constrained model (i.e., only one estimate of each model parameter was obtained from the male and female family data considered jointly). The social homogamy model allowed us to examine the relative contribution of additive genetic, shared, and nonshared environmental influences on delinquency because, like a traditional univariate model, the twin family model provides an estimate of A, C, and E (but it does so by utilizing covariance information from two sources: twins and parent-offspring). Additionally, we were able to examine (1) the relative effects of cultural transmission on the shared environment of the twins and (2) the contribution of assortative mating to delinquency. Cultural transmission effects on a phenotype reflect cultural, socioeconomic, and geographic location characteristics for each parent that are similar in the offspring (presumably because they were passed on from the parents) and have some effect on the phenotype of interest in both generations. Assortative mating, when present in traditional twin studies, leads to underestimation of the influence of genes on a trait because it can inflate the genetic similarity among DZ twins over the 50% similarity assumed in a classical twin analysis. Thus, high assortative mating parameter estimates have implications for the heritability estimate from the model.

Table 1
DBI Means (and Standard Deviations) for Parents and Twins

Data type	Father	Mother	Twin A	Twin B
<i>Male families</i>				
Raw				
MZ	4.48 (3.9)	1.80 (1.9)	4.35 (3.9)	4.03 (3.5)
DZ	4.95 (5.7)	1.42 (1.9)	4.66 (4.5)	5.11 (5.2)
Log ₁₀				
MZ	.63 (.33)	.35 (.28)	.61 (.33)	.59 (.32)
DZ	.64 (.34)	.28 (.29)	.61 (.37)	.65 (.34)
<i>Female families</i>				
Raw				
MZ	6.68 (4.8)	2.72 (2.6)	2.96 (3.5)	2.91 (3.9)
DZ	6.96 (5.7)	2.70 (2.6)	3.53 (3.6)	3.64 (3.7)
Log ₁₀				
MZ	.79 (.30)	.45 (.33)	.47 (.33)	.46 (.32)
DZ	.79 (.32)	.46 (.31)	.54 (.33)	.56 (.30)

Log₁₀ = Log-transformed (log₁₀[x + 1]).

Results

The upper and lower portion of Table 1 presents the means and standard deviations for the male and female families, respectively. The DBI data were transformed (log₁₀ [x + 1]) prior to analyses due to the substantial positive skew of the data. Descriptive data for both the raw and the log-transformed DBI are reported in the table; results from the biometrical analyses are reported only for the transformed DBI scores. The lower mean DBI scores for male (relative to female) family parents may be due to the aforementioned difference in response format across the male and female studies. The data in Table 1 reveal the greater incidence of delinquent behavior among males relative to females commonly found in studies of antisocial behavior. It should be noted that the standard deviations for the male and female samples were quite similar, and mean differences on the DBI across sexes would not affect our analyses of the variances and covariances.

A single correlation for each relative pair (e.g., MZ twins, mother-offspring, etc.) was derived from a model that constrained the parent-offspring and twin covariances to be the same in three- and four-member families within each sex. Table 2 presents both the formulas for the expected correlation under the social homogamy model as well as the maximum likelihood estimates of the twin and parent-offspring correlations. The common variance-covariance model allowed us to examine possible sampling differences between families with data on two parents versus those with data on only one parent given that the fit of the model was based on data from both types of families considered jointly. The overall fit of the common variance-covariance model was assessed with the chi-square (χ^2) goodness-of-fit statistic. Large (statistically significant) chi-square values implicate differences in the covariance matrix for the two types of families. The model was not rejected, indicating that a single variance-covariance matrix fit data from both three- and four-member families. The spouse and parent-child log-DBI correlations were similar for male and female families, but the twin correlations suggested genetic influence on delinquency for boys only.

The social homogamy model, depicted for DZ families in Fig. 1, was defined by the equations given in Table 2. The model for MZ families would deviate from the DZ model in that only one genetic path would run from each parent to a single latent A variable shared by the MZ co-

twins. In the social homogamy model, the additive genetic path (a), the shared environment path (c), and the nonshared environment path (e) are each constrained to

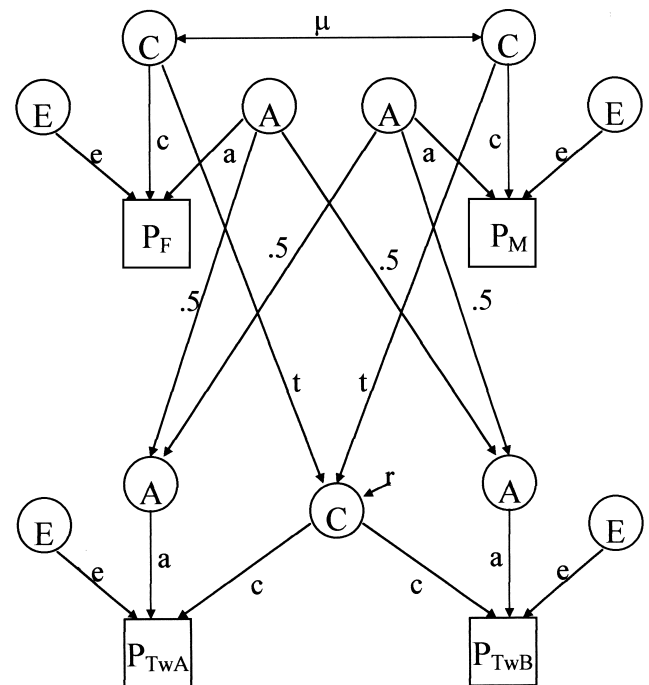


Figure 1. The social homogamy model for DZ twin families. The upper portion of the model represents the parent generation while the lower portion represents the offspring generation. The model assumes that the relative contribution of A, C, and E to the phenotype is similar across generations (i.e., there is system equilibrium). The model further assumes that phenotypic similarity between parents (assortative mating) arises from a similarity in social class, geographic location, and cultural beliefs (notice that the μ parameter path connects the shared environmental latent variables in the parent generation) and that these effects may account for some portion of the shared environment of their offspring (effects that are estimated as the t parameters which represent the transmission of shared environmental effects associated with social class, geographic location, and cultural factors from parents to offspring). P_F = father's phenotype; P_M = mother's phenotype; P_{TwA} = twin A's phenotype; P_{TwB} = twin B's phenotype; A = additive genetic effects; C = shared environmental effects; E = nonshared environmental effects; t = cultural transmission effects; μ = assortative mating; r = residual shared environmental effects.

Table 2
Expected Correlation Equations and Observed Log-DBI Correlations among Family Members

Relatives	Expected	Observed	
		Male families	Female families
Spouses	$c^2\mu$.226	.347
Mother-Child	$.5a^2 + c^2(t + t\mu)$.200	.201
Father-Child	$.5a^2 + c^2(t + t\mu)$.133	.181
MZ Twins	$a^2 + c^2x$.579	.462
DZ Twins	$.5a^2 + c^2x$.105	.482
$\chi^2(df)$		17.8 (31), $p = .97$	27.0 (37), $p = .89$

For the expected correlations between twin pairs, $x = 2(t^2) + 2(c^2\mu) + r$, where r is residual shared environmental effects. The equations for the expected correlation between each relative pair together define the social homogamy model fit to the family data. The observed correlations were derived from a model with a single variance-covariance matrix fit to data from both four- and three-member families within each sex. Statistics for the fit of the single variance-covariance model are given in the last line of the table. a = additive genetic path; c = shared environment path; e = nonshared environment path; μ = assortative mating path; t = cultural transmission path.

Table 3
Twin Family Model-fitting Analyses of Log-DBI Scores for the Fully Constrained (Combined-sex) Data

Model	A	C	E	μ	t	r	$\chi^2(df)$	p	AIC	$\chi^2\Delta(df)$	Sex diff. $\chi^2\Delta(df)$
ACE	.134 (.18) [.02-.32]	.162 (.26) [.16-.38]	.237 (.56) [.46-.68]	1.0	.187	1.17	63.14 (76)	.85	-88.86	—	5.83 (6)
AE	.207	—	.249	—	—	—	104.80 (80)	.03	-55.22	41.64 (1)*	0.11 (5)
CE	—	.190	.250	.804	.289	1.03	67.95 (77)	.76	-86.05	4.81 (1)*	4.00 (5)
$\mu = 0$.180	.102	.237	—	.000	1.99	90.35 (77)	.14	-63.65	27.21 (1)*	4.08 (5)
t = 0	.170	.144	.229	1.0	—	1.19	66.90 (77)	.79	-87.10	3.76 (1)	2.78 (5)
r = 0	.207	.129	.232	1.0	.000	—	88.31 (77)	.18	-65.69	25.17 (1)*	-0.20 (5)

The proportion of variance (in parentheses) and its 95% confidence interval (in brackets) is provided for A, C, and E for the best-fitting model. A = additive genetic; C = shared environmental; E = nonshared environmental; μ = assortative mating; t = cultural transmission; r = residual shared environmental; AIC = Akaike's Information Criterion. Sex diff. $\chi^2\Delta(df)$ = the test for differences between the fully constrained and sex-specific model.

* $p < .05$ (when testing the fit of reduced models against the full model).

be equal across generations (reflecting the system equilibrium assumption of the model). Further, the model specifies an assortative mating parameter (μ) between the latent shared environment variables of the parents as well as cultural transmission paths (t) to the latent shared environment of the twins. The residual shared environment path (r) captured the shared environmental variance not associated with cultural transmission and assortative mating. Values of the μ parameter were restricted to fall between 0 and 1.0.

Table 3 presents the social homogamy model-fitting results for data from the male and female families considered jointly in a fully constrained model. Table 4 contains the results of the sex-specific models. In the constrained model, a single estimate of each parameter (a, c, e, etc.) is estimated from all of the family data (regardless of the sex of the twins). In constraining the parameter estimates across sex, the model has increased statistical power to detect subtle effects such as shared environmental influence in the presence of additive genetic influence because of the increase in the sample size. The constrained model also provides a test of sex differences in the relative influences of genetic and environmental factors since the model will have a poor fit to the data if strong sex differences in the variance structure of the trait exist.

The best-fitting models among those presented in Tables 3 and 4 were selected using information from several selection statistics. The overall fit of each model was assessed with the chi-square goodness-of-fit statistic described earlier. Fitted full (e.g., ACE) and reduced (e.g., CE, t = 0, etc.) models were compared using the chi-square difference test, wherein the difference in chi-square values ($\chi^2\Delta$) is tested for significance using as its degrees of freedom the difference in the degrees of freedom of the two models. The reduced model is usually preferred when $\chi^2\Delta$ is not significant. The chi-square difference test was also used to select between the fully constrained model (in which parameters are required to be the same for both sexes) and the sex-specific models (in which the parameters may vary by sex). The tests for sex differences are presented in Table 3, where the fully constrained model-fitting results are summarized. Finally, Akaike's Information Criterion ($AIC = \chi^2 - 2df$; a measure that balances goodness-of-fit relative to model parsimony) was used to select the best-fitting reduced model as indicated by the lowest AIC value (Akaike, 1987). For each best-fitting model presented in Tables 3 and 4, the proportion of variance accounted for by A, C, and E is shown in parentheses below the estimate; the 95% confidence band for the proportion of variance is given in brackets.

Table 4
Twin Family Model-fitting Analyses of Log-DBI Scores in Sex-specific Models

Model	A	C	E	μ	t	r	χ^2	df	p	AIC	$\chi^2\Delta(df)$
Male families											
ACE	.184	.118	.232	1.0	.002	1.57	30.19	32	.56	-33.81	—
AE	.210	—	.248	—	—	—	41.50	36	.24	-30.50	11.31 (1)*
CE	—	.159	.264	.859	.359	1.31	36.83	33	.30	-29.17	6.64 (1)*
$\mu = 0$.191	.045	.242	—	.000	9.22	35.72	33	.34	-30.28	5.53 (1)*
t = 0	.184	.118	.232	1.0	—	1.56	30.19	33	.61	-35.81	0.00 (1)
	(.33)	(.14)	(.53)								
	[.19-.46]	[.02-.26]	[.41-.68]								
r = 0	.213	.109	.235	1.0	.000	—	37.77	33	.26	-28.24	7.58 (1)*
Female families											
ACE	.000	.211	.239	.787	.254	0.893	27.12	38	.91	-48.88	—
AE	.204	—	.249	—	—	—	63.19	42	.02	-20.81	36.07 (1)*
CE	—	.211	.239	.787	.254	0.893	27.12	39	.92	-50.88	0.00 (1)
		(.44)	(.56)								
		[.30-.55]	[.45-.70]								
$\mu = 0$.000	.211	.239	—	.361	0.838	50.55	39	.10	-27.45	23.43 (1)*
t = 0	.154	.167	.226	1.0	—	1.03	33.93	39	.70	-44.07	6.81 (1)*
r = 0	.000	.141	.275	1.0	.680	—	50.74	39	.10	-27.27	23.61 (1)*

For abbreviations see Table 3.

* $p < .05$ (when testing the fit of reduced models against the full model).

Sex differences in the estimates of variance associated with genetic and environmental influences among males and females were examined by fitting a fully constrained model in which one estimate of each parameter was obtained from all the family data. Table 3 presents the results from the fully constrained models. The AE model was the only fully constrained model that did not provide an adequate fit to the data. All of the reduced models, save that which set the cultural transmission path to zero, produced a significant decrease in fit from the full ACE model. We chose the ACE model as the best-fitting model because, although not significantly different in fit from the model with no cultural transmission ($t = 0$), it provided a better fit using the AIC criterion. In the best-fitting model, a little more than half of the variance in self-reported delinquency was associated with nonshared environmental effects, and only 18% of the remaining variance was associated with additive genetic effects. The assortative mating parameter, μ , was estimated at its upper boundary limit of 1.0 in most of the solutions, indicating some role for social class, geographic location, and cultural factors in the similarity in delinquency observed between spouses¹. However, there was evidence for only modest contributions of social background and cultural factors in the transmission of delinquency from parents to offspring.

Despite the large number of twin families included in this investigation, the power to detect a significant difference between the fully constrained and the sex-specific models was still fairly low. As can be seen by the chi-square difference statistics given in the last column of Table 3, the fit of the fully constrained models differed

from that of the sex-specific models by less than six chi-square points. The observed power for the differences observed between the male and female family solutions was .39. As pointed out by Eaves et al. (1997), only studies with very large samples can reject the fully constrained model in favor of the sex-specific models. In the present case as well as in the case of several studies in the literature, the fully constrained model does provide a good fit to the data and thus provides the most parsimonious summary of the data. Given that we cannot rule out the possibility of sex differences in the relative magnitudes of genetic and environmental influences on delinquency, we follow the lead of other investigators (e.g., Eaves et al., 1997; Silberg et al., 1994; Slutske et al., 1997) in presenting the sex-specific results in addition to the combined sex results.

The major differences evident in the sex-specific model-fitting results presented in Table 4 concern the magnitude of cultural transmission and shared environmental influences. As expected, shared environmental influence estimates were of greater magnitude among females than among males. The data also suggested a greater role for cultural transmission of delinquency among families of female (as compared to male) twins. However, in light of the good fit of the fully constrained models, it is important not to over-emphasize the results from the sex-specific models. The model that best summarized the data was a fully constrained model that indicated substantial environmental effects on adolescent delinquency among boys and girls.

Discussion

In the present study we used a twin family design to examine the relative contributions of additive genetic, shared, and nonshared environmental influences to self-reported adolescent delinquency and to investigate the vertical cultural transmission of delinquency from parents to offspring. To this end, we fit the social homogamy model to self-reported delinquency data from a large sample of families containing male or female adolescent twins. These data also allowed us to test for

¹ The assortative mating parameter, μ , was bound between 0 and 1.0. In several of the models, μ hit its upper boundary of 1.0. The μ parameter estimated from the model is not interpreted directly. That is, an estimate of 1.0 for μ does not indicate perfect assortative mating for the trait but instead indicates the strength of the correlation between the factors assumed to account for assortative mating under the model. Thus, when μ is 1.0, it indicates that spouses are perfectly correlated for social status, geographic location, and cultural background factors under the social homogamy model.

sex differences in the etiology and transmission of delinquency. Based in part on our previous work, we expected shared environmental factors to have a more substantial influence on delinquency among girls than boys. Based on recent developmental theories on the etiology of adolescent delinquency, we expected large environmental (relative to genetic) influence estimates.

Our expectation regarding sex differences in the relative magnitude of genetic and environmental influences on delinquency was not supported by the data. The fully constrained model provided a good fit to the data and could not be rejected in favor of the sex-specific models. The model that provided the best summary of the data indicated that mild to moderate types of delinquent behavior among older adolescent boys and girls was largely dependent on environmental effects while less than 20% of the variance in adolescent delinquency was dependent on additive genetic factors. These results are consistent with those reported by Eaves et al. (1997) for self-reported conduct problems. These results are also consistent with our expectations stemming from developmental theories of delinquency. Both DiLalla and Gottesman (1989) and Moffitt (1993) suggest that adolescent delinquency is largely the product of environmental factors. Although not suited to identifying specific environmental influences on delinquency, our data fit best to a model that estimated the effect of nonshared environmental influences at 46–68%. This is compatible with Moffitt's (1993) contention that delinquency arising in adolescence stems largely from peer pressures: factors that likely have unique effects on siblings.

The model-fitting analyses indicated that adolescent delinquency is, to some extent, transmitted from parents to offspring through characteristics associated with culture, geographic location, or SES. However, the magnitude of the effects of social background factors is much less than the magnitude of the effects of other shared environmental factors (as can be seen by the modest values of the t parameter as compared to the r parameter in Tables 3 and 4). This is consistent with Loeber and Stouthamer-Loeber's (1987) review of predictors of delinquency that showed that family "contextual" variables such as SES were not strong predictors of adolescent delinquency. Our data are consistent with the notion that individual differences in delinquency are associated with environmental factors shared by children in a family (e.g., parenting style), but not between parents and their children (e.g., SES). Certain parenting practices are associated with antisocial behavior in children (e.g., Patterson, 1986) and may account for some of the shared environmental effect we found. Although not trivial, our data suggest that shared environmental variation accounts for only a quarter of the variance in delinquency among adolescents; most of the variance in delinquency is associated with nonshared environmental factors.

The spousal correlations for delinquency were modest and positive, suggesting some degree of assortative mating based (we assume) on social background, geographic, and cultural characteristics. However, the overall best-fitting model (the fully constrained ACE model) indicated that the spousal correlation for shared environmental factors (μ) was perfect (1.0). This is not really a plausible solution and it suggests that perhaps alternative models should be considered. For example, it is possible that the modest spousal correlations reflect an association between general antisocial tendencies (such as risk-taking or affiliation with antisocial peers) versus

delinquency per se, and this interpretation has some support in the literature (Krueger, Moffitt, Caspi, Bleske, & Silva, 1998). The phenotypic assortment model (which assumes that assortative mating results from the tendency for individuals to choose mates who are phenotypically similar to themselves on a given trait; Cavalli-Sforza & Feldman, 1973; Cavalli-Sforza, Feldman, Chen, & Dornbusch, 1982) may be a useful alternative model for delinquency data. It is even plausible to consider the possibility that assortative mating may arise from a resemblance on nonshared environmental variables (e.g., similar peer group) induced by similarity on phenotypic traits (e.g., antisocial behavior). Ideally, one could fit both the social homogamy and the phenotypic assortment models and compare their performance, however, such a study is best suited for large data sets. The MTFS is an overlapping cohort longitudinal study and, as such, an empirical test between family models may be possible in the future when the sample of families with 17-year-old twins will more than double.

This study begins to fill in the holes in the literature created by the paucity of self-report data from older adolescent samples while also providing new information on the role of assortative mating and cultural transmission effects on delinquency. A notable limitation of this study was the use of a delinquency measure that did not afford us an opportunity to classify our participants into early-onset, persistent antisocial offenders vs. late-onset, transitory offenders as outlined in a recent developmental theory of delinquency (Moffitt, 1993). At a base rate of around 5% (Moffitt, 1993), we could have expected only 48 twins to meet "criteria" for early-onset, persistent antisocial behavior. As such, we are not overly concerned that these cases, if included in our sample, had a significant effect on our results. However, once the sample of families with 17-year-old twins participating in the MTFS is increased, it may be feasible to identify early-onset, persistent cases and either remove them from studies of adolescent delinquency or perhaps study them separately.

This investigation highlighted the association of environmental factors with individual differences in delinquency among adolescent boys and girls. Further work is needed to identify specific environmental factors related to adolescent antisocial behavior. Developmental theories of delinquency suggest that these factors may include substance use/abuse in adolescents and in the family; antisocial tendencies in family members; parenting styles; and peer influences. More work is also needed to determine whether similar genetic and environmental factors contribute to behaviors that commonly covary with delinquency (e.g., substance use; personality factors) in order to improve our understanding of delinquency in a developmental context.

Acknowledgements—This article was supported in part by NIH grants #AA00175, AA09367, and DA 05147 and from NIMH Training Grant MH17069.

References

- Akaike, H. (1987). Factor analysis and the AIC. *Psychometrika*, 52, 317–332.
- Cavalli-Sforza, L. L., & Feldman, M. (1973). Cultural versus biological inheritance: Phenotypic transmission from parents to child. *American Journal of Human Genetics*, 25, 618–637.

- Cavalli-Sforza, L. L., Feldman, M. W., Chen, K. H., & Dornbusch, S. M. (1982). Theory and observation in cultural transmission. *Science*, *218*, 19–27.
- DiLalla, L. F., & Gottesman, I. I. (1989). Heterogeneity of causes of delinquency and criminality: Lifespan perspectives. *Development and Psychopathology*, *1*, 339–349.
- Eaves, L. J., Last, K. A., Young, P. A., & Martin, N. G. (1978). Model-fitting approaches to the analysis of human behavior. *Heredity*, *41*, 249–320.
- Eaves, L. J., Silberg, J. L., Meyer, J. M., Maes, H. H., Simonoff, E., Pickles, A., Rutter, M., Neale, M. C., Reynolds, C. A., Erikson, M. T., Heath, A. C., Loeber, R., Truett, K. R., & Hewitt, J. K. (1997). Genetics and developmental psychopathology: 2. The main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. *Journal of Child Psychology and Psychiatry*, *38*, 965–980.
- Edelbrock, C., Rende, R., Plomin, R., & Thompson, L. A. (1995). A twin study of competence and problem behavior in childhood and early adolescence. *Journal of Child Psychology and Psychiatry*, *36*, 775–785.
- Eley, T. C. (1997). General genes: A new theme in developmental psychopathology. *Current Directions in Psychological Science*, *6*, 90–95.
- Eley, T. C., Lichtenstein, P., & Stevenson, J. (1999). Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: Results from two twin studies. *Child Development*, *70*, 155–168.
- Gibson, H. B. (1967). Self-reported delinquency among schoolboys, and their attitudes to the police. *British Journal of Social and Clinical Psychology*, *6*, 168–173.
- Grove, W. M., Eckert, E. D., Heston, L., Bouchard, T. J., Jr, Segal, N., & Lykken, D. T. (1990). Heritability of substance abuse and antisocial behavior: A study of monozygotic twins reared apart. *Biological Psychiatry*, *27*, 1293–1304.
- Heath, A. C., Kendler, K. S., Eaves, L. J., & Markell, D. (1985). The resolution of cultural and biological inheritance: Informativeness of different relationships. *Behavior Genetics*, *15*, 439–465.
- Krueger, R. F., Moffitt, T. E., Caspi, A., Bleske, A., & Silva, P. A. (1998). Assortative mating for antisocial behavior: Developmental and methodological implications. *Behavior Genetics*, *28*, 173–186.
- Loeber, R., & Stouthamer-Loeber, M. (1987). Prediction. In H. C. Quay (Ed.), *Handbook of juvenile delinquency* (pp. 325–382). New York: Wiley.
- Lyons, M. J., True, W. R., Eisen, S. A., Goldberg, J., Meyer, J. M., Faraone, S. V., Eaves, L. J., & Tsuang, M. T. (1995). Differential heritability of adult and juvenile antisocial traits. *Archives of General Psychiatry*, *52*, 906–915.
- Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, *100*, 674–701.
- Neale, M. C. (1997). *Mx: Statistical modeling* (3rd ed.). Box 710 MCV, Richmond, VA 23298: Department of Psychiatry.
- Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, The Netherlands: Kluwer Academic.
- Patterson, G. R. (1986). Performance models for antisocial boys. *American Psychologist*, *41*, 432–444.
- Rao, D. C., Morton, N. E., & Yee, S. (1976). Resolution of cultural and biological inheritance by path analysis. *American Journal of Human Genetics*, *28*, 228–242.
- Rowe, D. C. (1983). Biometrical genetic models of self-reported delinquent behavior: A twin study. *Behavior Genetics*, *13*, 473–489.
- Rowe, D. C. (1986). Genetic and environmental components of antisocial behavior: A study of 265 twin pairs. *Criminology*, *24*, 513–532.
- Silberg, J. L., Erickson, M. T., Meyer, J. M., Eaves, L. J., Rutter, M. L., & Hewitt, J. K. (1994). The application of structural equation modeling to maternal ratings of twins' behavioral and emotional problems. *Journal of Consulting and Clinical Psychology*, *62*, 510–521.
- Silberg, J., Rutter, M., Meyer, J., Maes, H., Hewitt, J., Simonoff, E., Pickles, A., Loeber, R., & Eaves, L. (1996). Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *Journal of Child Psychology and Psychiatry*, *37*, 803–816.
- Slutske, W. S., Heath, A. C., Dinwiddie, S. H., Madden, P. A. F., Bucholz, K. K., Dunne, M. P., Statham, D. J., & Martin, N. G. (1997). Modeling genetic and environmental influences in the etiology of conduct disorder: A study of 2682 adult twin pairs. *Journal of Abnormal Psychology*, *106*, 266–279.
- Taylor, J., McGue, M., Iacono, W. G., & Lykken, D. T. (in press). A behavioral genetic analysis of the relationship between the Socialization scale and self-reported delinquency. *Journal of Personality*.