

# Cohort differences in genetic and environmental influences on retrospective reports of conduct disorder among adult male twins

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## ABSTRACT

**Background.** Rates of child and adolescent conduct disorder (CD) have increased steadily over the past several decades. What is not known is whether the underlying genetic and environmental influences on individual differences in CD have also changed.

**Methods.** Retrospective reports of antisocial behaviour prior to age 18 were obtained from a population-based sample of 2769 adult males from male–male twin pairs born between 1940 and 1974. Using a summary score of number of CD symptoms, structural equation modelling was used to investigate whether mean level and variation in CD increased with more recent cohorts, and whether any increase in variance could be explained by familial or non-familial factors.

**Results.** Both mean level CD symptoms and variation were increased in more recent cohorts. Model fitting indicated that the primary increase in variance was due to familial factors, most notably, an increase in the shared environmental influences on CD, from 0.01 (95% CI = 0.00; 0.27) to 0.30 (95% CI = 0.01; 0.44). Heritability estimates remained largely unchanged, although an increase in genetic factors could not be ruled out.

**Conclusions.** Secular changes in sociodemographic factors responsible for increasing rates of CD may also account for the greater magnitude of shared environmental influences on variation in CD found among more recent cohorts.

## INTRODUCTION

Although twin and adoption studies have provided substantial evidence that genetic factors play a role in the aetiology of adult criminality (Crowe, 1974; Mednick *et al.* 1984; Cloninger & Gottesman, 1987; Mason & Frick, 1994; also see DiLalla & Gottesman, 1989; Miles & Carey, 1997, for reviews) and antisocial personality disorder (Cadoret, 1978), there is still considerable debate over the role that genetic factors play in the aetiology of childhood and adolescent conduct disorder (CD). For example, a number of studies have found evidence for significant

genetic influences on child and adolescent antisocial behaviour (Rowe, 1983, 1986; Graham & Stevenson, 1985; Ghodsian-Carpey & Baker, 1987; Silberg *et al.* 1996; Gjone & Stevenson, 1997; Slutske *et al.* 1997). However, other studies have failed to find genetic effects, concluding that shared environmental factors are largely responsible for sibling resemblance for childhood antisocial behaviour (Lyons *et al.* 1995; Thapar & McGuffin, 1996). Still other studies of adolescents find that both genetic and shared environmental factors contribute to variation in antisocial behaviour (Edelbrock *et al.* 1995; Simonoff *et al.* 1995; Eley *et al.* 1999).

There are a number of potential reasons for these inconsistent results. Different definitions and measures of CD and/or varied reporters

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may yield differences in the estimates of relative genetic and environmental influence, although no clear patterns have emerged (Simonoff *et al.* 1995; Eaves *et al.* 1997, for a comparison of heritability estimates using different raters). There is also considerable variation in samples. Studies that rely on clinical samples of children may not be representative of the general population, as it is unclear whether the factors that contribute to clinically-defined CD are similar to those that contribute to a broader range of antisocial behaviour. Similarly, studies that rely on volunteer samples may also be biased, as individuals with severe psychopathology are not likely to be included in such samples.

Investigations of the genetic and environmental influences on CD have also spanned a wide range of birth cohorts. Many of the studies of adolescent delinquency have relied on self-reports or parent reports from cohorts of adolescents born in the 1970s and 1980s (Edelbrock *et al.* 1995; Eley *et al.* 1999), whereas both Slutske *et al.* (1997) and Lyons *et al.* (1995) relied on retrospective ratings from adults born prior to 1964. Causes of individual differences in CD may have changed over past decades, accounting for some of the discrepancy across studies.

On the one hand, antisocial behaviour among juveniles has increased substantially over the past 50 years. For example, arrest rates among US juveniles aged less than 18 years increased by nearly 50% between the years 1970 and 1995 (Bureau of Justice Statistics, 1999), and similar increases in the prevalence of CD have also occurred (Robins, 1998). Other countries, such as Australia, England and Wales, also report increases in criminal behaviour and rates of CD (Biles, 1983; Robins, 1998). Given that these dramatic changes have occurred over relatively short periods of time, it is unlikely that genetic factors can explain the increased prevalence of CD. Environmental factors such as changes in family composition and income are commonly used to explain the increase in prevalence. Because these factors are shared within members of the same family, but vary between families, these factors are considered shared environmental influences. Thus, it is possible that shared environmental factors are more important in the aetiology of CD among younger cohorts.

On the other hand, studies of other phenotypes

have suggested that the heritability of certain behaviours has increased with more recent cohorts. Kaprio *et al.* (1991) reported higher heritability estimates for alcohol use among twins born between 1951 and 1957 than among those born 1932–1950, especially among male twins. A twin study examining cohort changes in rates of regular tobacco use found that, among females, as the prevalence of smoking increased among more recent cohorts, the heritability of smoking also increased (Kendler *et al.* 2000). Finally, Dunne *et al.* (1997) reported that the heritability of age of onset of sexual intercourse increased for both males and females with more recent cohorts. The authors of these latter two studies speculated that as social controls for smoking and adolescent sexual behaviour decreased, genetic influences on behaviour were more likely to be expressed. Thus, it is possible that higher rates of CD in more recent cohorts reflect declining social constraints on such behaviour, which would lead to a simultaneous increase in the heritability of CD.

It is also possible that rates of CD could change across birth cohort, while the genetic and environmental architecture underlying individual differences in CD remain unchanged. This situation could arise in at least two ways. First, the prevalence of CD might increase, but the variation in CD might remain constant. Alternatively, variation in CD might increase due in equal part to changes in genetic and environmental influences. Thus, the proportion of variation due to genetic and shared and non-shared environmental factors might remain constant, and would lead to similar estimates of heritability and shared environmental influences.

To date, only a handful of studies have tested for cohort differences in the genetic and environmental influences on CD. Slutske *et al.* (1997) reported that birth cohort explained approximately 1% of the variation in CD but that genetic and environmental parameters did not vary by cohort. However, this study used a volunteer sample of Australian twins born between 1919 and 1964, so the results may not generalize to a younger population-based sample of American twins. In a small study of conduct problems among American twins, Waldman *et al.* (1989) reported that although monozygotic (MZ) correlations were similar across cohort, dizygotic (DZ) twin correlations were higher in

more recent cohorts, suggesting an increase in shared environmental influences.

Thus, the purpose of the present study is to use a large, population-based sample of US male twins born 1940–1974 to investigate three questions. First, has the prevalence of CD increased in more recent birth cohorts? Secondly, has the increase in prevalence of CD been accompanied by a simultaneous increase in variation? Finally, if the variation in CD has changed, have the underlying genetic and environmental influences on variation in CD changed as well?

## METHOD

### Sample and procedure

Twins were part of the Virginia Stress and Coping Project, a longitudinal study of the genetic and environmental risk factors for common psychiatric and substance use disorders among male and female twins (see Kendler & Prescott, 1999, for details on subject ascertainment and collection of Wave 1 interviews). At least 1 year after the completion of the first-wave interview, which was performed, in most instances, by telephone, we contacted the twins again and attempted to schedule a second-wave interview. The number of subjects eligible for wave 2 interviews included 6814 individuals with complete wave 1 interviews as well as three subjects at wave 2 who were eligible at wave 1 but from whom completed wave 1 interviews were not obtained. Of the 6817 eligible individuals for the wave 2 interview, 5629 (82.6%) were successfully interviewed while 852 (12.5%) refused, 22 (0.3%) had incomplete interviews, 237 (3.5%) did not agree within the study time limit, 51 (0.7%) could not be located, and 26 (0.4%) were deceased or too ill to be interviewed. Where possible, the interview was completed face to face (79.4% of sample). In addition to the wave 2 personal interview, twins were also asked to fill out a self-report questionnaire (SRQ). In most instances, the SRQ was filled out at the end of the personal interview. However, in some cases, it was returned later by mail. We received 5338 SRQs (94.8%). The wave 2 interviews took place between 1994 and 1998.

The present analyses are restricted to 2769

male twins from male–male twin pairs from whom we had complete SRQ data. The sample consisted of 1065 complete twin pairs (634 MZ pairs, 431 DZ pairs), and nine male–male twin pairs formed from three triplet families (7 MZ pairs, 2 DZ pairs), for a total of 1074 twin pairs. In addition, we had data from 331 twins whose co-twins had participated at wave 1, but not at wave 2 (167 MZ, 164 DZ) and 299 twins whose co-twins had never participated in the study (136 MZ twins, 163 DZ twins). These 630 unpaired twins were used to test whether our results might be biased by non-participation. The average age of the twins at the wave 2 interview was 37.1 (s.d. = 9.2; range = 20–58 years). Twins had a mean of 13.4 years of education (s.d. = 2.6). This project was approved by our local Institutional Review Board. Subjects were informed about the goals of the study and provided verbal consent prior to telephone interviews and written informed consent prior to face to face interviews and collection of DNA samples.

### Measures

#### *Twin zygosity*

We began assessing zygosity by genotyping 227 male twin pairs with eight or more highly polymorphic DNA markers. Using these pairs, we developed a Fisher's discriminant function with PROC DISCRIM in SAS Version 6.12 (SAS, 1997), using self-reported height and weight, the twins' history of any blood tests and six standard self-report questions concerning similarity and confusability of appearance, and self-report of zygosity. The error rate for the reclassification of the DNA-typed twins was 6.6%. Using this discriminant function, we could confidently assign zygosity to all but 97 of the remaining twin pairs (zygosity was established via an estimated probability of monozygosity of  $\leq 10\%$  or  $\geq 90\%$ ). Of these 97 pairs, we had usable DNA from both members of 65 pairs from which we obtained zygosity. The remaining 32 cases were reviewed by our two raters (K.S.K. and C.A.P.) who assigned zygosity based on self-report measures and, in most cases, photographs.

Assignment of zygosity for twins without an interviewed co-twin was done using a discriminant function analysis of items regarding twin similarity and confusability of appearance and twin self-report of zygosity, with DNA-

typed twins as the comparison group. Singleton twins were classified as MZ if the probability of being MZ was greater than 0.5, and as DZ if the probability of being MZ was less than or equal to 0.5. Over 86% of the 512 singleton male twins had a probability of MZ of  $\leq 10\%$  or  $\geq 90\%$ . Only 13 males (2.5%) fell in the range of probability of MZ between 0.40 and 0.60.

#### *Birth cohort*

Birth cohort was coded as 1 = 1940 to 35 = 1974 and was used as a continuous variable in the model fitting analyses. For descriptive purposes, we also divided the sample into approximate thirds based on year of birth: 1940–1954; 1955–1964; and 1965–1974.

#### *Conduct disorder*

In the SRQ twins were asked to report how often they had engaged in 11 specific antisocial behaviours (e.g. 'played hooky from school', 'physically hurt other people', and 'used a weapon in a fight'). These items correspond closely to 11 of the 13 symptoms used to establish conduct disorder (CD) in the DSM-III-R (APA, 1987).<sup>1</sup> Responses ranged from 0 = 'never' to 3 = '6 or more times'. Because the DSM-III-R considers CD prior to age 15 to be a requirement for the diagnosis of antisocial personality disorder (ASPD), nine of the 11 items were asked twice: once for the period prior to age 15, and once for the period age 15 to 17. The remaining two items referred to the frequency with which the respondent, prior to age 15, (1) started physical fights; and, (2) lied (with 0 = 'never' and 3 = 'often').

To create a summary score of symptoms, each item was summed across the two time points (with the exception of the two items that were only asked about behaviour prior to age 15) and then a computer algorithm was applied to the summed score to create a criterion for whether that particular symptom was present (1) or absent (0). These criteria corresponded closely

to the DSM-III-R. Symptoms were then summed to create a continuous measure. Summary scores of symptoms are similar to the variety or breadth scores typically used in studies of delinquency and antisocial behaviour, and have the advantage over frequency-based measures in that summary scores take into account the severity of each item in assigning a cut-off criterion. Because the distribution of scores for this variable was positively skewed, a square-root transformation was employed, reducing the amount of skewness.

Missing values were assigned if respondents had 50% or more missing items (two cases). Among the 2148 twins from complete twin pairs used in the structural equation modelling analyses, 12 twins (0.5%) had missing data on 1 or 2 items. Among the 630 singleton twins, four twins (0.7%) had missing data on 1 or 2 items. No twins included in the analyses had more than two missing items.

#### *Equal environments variables*

Two variables were created to test whether our results could be biased by similarity of childhood environment and/or by amount of contact. The first variable consisted of four items: 'how often did you and your twin share the same room,' 'how often did you and your twin have the same playmates,' 'how often did you dress alike,' and 'how often were you in the same classes at school'. These items were recorded so that 0 = 'never' and 3 = 'always', and were then summed to form a scale that ranged from 0–12, with higher values representing more similar childhood environments. The second variable was a single item that asked how often the respondent had contact with his twin during the year prior to the interview, with responses ranging from 0 = 'never' to 5 = 'more than once a week'. Scores on these two variables were averaged across twins to create a single score for each twin pair ( $r_{\text{twin}} = 0.77$ , for contact;  $r_{\text{twin}} = 0.55$ , for similarity of childhood environment).

#### **Data analysis**

The structural equation modelling program, *Mx* (Neale, 1999) was used to test for cohort differences in means and variances. For all analyses, models were fit to raw data. In a standard univariate behavioural genetic analysis of a continuously-distributed variable, variation in a measured phenotype (V) can be decomposed

The items in the SRQ differ from the DSM-III-R as follows: (1) the item regarding forced sex was eliminated, as it very infrequently endorsed in other samples and can be offensive to some respondents; (2) two of the DSM-III-R items ('has broken into someone else's house, building, or car' and 'has stolen without confrontation') were combined into a single item, namely 'stole things from a store or from someone I knew'.

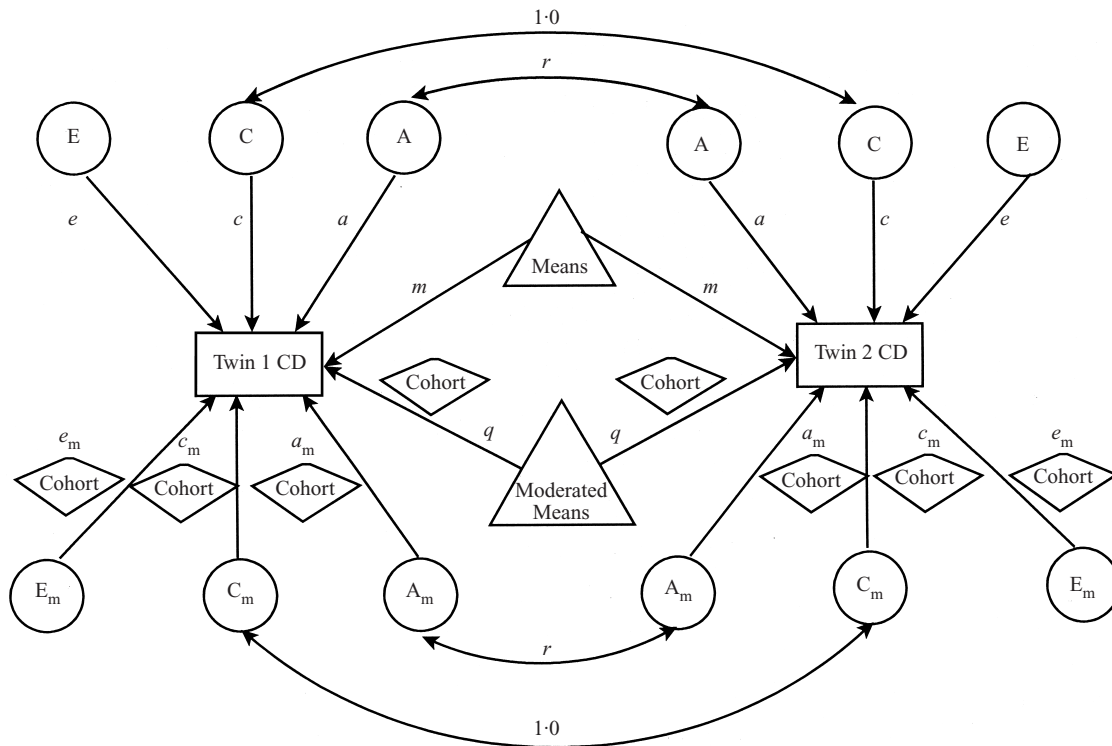


FIG. 1. The Moderated-Means, Moderated-Covariance Model. CD, Conduct disorder; A, latent genetic influences; C, latent shared environmental influences; E, latent non-shared environmental influences, represented by the parameters *a*, *c* and *e*, respectively. These parameters are unaffected by cohort. A<sub>m</sub>, moderated genetic influences; C<sub>m</sub>, moderated shared environmental influences; E<sub>m</sub>, moderated non-shared environmental influences, represented by the parameters *a<sub>m</sub>*, *c<sub>m</sub>* and *e<sub>m</sub>* (all latent variables have unit variance); *m*, means, unaffected by cohort; *q*, moderated means; *r*, coefficient of genetic relatedness (*r* = 1.0, for MZ twins; *r* = 0.5, for DZ twins).

into additive genetic (*a*), shared environmental (*c*) and non-shared environmental (*e*) influences (i.e.  $V = a^2 + c^2 + e^2$ ), and means (M) can also be estimated. To test whether means varied across cohort, an additional parameter (*q*) was estimated that was multiplied by birth cohort and added to the means estimated in the standard model: Moderated Means = M + *q* \* cohort. If means do not vary across cohort, then *q* would not be significantly different from zero, and the moderated-means model would not fit significantly better than the standard model.

To test whether the variance of CD changed across cohort, another parameter (*s*) was estimated that was also multiplied by birth cohort and added to the *a*, *c* and *e* parameters estimated in the standard model: Moderated V = ( $a^2 + c^2 + e^2$ ) + *s* \* cohort \* ( $a^2 + c^2 + e^2$ ). If the moderated-variance model fit significantly better than the standard model, this indicates that

variance in CD is significantly changing across cohort.

Because the moderated-variance model assumes that *a*, *c* and *e* are all varying proportionately, it does not test whether patterns of MZ/DZ twin covariance change across birth cohorts. To conduct this test, we compared the fit of the moderated-variance model with a model that dropped the *s* parameter and instead added three additional parameters that corresponded to cohort differences in genetic (*a<sub>m</sub>*), shared environmental (*c<sub>m</sub>*) and non-shared environmental (*e<sub>m</sub>*) influences (the moderated-covariance model). Each of these parameters was multiplied by birth cohort. Thus, the equation for variation in CD was  $V = (a^2 + c^2 + e^2) + \text{cohort} * (a_m^2 + c_m^2 + e_m^2)$ . Fig. 1 shows the full moderated-covariance model.

Depending upon the relative strength of the *a<sub>m</sub>*, *c<sub>m</sub>* and *e<sub>m</sub>* parameters, patterns of co-

variation among twins will also vary across cohort. If the underlying source of increasing variance is non-shared environment ( $e_m$ ), then twin correlations for both MZ and DZ twins would decrease across cohort, as would estimates of both heritability and shared environmental influences. In contrast, if increasing variation in CD is largely due to  $a_m$ , then correlations for MZ twins should increase more than correlations for DZ twins, and heritability estimates would also increase. Finally, if increasing variation in CD is largely due to  $c_m$ , the absolute increase in correlations should be similar in MZ and DZ twins, and estimates of shared environmental influences would also increase. The significance of the  $a_m$ ,  $c_m$  and  $e_m$  parameters can be tested by comparing the fit of the full model to reduced models that drop one or more parameters (Neale & Cardon, 1992). Two criteria to assess model fit are used here: the likelihood-ratio chi-square (LRC) statistic and Akaike's Information Criterion (AIC; Akaike, 1987; Williams & Holahan, 1994). If the LRC between two models is non-significant, then the model with fewer parameters is generally accepted as the more parsimonious model. The AIC is an index of both goodness-of-fit and parsimony; the more negative the AIC, the better the balance between goodness-of-fit and parsimony.

Finally, if patterns of twin resemblance do change across cohort, it is possible that this result might occur from violations of the equal environments assumption, or from differences in amount of adult contact among twins in older and younger cohorts. To test these possibilities, we added to the moderated-covariance model three additional parameters that allowed for the genetic, shared environmental, and non-shared environmental influences to vary depending upon level of adult contact or by similarity of childhood environment.

## RESULTS

### Reliability analyses

To assess the test-retest reliability of our assessments, 131 members of male-male twin pairs filled out a second SRQ an average ( $\pm$ S.D.) of 4.4 ( $\pm$ 1.1) months after their initial interview. The summary score showed adequate reliability, as indexed by the intraclass correlation,  $r = 0.70$  ( $P < 0.001$ ). Test-retest reliability was not

linearly associated with birth year ( $b = 0.0044$ , S.E. = 0.005,  $P = 0.35$ ).

### Descriptive statistics

Table 1 presents the mean summary score and standard deviations, separately by birth cohort and zygosity. Among both MZ and DZ twins, the mean number of CD symptoms systematically increased across birth cohort, and there was also a slight increase in variance. Regression analyses using PROC GENMOD (SAS, 1997) to control for correlated observations within families (results not shown) indicated that the linear effect of birth cohort was significant for both the raw and the square-root transformed variables ( $P_s < 0.001$ ). Adding non-linear effects of birth cohort did not significantly improve fit (LRC = 4.98,  $df = 32$ ,  $P = 0.99$ , for the raw variable; LRC = 12.64,  $df = 32$ ,  $P = 0.99$ , for the square-root transformed variable).

Table 1 also presents the twin correlations for CD, separately by zygosity and birth cohort. Both the MZ and DZ twin correlations increased across cohort, with the increase being greater among DZ twins. Although the MZ twin correlation was greater than the DZ twin correlation for twins born 1940-1954 and 1950-1964, the MZ and DZ twin correlations were similar among twins born 1965-1974.

### Model fitting

Prior to estimating cohort effects on means and variation in CD, we first ran two models to test whether the mean number of CD symptoms could be equated across twin 1 and twin 2, and across MZ twins and DZ twins. Neither model was significantly different from the full model (LRC = 0.33,  $df = 2$ ,  $P = 0.84$ ; LRC = 0.04,  $df = 1$ ,  $P = 0.84$ , respectively); thus means were constrained to be equal in all subsequent models.

The  $-2$  log-likelihood ( $-2$  LL) function from the model constraining both means and variance to be equal across cohort was 4734.45 ( $df = 2144$ ). Adding the  $q$  parameter to allow for cohort differences in means (the moderated-means model) increased the fit of the model to 4722.11 ( $df = 2143$ ), a significant change (LRC = 12.34,  $df = 1$ ,  $P < 0.001$ ), signalling significant cohort effects for mean number of CD symptoms. The estimate of  $q$  was positive, indicating that as expected, mean number of CD symptoms increased with more recent birth

Table 1. Descriptive statistics and twin correlations for conduct disorder, by birth cohort and zygosity

Mean number of CD symptoms*	Birth cohort					
	1940–1954		1955–1964		1965–1974	
	<i>N</i>	Mean (s.d.)	<i>N</i>	Mean (s.d.)	<i>N</i>	Mean (s.d.)
MZ	394	0.70 (0.71)	428	0.84 (0.77)	460	0.88 (0.75)
DZ	336	0.70 (0.69)	284	0.85 (0.74)	246	0.89 (0.81)

Twin correlations†	1940–1954		1955–1964		1965–1974	
	<i>N</i>	<i>r</i> (95% CI)	<i>N</i>	<i>r</i> (95% CI)	<i>N</i>	<i>r</i> (95% CI)
MZ	197	0.24 (0.15–0.33)	214	0.29 (0.20–0.38)	230	0.40 (0.32–0.48)
DZ	168	0.15 (0.05–0.26)	142	0.18 (0.07–0.29)	123	0.41 (0.30–0.51)

CD, Conduct disorder; MZ, monozygous; DZ, dizygous.

\* Raw number of CD symptoms.

† Correlations based on square-root transformed CD symptoms.

Table 2. Model-fitting results

Model	Model	Model fit statistics				Unstandardized parameter estimates						
		–2 LL	df	LRC	df	AIC	<i>a</i>	<i>c</i>	<i>e</i>	<i>a<sub>m</sub></i>	<i>c<sub>m</sub></i>	<i>e<sub>m</sub></i>
1	<i>ace + a<sub>m</sub>c<sub>m</sub>e<sub>m</sub></i>	4709.22	2140	—	—	–18.80	0.2375	0.0000	0.6164	0.0175	0.0735	0.0000
2	<i>ace + a<sub>m</sub>c<sub>m</sub></i>	4709.22	2141	0.00	1	–20.80	–0.2375	0.0000	0.6164	–0.0175	0.0735	—
3	<i>ace + c<sub>m</sub>e<sub>m</sub></i>	4709.23	2141	0.01	1	–20.78	0.2403	0.0000	0.6169	—	0.0749	0.0000
4	<i>ace + a<sub>m</sub>e<sub>m</sub></i>	4710.98	2141	1.76	1	–19.04	0.0000	0.2550	0.6114	–0.0746	—	0.0000
5	<i>ace + a<sub>m</sub></i>	4710.98	2142	1.76	2	–21.04	0.0000	0.2550	–0.6114	0.0746	—	—
6	<i>ace + c<sub>m</sub></i>	4709.23	2142	0.01	2	–22.79	0.2403	0.0000	0.6169	—	0.0749	—
7	<i>ace + e<sub>m</sub></i>	4720.28	2142	11.06**	2	–11.73	0.2845	0.2985	–0.5767	—	—	0.0485

–2 LL, –2 log-likelihood function; LRC, likelihood-ratio chi-square; df, degrees of freedom; AIC, Akaike’s Information Criterion; *a*, genetic influences; *c*, shared environmental influences; *e*, non-shared environmental influences; *a<sub>m</sub>*, moderated genetic influences; *c<sub>m</sub>*, moderated shared environmental influences; *e<sub>m</sub>*, moderated non-shared environmental influences.

cohorts. Adding the *s* parameter for cohort effects on the variance of CD (the moderated-variance model) further improved the model fit to 4717.08 (df = 2142). This increase in fit was also significant (LRC = 5.06, df = 1, *P* < 0.02), and the *s* parameter was estimated at +0.10 (95% CI = 0.03–0.14), representing increasing variance in CD with more recent cohorts.

The moderated-variance model assumed that the increase in variation with more recent cohorts was due to proportional increases in *a*, *c* and *e*, and therefore that patterns of covariance among twins were unaffected by cohort. To test this assumption, we compared the fit of the moderated-variance model to the fit of a model that allowed for separate cohort effects on the additive genetic, shared environmental, and non-shared environmental influences (*a<sub>m</sub>*, *c<sub>m</sub>* and *e<sub>m</sub>*). The moderated-covariance model fit the data significantly better than the moderated-variance model (LRC = 7.86, df = 2, *P* < 0.02),

indicating that patterns of twin covariance also systematically changed across cohort.

Table 2 presents the results from this model (Model 1). The effect of cohort on non-shared environmental influences was estimated at 0.00, suggesting that the increase in variation across cohort is due to familial factors. Furthermore, the majority of the increase in variation across cohort appeared to be due to increases in shared environmental influences. To test this formally, we ran a final series of nested models estimating the significance of the *a<sub>m</sub>*, *c<sub>m</sub>* and *e<sub>m</sub>* parameters. The results from this series of model fitting are also shown in Table 2.

By the LRC criteria, Model 7, the model that assumed that non-shared environmental influences were the sole source of the increase in variance associated with increasing cohorts, was the only model that fit the data more poorly than the full model (Model 1). By comparison, neither the model that had genetic factors

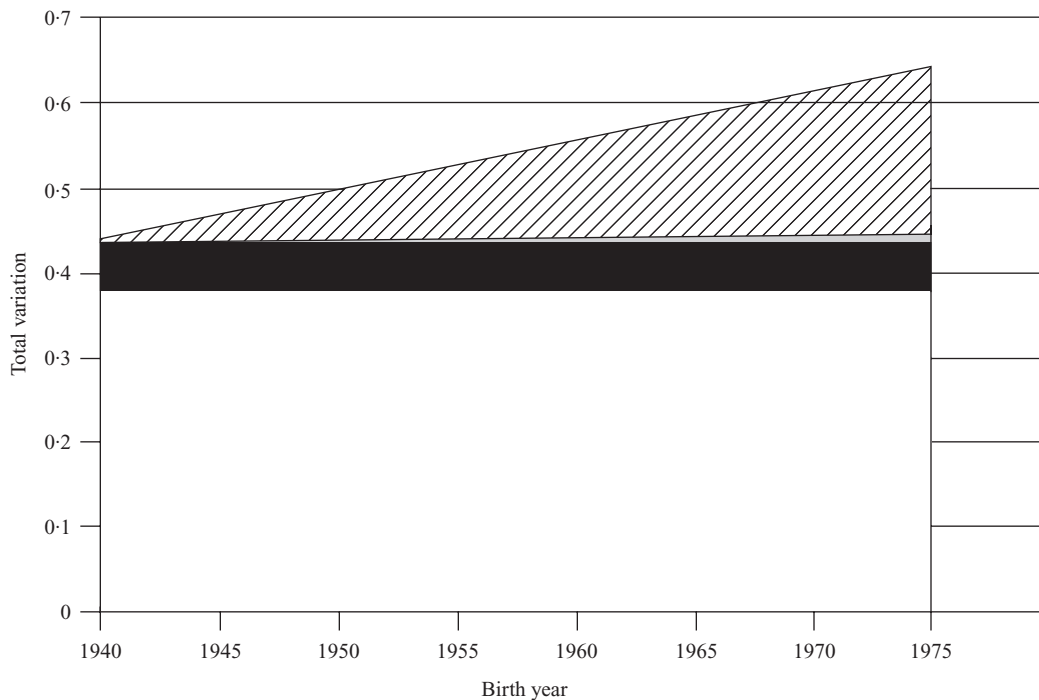


FIG. 2. Sources of variation in conduct disorder across birth cohort.  $\square$ ,  $C_m$ , moderated shared environmental influences;  $\square$ ,  $A_m$ , moderated genetic influences;  $\blacksquare$ ,  $A$ , genetic influences, unaffected by cohort;  $\square$ ,  $E$ , non-shared environmental influences, unaffected by cohort.

specified as the sole source of the increase in variance (Model 5) nor the model that had shared environmental influences specified as the sole source of the increase in variance (Model 6) fit the data more poorly than Model 1. Thus, although the increase in variance associated with cohort appears to be due to familial factors, we could not distinguish between an increase in genetic factors and an increase in shared environmental factors. However, by AIC criteria, Model 6 was the best-fitting model. Nevertheless, because we could not statistically differentiate between Models 5 and 6, we used the parameter estimates from the full model to calculate the changes in the underlying sources of variance due to cohort. Fig. 2 depicts how the genetic and environmental sources of variation have changed across cohorts.

Because the  $e_m$  parameter was estimated at 0 in the full model, non-shared environmental influences ( $e$ ) on variation in CD remained constant across cohort. In addition, non-shared environmental influences accounted for the single largest proportion of variation. Con-

versely, with  $c_m$  in the model, the residual effects of shared environmental influences ( $c$ ) were estimated at 0. As can be seen in Fig. 2, variance increased steadily for twins born between 1940 and 1974, and the primary source of this increase was due to increasing shared environmental influences. Increases in genetic factors accounted for a small proportion of the total increase in variance.

Using the parameter estimates from the full model, it was possible to calculate the heritability and shared environmental influences for twins born in different birth cohorts. Because genetic factors changed at a significantly slower rate than the overall change in variance, the heritability of number of CD symptoms decreased slightly, from  $h^2 = 0.13$  (95% CI = 0.00–0.27) among twins born in 1940, to  $h^2 = 0.10$  (95% CI = 0.00–0.42) among twins born in 1974. The most striking changes were seen in estimates of shared and non-shared environmental influences. Shared environmental influences accounted for approximately 1% (95% CI = 0.1%–23%) of the variance among twins born



in 1940, but nearly 30% (95% CI = 1%–44%) of the variance among twins born in 1974. Non-shared environmental influences decreased from 86% to 60% among twins born in 1940 and 1974, respectively.

#### Testing assumptions about equal environments

Regression analyses (not shown) revealed significant main effects of both zygosity and birth cohort for similarity of childhood environment and amount of contact with co-twin. MZ twins reported more similar environments and higher levels of contact than DZ twins; twins born in more recent cohorts reported less similar childhood environments and higher levels of contact than twins born in earlier cohorts. The interaction between zygosity and birth year was also significant; there was a greater difference between MZ and DZ twins in both similarity of childhood environment and amount of contact among twins born in earlier cohorts than among twins born in more recent cohorts.

Nevertheless, neither the addition of the similarity of childhood environment nor the addition of amount of contact as moderators of  $a$ ,  $c$  and  $e$  significantly improved model fit compared to the model which allowed  $a$ ,  $c$  and  $e$  to vary only by cohort (LRC = 0.38,  $df = 3$ ,  $P = 0.94$ , for equal environment; LRC = 0.08,  $df = 3$ ,  $P = 0.99$ , for contact). Moreover, models allowing for only the moderating effects of similarity of childhood environment or amount of contact fit the data significantly more poorly than models that included cohort effects as well (LRC = 13.27,  $df = 3$ ,  $P < 0.004$ , for equal environment; LRC = 10.07,  $df = 3$ ,  $P < 0.02$ , for contact). Thus, the cohort effects found in the present study could not be accounted for by differences in similarity of childhood environment or amount of contact with co-twin.

#### Bias due to non-participation

To assess potential bias due to non-participation, we ran logistic regression analyses using PROC GENMOD (SAS, 1997) to predict participation of co-twin. Results revealed that participation of co-twin was predicted by zygosity, with MZ twins more likely to participate than DZ twins, birth year, with co-twins from more recent cohorts more likely to participate than co-twins of earlier cohorts, and by education level, with co-twins of more educated twins more likely

to participate than co-twins of less educated twins. Twin CD score, however, failed to predict participation of co-twin. In addition, neither the interaction between CD score and zygosity nor that between CD score and birth year was significant.

#### DISCUSSION

The present study found secular changes in both the mean level and variation in retrospective reports of CD obtained from male twins born between 1940 and 1974. The increase in mean level CD parallels the results from official sources indicating that antisocial behaviour among juveniles increased during the twentieth century (Bureau of Justice Statistics, 1999) and is consistent with increasing rates of CD found in national data sets (Robins, 1998). Variation in CD also increased with more recent cohorts; moreover, this increase in variation could not be explained solely by non-shared environmental factors, indicating that familial factors are responsible for the rise in variation. Although genetic factors could not be ruled out as the source of increasing familial resemblance, the best-fitting, most-parsimonious model suggested that the change in family resemblance for CD is due to an increase in shared environmental influences.

To our knowledge, only two other studies have examined cohort differences in the genetic and environmental architecture of CD. Our results are similar to those obtained by Waldman *et al.* (1989), who reported that the difference between MZ and DZ twin correlations was greatest among twins from older cohorts. In contrast, Slutske *et al.* (1997) reported that cohort failed to moderate genetic and environmental influences on a dichotomous measure of diagnosable CD. However, the Slutske *et al.* study differed from the present study in sample composition and measurement of CD, making direct comparisons difficult.

Results from the present study are particularly intriguing because they stand in contrast to results from studies examining cohort differences in alcohol use, smoking and age of sexual intercourse, all of which found that heritability increased with more recent cohorts (Dunne *et al.* 1997; Kaprio *et al.* 1991; Kendler *et al.* 2000). This discrepancy suggests that the mechanisms

responsible for the increase in CD may differ from the mechanisms that have caused secular changes in alcohol use, smoking and teenage sexual activity. Specifically, whereas the loosening of social restrictions against smoking, drinking and teenage sexual behaviour may have increasingly allowed for the expression of genetic influences on these behaviours, our results indicate that differences in between-family environmental factors have become an increasingly more powerful predictor of individual differences in CD.

This increase in shared environmental influences may be due to secular changes in certain sociodemographic factors, such as changes in the proportion of children living in poverty (Preston, 1984), and increasing rates of divorce, remarriage; and single parenthood (Cherlin, 1981; Hernandez, 1988). Factors such as income, overcrowding, and the quality of neighbourhoods and schools have been found to be related to CD among youth (Kazdin, 1995), as has family structure (see Demo & Acock, 1988, for a review). Because factors such as these are typically shared between members of the same family, but differ from family to family, they may explain both the increase in prevalence of CD as well as the increase in shared environmental influences on individual differences in CD.

A second set of possible mechanisms includes changes in parental attitudes and rearing practices. Mediational models suggest that the relationship between stressors associated with economic hardship, divorce, and single-parent-hood and increased child and adolescent behavioural problems can largely be explained by inefficient parenting practices (Forgatch *et al.* 1988; Conger *et al.* 1992). In particular, mediational variables such as parental monitoring and supervision are thought to be particularly important, and are commonly associated with levels of adolescent delinquency in cross-sectional samples (Patterson & Stouthamer-Loeber, 1984; Barber *et al.* 1994; Jacobson & Crockett, 2000). Likewise, the steady increase in maternal employment (Bianchi & Spain, 1986; Wilkie, 1991) may correspond to fewer opportunities for direct parental supervision of children. Studies have found that adolescents more removed from adult supervision are at increased risk for association with

deviant peers and for antisocial behaviour (Steinberg, 1986; Galambos & Maggs, 1991), and at least two studies found that the relationship between parental monitoring and adolescent behaviour problems was stronger among male adolescents whose mothers worked full time than among male adolescents whose mothers did not work (Crouter *et al.* 1990; Jacobson & Crockett, 2000). Behavioural genetic studies have demonstrated that although many affectional dimensions of parenting are at least partly heritable, familial resemblance for control-related dimensions of parenting, such as monitoring and discipline practices, are predominantly due to shared environmental influences (Rowe, 1981; Plomin *et al.* 1988; Kendler, 1996). Thus, factors such as parental monitoring and supervision may serve as shared environmental influences on antisocial behaviour.

Finally, studies of adolescent antisocial behaviour also point to peers as an important source of environmental influence (Patterson & Dishion, 1985; Snyder *et al.* 1986). To the extent that adolescent twins are likely to spend time in the same peer group, then the peer environment would be an important source of shared environment. One source of peer influence among same-sex twins might be their co-twin, and some researchers speculate that twin interaction effects are particularly important for youth antisocial behaviour. Failing to control for such effects might overstate estimates of heritability (Carey, 1992; Slomkowski *et al.* 1997). There is some evidence that adolescent twins do perform antisocial acts jointly (Rowe, 1985), and Rowe & Gulley (1992) found that adolescent siblings who spent more time together were more alike in deviant behaviour than those who spent less time together. However, in the present data set, there was no evidence for twin imitation effects, as the means and variance of CD were similar among MZ and DZ twins.

### Strengths and limitations

The present study has a number of different strengths, most notably that it uses data from a large, population-based sample of twins spanning a wide age range, allowing for an investigation of potential cohort effects. We also had data on the childhood environmental similarity of twins and amount of current contact with co-

twin, which allowed us to investigate whether the cohort effects found in the present study could be explained by secular trends in how parents treat their twin children, or by the increased amount of contact with co-twin found among younger cohorts. In addition, results predicting participation of co-twin found no evidence that participation rates were associated with CD scores, indicating that results are not likely to be biased by non-participation. The use of retrospective self-reports of CD is also a strength. Evidence suggests that individuals are more willing to report negative behaviours in self-report questionnaires than in structured face-to-face or phone interviews (Siemiatycki, 1979). Individuals can also be seen as more valid reporters of their own behaviour, as compared to parent or teacher reports (Robins, 1998). Finally, the use of retrospective data has at least two advantages: all individuals have passed through the age of risk, so developmental differences in rates of CD cannot bias results, and the same definition of CD can be applied to individuals in different cohorts (Robins, 1998).

The use of retrospective reports also has some limitations. In a prospective study, Henry *et al.* (1994) reported only a 0.25 correlation between self-report of delinquent behaviour assessed at age 13 and retrospective reports of delinquent behaviour prior to age 13 obtained when subjects were 18. Age may be associated with accuracy of recall; hence, the greater twin resemblance found among twins from more recent cohorts could be due to their younger age. If there are genetic influences on age changes in memory, older MZ twins may agree to a greater extent than older DZ twins, which is the pattern found in the present study. Unfortunately, the use of retrospective reports means that ageing effects on memory and cohort effects are inherently confounded. Future studies could use prospective, longitudinal designs to examine cohort effects, or could rely on official statistics, such as arrest rates, rather than self-reports of behaviour. Nevertheless, in the current data set, birth year was unrelated to short-term test-retest reliability, and a separate report considering the reliability of antisocial behaviour assessed at two different waves of data (approximately 18 months apart) found that reliability across waves was unrelated to birth year (Jacobson *et al.* 2000). Finally, Robins (1998) used data from

three national data sets to examine changes in the prevalence of CD across cohort. Although all three studies sampled individuals from the same birth cohorts, the studies themselves were conducted at different times; thus, birth cohort effects could be separated from age at recall effects. Robins found that all three studies demonstrated a systematic increase in the prevalence of CD across cohort, and that this increase could not be accounted for by recall bias.

A second primary limitation is that the sample is restricted to White male twins born in Virginia between 1940 and 1974. As the increase in shared environmental influences may be related to secular changes in sociodemographic factors, our results may not generalize to samples of males from different cultures, in different ethnic groups, or during different historical periods. Likewise, there is clear evidence that rates of CD are higher among adolescents than female adolescents (Cohen *et al.* 1993). Similar to the questions addressed in the present study, mean level sex differences in CD may or may not be related to differences in the underlying sources of variation. Some studies suggest that genetic and environmental influences on antisocial behaviour differ for males and females (Eley *et al.* 1999), whereas other studies find no significant sex differences (Slutske *et al.* 1997). An investigation into cohort differences in CD among females remains to be done.

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