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Twin Imitation for Antisocial Behavior: Implications for Genetic and Family Environment Research

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When twin pairs influence each other's behavior, observed variance is greater for MZ twins than for DZ twins under at least 1 of 2 conditions: (a) the trait has some heritability and (b) MZ twins influence each other more than do DZ twins. Applied to a trait that has an underlying continuous distribution but is measured as a dichotomy, the presence of reciprocal twin influence predicts that if the base rate for the trait is not exactly 50%, then the prevalence of the trait should differ in MZ and DZ twin pairs. This prediction held for registered criminality in a large twin cohort. Methods of analysis that permit reciprocal twin interaction not only provide better statistical fits to the data but also yield estimates of heritability that agree with adoption data. The results suggest that the genetic influence on registered criminality may be more modest than previously thought.

In the study of externalizing antisocial behavior (i.e., behavior that comprises the symptoms and signs of antisocial personality and some forms of drug and alcohol abuse), it has been widely demonstrated that deviance of an individual is influenced by the deviance of that individual's peer group. Although the fact that this empirical correlation is significantly different from 0.0 goes unquestioned, the causal pathways behind the observed relation remain obscure. To what extent is this association a causal effect of peer influence, and to what extent does it reflect assortative friendship? The answers to these questions have important implications not only for science but also for practical interventions into externalizing behaviors. If the causal pathway is strong, then interventions in which a vulnerable individual is identified and a deviant peer group is supplanted with one that engages in more acceptable social behavior may have a noticeable impact on the prevalence of externalizing behaviors. On the other hand, if the "birds-of-a-feather-flock-together" pathway explains all but a small part of the association, other types of interventions must be explored.

Empirical studies of individuals suggest that both types of pathways operate during the teen years (Cohen, 1977; Duncan, Haller, & Portes, 1968; Jessor & Jessor, 1977; Kandel, 1978; Kandel, Kessler, & Margulies, 1978). If siblings act as peers with one another, studies of twins and adoptive siblings should reveal what behavioral geneticists refer to as "common" sibling environment. In this context, common sibling environment is a latent variable that includes all those environmental effects that would make siblings *similar* in antisocial behavior. However, the empirical literature on genetics of crime and antisocial behavior has not revealed strong effects for common sibling environment (Mednick, Gabrielli, & Hutchings, 1984; Rowe, 1983; Rowe & Osgood, 1984). What is responsible for this discrepancy?

This study demonstrated that the published twin data for registered criminality in Denmark are better explained by a model that allows for the reciprocal influence of twins on each other than by a traditional model of twin similarity. In addition, estimated heritability from the peer influence model also agrees better with the estimates from Danish adoption data than do the estimates obtained from the traditional twin model.

Method

Christiansen (1968) identified twin pairs born in Denmark between 1880 and 1910 and followed them through official police and court records. He defined as "criminal" those persons who were recorded to have committed an act roughly equivalent to a felony in current United States jurisprudence. The follow-up was reported at various stages of completion (Christiansen, 1970, 1974; Cloninger, Christiansen, Reich, & Gottesman, 1978; Gottesman, Carey, & Hanson, 1983). The published data from the final follow-up (Cloninger & Gottesman, 1987) are given in this article.

The model used for data analysis is depicted in Figure 1 for pairs of opposite-sex, dizygotic twins. Technical aspects of the model, estimation of parameters, and the assessment of fit are given in the Appendix. In Figure 1, only the general features are explicated. In the model, G denotes a person's genotypic liability to criminality, C denotes the common environment liability, and U denotes unique environment. Common environment includes all environmental factors shared by siblings, with the notable exception of their phenotypes, that make them *similar*. Variables that would be included under common environment are parental age, occupational status, all types of community variables, and so forth. The unique environment includes all those environmental factors that make siblings *different* from each other. P denotes phenotypic liability, which is the weighted sum of genetic, common environmental, and unique environmental liabilities, the re-

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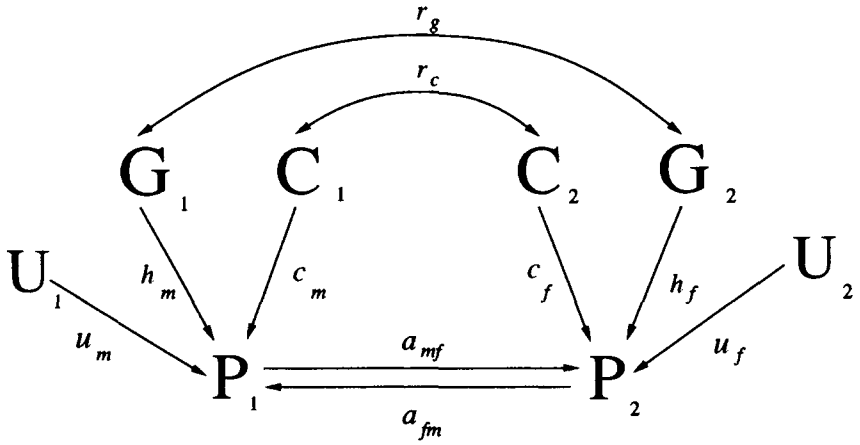

Figure 1. A linear model for twin resemblance under reciprocal twin interactions. (Opposite-sex fraternal pairs are depicted. r_g and r_c = correlations between genotypes and between common environments, respectively, of twin pairs. G = genotype; C = common environment; U = unique environment; and u, h, and c = weights of their respective liabilities. P = phenotype; a = imitation parameter. Subscripts 1 and 2 denote the first and second member of the pair, taken here as the male [subscript m] and the female [subscript f], respectively.)

spective weights being h, c, and u. Subscripts 1 and 2 refer to the first and second member of a twin pair (in Figure 1, the male and the female member, respectively). Subscripts m and f denote possible gender differences for male and female twins in the extent to which genetic factors, common environment, and unique environment contribute to liability. The proportion of total liability contributed by genetic individual differences, or heritability, is h^2 (regardless of gender), the proportion attributable to common environment variance is c^2, and the proportion attributable to unique environment variance is u^2.

The correlation between the genotypes of twin pairs is denoted by r_g. For identical, or monozygotic (MZ), pairs, $r_g = 1.0$; for same-sex fraternal, or dizygotic (DZ), twins, r_g is a function of assortative mating and nonadditive genetic variance; and if the genetic influences in male pairs are the same as those in female pairs (a phenomenon termed Sex × Genotype interaction), r_g for opposite-sex dizygotic pairs equals r_g for same-sex pairs. In the model, it is assumed that nonadditive gene action and assortative mating for criminality are small. Hence r_g is assumed to be .50 for same-sex DZ twins but may vary for opposite-sex DZ twins.

At this point, I digress to explain what the sex differences in parameters mean. If there were a difference between h_m and h_f but $r_g = .50$ for opposite-sex twins, one would conclude that the genetic factors that contribute to male antisocial behavior are the same as those that contribute to female antisocial behavior, but the genetic factors have different *impacts* in male and female members. If $r_g < .50$ but $h_m = h_f$, one would conclude that genetic factors have equal impact in male and female siblings but that the *genetic mechanisms* are not the same in the sexes. This might occur if aggression were a more important trait for male antisocial behavior but disinhibition were more important for female antisocial behavior.

The correlation between the common environments of male and female siblings is denoted by r_c. For both MZ and DZ same-sex pairs, $r_c = 1.0$. For opposite-sex pairs, r_c is a free parameter that measures the extent to which common environmental factors in male siblings are the same as those in female siblings. For example, if housing density during the formative years were an important factor in female but not male antisocial behavior, $r_c < 1.0$. The meanings of c_m, c_f, and r_c are analogous to those of h_m, h_f, and r_g, except that the conclusions pertain to the common environmental effects and not to the genes.

There are two further components to the model: the threshold component and the reciprocal interaction component. According to the threshold part of the model, the *G*s, *C*s, *U*s, and *P*s in Figure 1 are latent variables that denote *liability* toward criminality, as defined earlier. It is assumed that all these liabilities are continuous variables and that there is some mathematical function that can transform their scales into multivariate normal distributions. It is further assumed that persons whose phenotypic liabilities exceed a certain threshold, t, are registered for criminality. This type of model—in which an observed dichotomy is assumed to be the result of continuous underlying liability—has been applied in many areas of genetics ranging in diversity from animal breeding to medical conditions such as polydactyly to behavioral syndromes such as schizophrenia (Gottesman, 1990; Gottesman & Shields, 1967; Reich, Rice, Cloninger, Wette, & James, 1979; Rice & Reich, 1985) and is also the basis for interpretation of the tetrachoric correlation in many psychometric models.

The second component of the model is the reciprocal interaction between the two siblings, represented by the arrows from P_1 to P_2 and from P_2 to P_1. When the imitation parameter, a, exceeds 0.0, antisocial behavior on the part of one member of the twin pair increases the probability that the other member will engage in antisocial behavior. The same principle holds at the other end of the liability distribution: When $a > 0.0$, prosocial behavior of one member increases the probability of prosocial behavior of the other. This latter process has been termed *cooperation* or *imitation* (Carey, 1986a; Eaves, 1976; Heath, 1990; Heath, Neale, Hewitt, Eaves, & Fulker, 1989). When $a < 0.0$, *competition* or *contrast* is modeled; that is, the antisocial behavior of one member increases the probability of prosocial behavior of the twin partner.

The joint occurrence of a threshold trait and reciprocal sibling interaction makes a nonobvious prediction: When the prevalence of the dichotomous trait is not equal to .50 and when there is some heritable influence, the prevalence of the trait differs between samples of MZ and DZ twins. When paths a and h in Figure 1 both exceed 0.0, the variance in phenotypic liability for MZ twins is greater than the variance for same-sex DZ twins (Carey, 1986a; Eaves, 1976). A similar increase in variance would also occur when the value of a is greater for MZ pairs than for DZ pairs—that is, when MZ twins tend to imitate each other more than do DZ twins (Carey, 1986a).

This situation is illustrated in Figure 2 for a single gender. In Figure 2, phenotypic liability for DZ pairs is scaled as a standard normal. The population prevalence of the trait in a zygosity is the area under the curve from the threshold t to positive infinity. If t is not at the mean, the prevalences of the trait differ among MZ and DZ pairs (see Figure 2). Hence if members of twin pairs are peers and positively influence each other's liability, MZ twins should have higher rates of criminality than do the comparable same-sex twins. Similarly, if the peer influence is less in opposite-sex pairs than it is in same-sex pairs, then the prevalence of registered criminality is greater in same-sexed DZ pairs than in opposite-sex pairs. In this way, a simple test for the presence of sibling effects is a test of the equality of base rates for criminality among MZ, same-sex DZ, and opposite-sex DZ pairs. A difference in these base rates would constitute evidence of sibling effects.

In addition to detecting the presence or absence of sibling effects, it is also desirable to obtain some estimate of their magnitude and the extent to which they influence estimates of heritability and common environment. To do this, mathematical models must be fitted to the data.

A complete model would have 14 free parameters: h_m and h_f, to allow for differences in heritability for male and female siblings; c_m and c_f, to permit gender differences in the common environment; r_g, the genetic correlation between male and female siblings or, in other words, the extent to which genotypic values in male siblings predict those in female siblings; r_c, the extent to which common environmental factors in male siblings are correlated with those in female siblings; t_m and t_f, to permit different thresholds in male and female siblings and allow for the fact that the prevalence of registered criminality in men exceeds that in women; and, finally, six imitation parameters (a_{mzm}, a_{mzf}, a_{dzmm}, a_{dzff}, a_{dzmf}, and a_{dzfm}) for, respectively, MZ male pairs, MZ female pairs, DZ male pairs, DZ female pairs, DZ male–female pairs, and DZ female–male pairs. Heath et al. (1989) may be consulted for further details about this type of model.

However, this complete model is not identified; that is, information in the data is not sufficient to permit estimation of each of these 14 unknowns. Instead of comparisons of all possible models with one another, an a priori hierarchy was established, and three general models were fitted to the data. Specific details of these three models are given in the Appendix. Here in text, the broad outline is given.

In all three general models, $a_{dzmf} = a_{dzfm} = 0.0$. In the first, $r_g = .50$ for opposite-sex pairs and r_c was free. In the second, r_c was constrained to be 1.0 and r_g was free. In the third, $r_g = .50$ and $r_c = 1.0$. The third model gave a satisfactory fit to the data and was accepted as the general model for further hypothesis tests. The next set of hypotheses involved tests for gender differences in h, c, and a. Again, the most parsimonious model was accepted for further tests. The final set of hypotheses involved setting h, c, and a to 0.0.

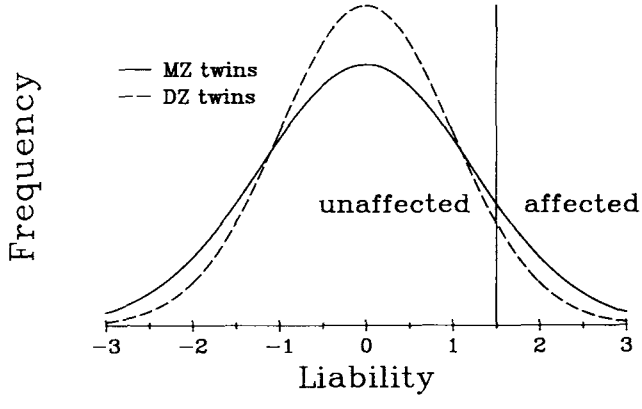

Figure 2. Distribution of phenotypic liability in monozygotic (MZ) and same-sex dizygotic (DZ) twins and the proportion affected when the trait is dichotomized at threshold t.

Two statistics were used to assess the adequacy of a model's fit to data. The first was the chi-square goodness of fit, which indexed the extent to which a model fit the data in relation to a hypothetical model that would perfectly explain all the observed data in Table 1. A low value of chi-square for the degrees of freedom suggests a good fit of the model; high chi-square values suggest that the model should be rejected. The second test statistic was the likelihood ratio chi-square, for which the fit of one model was compared with that of a nested model. It is possible (and actually happened in this study) that a model that fits satisfactorily with the goodness of fit can be rejected with a likelihood ratio chi-square.

Results

The observed data on registered criminality are given in Table 1. A very simple test of the model is a chi-square goodness of fit for the null hypothesis that the marginal prevalence of registered criminality is the same in all zygosity groups. This test yielded significant results for male twins and marginally significant results for female twins (Table 2). Because the prevalence of criminality was highest in MZ twins, lower in same-sex DZ twins, and lowest in opposite-sex DZ, a cooperation or imitation process appears plausible.

A summary of the model fitting is given in Table 3. The three general models all fit the data very well, and so the most parsimonious (i.e., wherein no gender differences in r_g and r_c were assumed) was selected as the general model for testing gender differences in the coefficients h and c. Except for the obvious difference in thresholds, there is no evidence that heritability or common environment differs in male and female twins. These tests are the chi-squares for row $h_m = h_f$ and for row $c_m = c_f$, respectively. Nor is there evidence that the imitation (a) parameters differ between MZ male and MZ female twins. Hence the fourth model under gender differences was accepted as a general model for the further set of tests.

For the models that do not include gender differences, one encounters the unusual finding in twin studies that the heritability can be set to 0.0 without a dramatic loss in fit. Similarly, the model that sets c to 0.0 and the model that equates a in MZ and DZ twins provide satisfactory fits.

However, three models can be rejected by the likelihood ratio test. In two of these models, the imitation parameter is set to 0.0; in the third, it is set equal for MZ and DZ twins with no heritability. This pattern strongly suggests the importance of some form of reciprocal sibling influence in the development of liability toward crime.

Of equal importance to the statistical fit of a model is the magnitude of the parameter estimates because large samples can often reveal statistically significant effects that are trivial in magnitude. Table 4 contains estimates from several parsimonious models that involve peer influence. Also given are the parameter estimates from two traditional models for twin data. (Because several ad hoc assumptions had to be made in the analysis, the parameter estimates are not unbiased. The extent of bias is discussed in the Appendix.)

Although my analysis rejects the two traditional models, the

Table 1
Concordance for Registered Criminality Among Danish Twins

Type of twin	No. of twin pairs registered			Tetrachoric correlation
	Both	One	Neither	
Monozygotic male	25	48	292	.74
Dizygotic male	26	120	554	.47
Monozygotic female	3	12	332	.74
Dizygotic female	2	26	662	.46
Dizygotic male–female	7	215[a]	1,851	.23

Note. Data are from Cloninger and Gottesman (1987).
[a] One hundred ninety-two male twins registered, female twins not registered; 23 female twins registered, male twins not registered.

estimates from them are the ones disseminated in the literature on the genetics of antisocial behavior. The traditional models suggest a strong heritability: the genotype contributes to between 57% and 71% of the variance in liability. The model that permits peer influence suggests more modest estimates of heritability, in one case actually approaching 0.0.

Discussion

Several strong conclusions (and several weak ones) can be drawn from this analysis. A weak conclusion is that there is no evidence in the twin data for gender differences in heritability or in the common environment that contribute to criminal liability. These results suggest that the *same* familial factors (both genetic and family–environmental) that contribute to liability toward criminality in male twins also contribute to the liability for female twins. It simply takes *more* of these factors to make female twins behave in a criminal manner. The nature of these familial factors and the reason why it may take more of them to produce deviant behavior in female twins cannot be determined from this analysis. This conclusion is weak because it is based on several untested assumptions. The data crucial for strong hypothesis testing (those on same-sex and opposite-sex siblings, adoptive siblings, and reared-apart siblings) are not available.

A strong conclusion is that the assumptions of the traditional twin method may be violated for phenotypes related to externalizing antisocial behavior. All models that set the imitation parameter, *a*, to 0.0 were rejected. When imitative dyadic sibling interaction is present but is ignored in data analysis, its effects are confounded with both genetic and common environment effects. If MZ twins influence each other more than do DZ twins—a hypothesis that cannot be rejected in this analysis—genetic effects for criminal liability may actually be small.

Other investigators have not found statistical evidence for such strong interaction, but at the same time they have reported *substantive* evidence congruent with reciprocal twin influence (Dalgaard & Kringlen, 1976; Rowe, 1985, 1986). Statistical evidence would consist of model fits that demonstrate a greater variance among MZ pairs than among DZ pairs. The reason why Rowe (1985, 1986) did not report such evidence and why Dalgaard and Kringlen's (1976) results did not reach significance may be attributable to small sample sizes. With only twin data, a sample involving hundreds of pairs is required in order to achieve sufficient power for detecting this type of reciprocal interaction (Carey, 1986a; Eaves, 1976).

With regard to substantive evidence, Rowe (1985, 1986) reported that adolescent twins are likely to engage in the same antisocial acts together. Dalgaard and Kringlen (1976) reported that the twin concordance was correlated with the extent to which the twins identified with each other. Both findings are consistent with the hypothesis that some reciprocal interaction may influence antisocial tendencies.

Although this discussion might be interpreted as challenging the utility of the twin method for the study of antisocial behavior, another strong conclusion is, paradoxically, that large-scale

Table 2
Prevalence of Registered Criminality in Danish Twins by Sex and Zygosity

Zygosity	Male twins[a]		Female twins[b]	
	Number	Percentage criminal	Number	Percentage criminal
Monozygotic	730	13.42	694	2.59
Same-sex dizygotic	1,400	12.29	1,380	2.17
Opposite-sex dizygotic	2,073	9.55	2,073	1.47

Note. Number = total number of subjects.
[a] χ^2 (2, N = 4,233) = 11.00, $p < .001$. [b] χ^2 (2, N = 4,167) = 4.70, $.05 < p < .10$.

Table 3
Chi-Square Goodness of Fit for Registered Criminality Among Danish Twins

Constraints	*df*	Likelihood ratio χ^2
General model		
$r_c = 1.0$	5	1.89
$r_g = 0.5$	5	2.06
$r_c = 1.0$, $r_g = 0.5$	6	2.06
Model of gender differences		
$h_m = h_f$	7	2.22
$c_m = c_f$	7	2.06
$a_m = a_f$	8	2.42
$h_m = h_f$, $c_m = c_f$, $a_m = a_f$	10	2.43
Model of no gender differences		
$h = 0$	11	3.81
$c = 0$	11	2.43
$a_{mz} = a_{dz}$	11	3.37
$a_{mz} = a_{dz} = 0$	12	14.15**
$h = 0$, $a_{mz} = a_{dz}$	12	9.98*
$h = 0$, $a_{mz} = a_{dz} = 0$	13	29.25**

Note. r_c = correlation between common environments of male and female siblings; r_a = correlation between genotypes of twin pairs; h_m and h_f = weights of genetic liability for male and female siblings, respectively; c_m and c_f = weights of common environmental liability for male and female siblings, respectively; a_m, a_f, a_{mz}, and a_{dz} = imitation parameters for male siblings, female siblings, monozygotic twins, and dizygotic twins, respectively.
* $p < .05$ ** $p < .001$.

studies of twins are exceptionally important for the study of sibling interactions. The twin method is not at fault; the problem is the inappropriate use of the method. Indeed, if causal peer effects are important, the twin method used longitudinally with measures on the best friends of the twins may be powerful enough to uncover such effects because, depending on the model, these effects predict different variances and covariances among twins and their friends as a function of zygosity. The methods developed in the field of genetic epidemiology for modeling reciprocal interactions (Carey, 1986a; Eaves, 1976; Eaves, Eysenck, & Martin, 1989; Heath et al., 1989), together with those used for modeling assortment processes (Carey, 1986b; Cloninger, 1980; Van Eerdewegh, 1982) and cohabitation effects (Hopper & Culross, 1983), are powerful tools that cannot be ignored by contemporary psychologists. Not only do these techniques permit stringent data analysis, but they also highlight the types of relationships for which data should be gathered (e.g., children with no siblings; adoptive siblings) in order to resolve competing hypotheses.

What can be said about the genetics of antisocial behavior from this analysis? The literature on biological relatives reared apart suggests a genetic effect on criminality that cannot be confounded by imitative phenomena (Bohman, Cloninger, Sigvardsson, & von Knorring, 1982; Cadoret, 1978; Crowe, 1972, 1974; Hutchings & Mednick, 1971, 1975; Mednick et al., 1984). Analyses of the Danish adoption cohort through use of the threshold model yielded heritability estimates of approximately .30 (Baker, 1986; Baker, Mack, Moffitt, & Mednick, 1989), which is in quite good agreement with the one obtained in my research when genetic effects along with sibling influence are modeled. The adoption data, however, are not in good agreement with the .60–.70 estimate based on the traditional twin analysis. Hence a parsimonious interpretation of the Danish twin and adoption data is that both processes—heritability and sibling interaction—contribute to liability toward criminal, antisocial behavior. Naturally, these results from a country such as Denmark, which has a homogeneous social structure, cannot be readily extrapolated to a country such as the United States, which has a more heterogeneous social structure. The available adoption evidence suggests some heritability in U.S. populations (Cadoret, 1978; Cadoret, Cain, & Crowe, 1983; Crowe, 1972, 1974), but there is a lack of large-scale, population-based studies with U.S. samples that would permit more detailed examination of the interplay between heredity and environment in the development of antisocial behavior.

Table 4
Parameter Estimates From Peer Influence Models and From Traditional Twin Models for Registered Criminality in Danish Twins

	Peer influence			Traditional	
Parameter	General model	$a_{mz} = a_{dz}$	$h^2 = 0$	General model	$c^2 = 0$
Threshold					
Male twins	1.28	1.28	1.28	1.23	1.23
Female twins	2.18	2.17	2.19	2.08	2.08
Heritability (h^2)	.37	.45	.00	.57	.71
Common environment (c^2)	.00	.00	.28	.34	.00
r_g	.64	.52	.00	.24	.36
a_{mz}	.22	.16	.27	.00	.00
a_{dz}	.16	.16	.15	.00	.00
χ^2	2.06	3.37	3.81	14.15	14.25
df	6	11	11	12	13

Note. r_g = correlation between genotypes of twin pairs; a_{mz} and a_{dz} = imitation parameters for monozygotic and dizygotic twins, respectively.

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(Appendix follows on next page)

Appendix

Because *G, C, U,* and *P* are latent and unscaled, they are conveniently taken as standard normal variables with the constraint that $h^2 + c^2 + u^2 = 1.0$. The general model has the 14 free parameters specified in the text. Unfortunately, without other data such as those on only children, who lack the reciprocal influence of a sibling, the full model is not identified. To solve the problem, the opposite-sex (OS) pairs were treated as a "baseline" group. It was assumed that imitation was very low for these pairs so that the two imitation parameters for this grouping could effectively be treated as 0.0. The methods for analytical derivation of the expected variances and covariances for the latent liability variables are given elsewhere (Carey, 1986a). The expectations are given in Table A1.

Given the model, the phenotypic liability for the OS pairs was scaled as a normal with a mean of 0.0 and a variance of 1.0 but with different thresholds, t_m and t_f, for the male and female member of the pair, respectively. The prevalence of registered criminality among same-sex DZ and MZ pairs may thereby be calculated in terms of the phenotypic variance for the respective groups. For example, the standardized threshold for MZ male twins is

$$z_{mzm} = t_m/\sqrt{V(P)_{mzm}}, \tag{A1}$$

where $V(P)_{mzm}$ is as given in Table A1. The proportion of expected criminal MZ male twins is the area to the right of normal curve for this value of z_{mzm}. Similar expressions can derive *z*s for DZ same-sex male twins, MZ female twins, and DZ same-sex female twins, except that the denominators would be their own values of the variance in liability. Through the expressions in Table A1 and Equation A1, it can be readily verified that when $a_{mz} \geq a_{dz}$ and $a_{mz} > 0$, the threshold trait has a higher prevalence in MZ than in DZ twins if $t \neq 0.0$.

If the assumption about opposite-sex pairs is violated (i.e., the imitation parameters for these pairs are indeed greater than 0.0), the test for the *presence* of reciprocal peer influences is unaffected. However, the parameter estimates from the model are biased to an unknown degree. Other models that contain different assumptions (e.g., allowing the influence of a female twin on a male cotwin to be same as that of a female twin on a female cotwin) may be fit, but for reasons of parsimony, our model did not contain such assumptions.

One further assumption is required about the opposite-sex DZ pairs. With my data, it was not possible to simultaneously estimate parameters r_g and r_c. Hence three general models were initially fit to the data. One fixed r_g at .50, the second fixed r_c at 1.0, and the third fixed both r_g and r_c to these values. Although all three models fit very well, conclusions about potential gender differences in either genes or environment should await the pertinent data critical for this issue (e.g., data on adoptive siblings and siblings reared apart).

The observed data for each sex and zygosity combination may be represented as a 2×2 contingency table. Let N_{ijk} denote the observed number of pairs for cell *jk* for the *i*th sex–zygosity grouping with *j* and *k* being either 0 (not registered for criminality) or 1 (registered). Because the phenotypic liability is distributed as a bivariate normal, the proportion of pairs expected in this cell is a function of the phenotypic variance and the covariance for the *i*th sex–zygosity group. For example, the proportion of MZ female pairs in which the first twin is unregistered (0) and second twin is registered (1) is given by the bivariate integral

$$p_{mzf01} = \int_{-\infty}^{t} \int_{t}^{\infty} \phi(P_1, P_2) dP_1 dP_2, \tag{A2}$$

where $\phi(P_1, P_2)$ is the normal density function with means of 0.0, variances of $V(P)_{mzf}$ as given in Table A1, and $\text{cov}(P_1, P_2)_{mzf}$ as given in Table A1. Parameters of the model may be estimated, and a statistical index of goodness of fit may be derived by maximizing the log of the conditional likelihood, *L* (i.e., the likelihood given the sex–zygosity group), over all the observed cells:

$$L = \sum_i \sum_j \sum_k N_{ijk} \log (p_{jk|i}), \tag{A3}$$

where $p_{jk|i}$ is the predicted proportion of the *i*th zygosity in cell *jk*. Numerical methods were used to perform the integration in Equation

Table A1

Expected Phenotypic Variances and Covariances for Monozygotic and Dizygotic, Male and Female, Same-Sex and Opposite-Sex Twin Pairs in the Presence of Imitation–Contrast Effects

$$V(P)_{mzm} = [1 + a_{mzm}^2 + 2a_{mzm}(h_m^2 + c_m^2)]/(1 - a_{mzm}^2)^2$$
$$V(P)_{mzf} = [1 + a_{mzf}^2 + 2a_{mzf}(h_f^2 + c_f^2)]/(1 - a_{mzf}^2)^2$$
$$V(P)_{dzm} = [1 + a_{dzm}^2 + 2a_{dzm}(.5h_m^2 + c_m^2)]/(1 - a_{dzm}^2)^2$$
$$V(P)_{dzf} = [1 + a_{dzf}^2 + 2a_{dzf}(.5h_f^2 + c_f^2)]/(1 - a_{dzf}^2)^2$$
$$V(P)_{dzm\text{-}os} = 1$$
$$V(P)_{dzf\text{-}os} = 1$$
$$\text{cov}(P_1, P_2)_{mzm} = [(h_m^2 + c_m^2)(1 + a_{mzm}^2) + 2a_{mzm}]/(1 - a_{mzm}^2)^2$$
$$\text{cov}(P_1, P_2)_{mzf} = [(h_f^2 + c_f^2)(1 + a_{mzf}^2) + 2a_{mzf}]/(1 - a_{mzf}^2)^2$$
$$\text{cov}(P_1, P_2)_{dzm} = [(0.5h_m^2 + c_m^2)(1 + a_{dzm}^2) + 2a_{dzm}]/(1 - a_{dzm}^2)^2$$
$$\text{cov}(P_1, P_2)_{dzf} = [(0.5h_f^2 + c_f^2)(1 + a_{dzf}^2) + 2a_{dzf}]/(1 - a_{dzf}^2)^2$$
$$\text{cov}(P_1, P_2)_{dzos} = r_g h_f h_m + r_c c_f c_m$$

Note. It is assumed that the imitation contrast for opposite-sex dizygotic pairs is effectively 0.0. $V(P)$ = phenotypic variance; a = imitation parameter; h = weight of heritability liability; c = weight of common environmental liability; *mzm* = monozygotic male twins; *m* = male; *mzf* = monozygotic female twins; *f* = female; *dzm* = dizygotic male twins; *dzf* = dizygotic female twins; *dzm-os* = dizygotic male twins–opposite sex; *dzf-os* = dizygotic female twins–opposite sex; P_1 and P_2 = phenotypic liabilities for the first and second members, respectively, of a twin pair; *dzos* = dizygotic opposite-sex twins; r_g = correlation between genotypes of twin pairs; r_c = correlation between common environments of twin pairs.

A2 and to find the set of parameter values that maximized L in Equation A3.

Because the data are from a full population cohort, the intraclass relationship among twins dictates that the two "discordance" cells in the 2×2 tables are equal for the same-sex pairs. In effect, three instead of four cells per contingency table are obtained for the four same-sex pairs. Together with the four free cells for the opposite-sex DZ pairs, a total of 16 degrees of freedom are available for the general model. A chi-square goodness of fit may then be constructed by fitting a model that completely explains the data and comparing the fit with that of a reduced model. The degrees of freedom for this chi-square are 16 minus the number of free parameters in the reduced model.

If L_j is the log likelihood for model j and L_k is the log likelihood for a model nested within j, then twice the difference in log likelihoods, or $2(L_j - L_k)$, is approximately a chi-square with degrees of freedom equal to the number of free parameters in model j minus the number of free parameters in model k.

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