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# The Heritability of Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies

**Dehryl A. Mason[1] and Paul J. Frick[1,2]**

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*In this paper, we describe a quantitative summary of 12 twin (n = 3795 twin pairs and 3 adoption studies = 338 adoptees) published since 1975 which provided 21 estimates of the heritability of antisocial behavior. Medium to large effect sizes were found for genetic influences across studies, with approximately 50% of the variance in measures of antisocial behavior attributable to genetic effects. Although effect sizes did not vary across different definitions of antisocial behavior (criminality, aggression, or antisocial personality), significantly larger estimates of genetic effect were found for severe manifestations of antisocial behavior. The importance of severity was further underscored by the significantly larger effects obtained in studies using clinic-referred samples compared to the effects obtained in studies using volunteer samples. Demographic characteristics of the samples did not influence effect sizes, although studies using more stringent methodology tended to find larger effects. These results must be interpreted in light of the small literature that was suitable for the meta-analysis due to numerous methodological limitations in existing studies.*

**KEY WORDS:** genetics; antisocial behavior; criminality; aggression; meta-analysis.

## INTRODUCTION

There is a substantial literature on the presence of a familial link to antisocial behavior (see Frick, 1994). However, there is great debate over the mechanisms involved in the intergenerational transmission (Patterson, Reid, & Dishion, 1992). Because family history studies confound genetic

[1]Department of Psychology, University of Alabama, P.O. Box 870348, Tuscaloosa, Alabama 35487.
[2]To whom correspondence should be addressed.



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and environmental influences (i.e., children and parents share both genes and environment), the intergenerational link to antisocial behavior has been a focal point to the longstanding "nature vs nurture" debate (DiLalla & Gottesman, 1991; Widom, 1991).

To begin to tease apart the confounding influences of genetics and environment, many behavioral genetic researchers have used twin and adoption methods to clarify the potential role of heredity in the development of antisocial behavior. The purpose of this paper is to provide a quantitative summary of these studies. However, our goal is not to test whether or not hereditary influences are the *sole* causes of antisocial behavior. We hold strongly to the belief that pitting genetic against environmental influences as competing causes is inappropriate for the study of any psychological syndrome. Instead, it is more appropriate to view psychological disorders as being a function of both biological predispositions and environmental influences. The purpose of this study is to provide an estimate of the *strength* of genetic influences to antisocial behavior and, more importantly, to determine if there are moderators to this estimate.

Although there are numerous qualitative reviews of the genetic research on antisocial behavior (e.g., Dilalla & Gottesman, 1991; Mednick & Kandel, 1988; Plomin, Nitz, & Rowe, 1991; Rutter *et al.,* 1990), a quantitative review is especially important in this area of research because of the strong philosophical beliefs that are held on the potential role of genetics in the development of antisocial behavior (see Adler, 1992). As a result, a quantitative meta-analysis limits the potential for bias on the part of a reviewer and provides a more objective summary of the empirical evidence.

In addition, a meta-analysis provides a mechanism for testing potential moderating influences to the effect sizes across studies (Wolf, 1988). For example, there have been many definitions of antisocial behavior used in twin and adoption studies, such as legal definitions of criminality, verbal or physical aggression, and personality characteristics associated with antisocial behavior. Distinguishing among these definitions is important because research with children and adults has indicated that these definitions are related but are not synonymous (Hare, Hart, & Harpur, 1991; Kelso & Stewart, 1986; Loeber, 1990; Tremblay *et al.,* 1991). As noted by Plomin *et al.* (1991), despite the overlap between these behavioral definitions, the "genetic relationships among these disorders is unknown" (p. 122). Therefore, it is important to test whether the estimated genetic effects are influenced by the definition of antisocial behavior that is employed.

A second potential moderator is the severity of antisocial behavior. Twin and adoption studies have varied greatly on the severity of antisocial behavior that is measured, ranging from the number of hits to a toy Bobo doll (Plomin, Foch, & Rowe, 1981) to the involvement in criminal activity,

including violent crimes (Cloninger, Christiansen, Reich, & Gottesman, 1978). Severity has already been identified as an important variable in the chronicity of antisocial behavior (Hare *et al.*, 1991; Loeber, 1991) and in the strength of the intergenerational link to antisocial behavior (Mednick, Gabrielli, & Hutchings, 1984; Osborn & West, 1979; Wilson, 1975). Therefore, it is possible that severity may also be a moderator to the degree of variance explained by heredity.

Several demographic variables are also potential moderators. Like severity, age has been linked to the chronicity of antisocial behavior, with the onset of problem behavior prior to adolescence leading to an increased likelihood of continuation into adulthood (Loeber, 1991). For example, Robins (1966) reported that when antisocial behavior began prior to the child's 11th birthday, children were twice as likely to exhibit antisocial behavior as an adult (Robins, 1966). Similarly in the delinquency literature, recidivism has been linked to early onset of delinquent behavior (Hanson, Hennggeler, Haefle, & Rodick, 1984; Loeber & Dishion, 1983). Gender is another demographic variable that might moderate the effects of heredity. Gender differences have been found in the stability of aggression over time, with male aggression appearing more stable (Huesmann, Eron, Lefkowitz & Walder, 1984). However, it appears that offspring of antisocial mothers might be at greater risk for antisocial behavior than offspring of antisocial fathers (see Robins, 1991).

In summary, the literature on antisocial behavior has indicated that there are several definitional and demographic distinctions within the broad category of antisocial behavior that have proven important for understanding the chronicity of antisocial behavior and the risk in offspring of antisocial parents. Therefore, in this paper we present a quantitative summary of recent twin and adoption studies in which we test whether or not these distinctions are systematically related to the degree of hereditary influence. Specifically, in addition to providing an overall estimate of the influence of heredity on measures of antisocial behavior, we test whether or not the type of antisocial behavior, the severity of the antisocial behavior, or the demographic composition of the samples moderate the estimated degree of genetic involvement.[3]

[3]During data collection for the present study, another meta-analysis on the genetic contributions to crime was published by Walters (1992). This meta-analysis found that the genetic contribution to measures of criminal behavior was general weak. We feel that the Walters (1992) meta-analysis was limited methodologically in several respects, which makes interpretation of the data difficult. First, the method of obtaining studies for the initial item pool was not well specified, nor was the method for retaining studies for the meta-analysis. Second, data from overlapping or identical samples were included "if they shed new light on the gene-crime question" (p. 598, Walters, 1992). This not only inflates the contributions of any single sample, depending on how many times it was included, but it also makes the

## METHOD

### Selection of Studies

An initial pool of studies was located by several methods. Computerized searches of *Psychological Abstracts* and *Dissertation Abstracts* were conducted. Key words employed in these searches included gene, inherit, heritable, heredity, biology, predisposition, predict, violent, conduct disorder, antisocial, sociopath, juvenile delinquency, crime, cruelty, behavior disorder, behavior problems, explosive personality, vandalism, torture, recidivism, temper, psychoticism, personality, negative emotionality, steal, bully, twin, adoptee, and adoptive. Early studies of the genetic influence on antisocial behavior were plagued with methodological problems (Mednick & Kandel, 1988; Rosenthal, 1975). Because of this and the fact that the relatively few studies conducted before 1975 failed to report data in a manner which met the statistical criteria for this meta-analysis (discussed below), only articles published between 1975 and 1991 (the end of data collection) were included in this meta-analysis.

Attempts to locate appropriate articles published during the period between 1975 and 1991 also included searches of individual journal volumes published during these years for the following periodicals: *Aggressive Behavior*, *Archives of General Psychiatry*, *Biological Psychiatry*, *Behavior Genetics*, *Crime and Delinquency*, *Diseases of the Nervous System*, *Journal of Abnormal Child Psychology*, *Journal of the American Academy of Child and Adolescent Psychiatry*, *Journal of the American Academy of Child Psychiatry*, *Journal of Clinical Psychiatry*, *Journal of Crime and Delinquency*, *Journal of Nervous and Mental Disease*, *Journal of Personality and Social Psychology*, *Neuropsychobiology*, *Psychological Bulletin*, and *Social Biology*.

A search for appropriate articles also included written correspondence soliciting information about twin and adoption studies appropriate for this meta-analysis to the following researchers who have published recent articles in the area: Laura Baker, Michael Bohman, Thomas Bouchard,

error variance between effects sizes nonindependent, violating a basic assumption of most meta-analytic techniques (Wolf, 1986). Third, the Walters (1992) meta-analysis included many twin and adoption studies that confounded antisocial/criminal behavior with other psychological dysfunctions (e.g., substance abuse, general psychological dysfunction), making it unclear whether the effect size was actually measuring the genetic contribution to antisocial behavior/crime or psychological dysfunction in general. Fourth, it included adoption studies in which the same measures of antisocial/criminal behavior were not used in comparing the biological and adoptive backgrounds of probands, making it possible that effect sizes were affected by the assessment of different constructs in biological and adoptive relatives. Fifth, the Walters (1992) study included many studies conducted prior to 1975 in which the methodology was so flawed, that obtaining meaningful conclusions from the data would be difficult (Mednick & Kandel, 1988; Rosenthal, 1975).

Remi Cadoret, David Fulker, William Grove, Barry Hutchings, Sarnoff Mednick, Terrie Moffit, David Owen, Robert Plomin, David Rowe, Nancy Segal, and Phillipe Rushton.

From these searches, an initial pool of 70 studies was found. These studies were then reviewed to determine if they met five inclusionary criteria. First, studies must have used a methodology that either compared monozygotic (MZ) and dizygotic (DZ) twins or compared the association between antisocial behavior in adoptees with antisocial behavior in biological and adoptive parents. Second, the studies must have measured some aspect of antisocial behavior, through measures of either antisocial personality, aggression, or criminal activity, but these measures could not be confounded with other forms of psychological disturbance. Although studies were included if substance abuse was considered *part* of the definition of antisocial behavior, studies in which substance abuse alone was considered a measure of antisocial behavior were eliminated from consideration.[4] Third, the studies must have used a research design that compared Mz and Dz twins on the same type of antisocial behavior. Similarly, the association between adoptee and the behavior of both the biological and the adoptive parents must be compared on the same measure of antisocial behavior. For example, a study that measured the relationships between antisocial personality in adoptive parents, criminality in biological parents, and criminal behavior in adoptees was excluded (Crowe, 1975). Fourth, studies which included analyses only on a subset of a sample that was already part of our meta-analyses were excluded. Finally, data from each study had to be presented in a form that allowed for the calculation of at least one effect size, using the procedure described below.

As a result of this procedure, only 15 (21%) of the original pool of 70 studies met the inclusionary criteria. These included 12 twin studies and 3 adoption studies, which led to the calculation of 21 effect sizes (18 from twin studies and 3 from adoption studies). The 15 studies included in the meta-analysis are listed in the Appendix. The most common reason for exclusion was due to (1) a failure of adoption studies to compare the association of adoptee antisocial behavior with both biological and adoptive

[4]There is substantial evidence that substance abuse and antisocial behavior should not be used interchangeably. Specifically, although substance abuse can occur as one aspect of a broader pattern of impulsive and antisocial behavior, this only accounts for a small percentage of people who abuse substances (e.g., Stewart & Wilcox, 1987). More importantly, the adoption study by Cadoret, Troughton, O'Gorman, and Heywood (1986) provides convincing evidence that the hereditary influences to antisocial behavior and substance abuse are largely independent. Therefore, we felt that any study which confounded substance abuse and antisocial behavior ($n = 6$) should be eliminated from review because it would be impossible to determine whether the estimate of heritability was a function of the substance abuse, antisocial behavior, or both.

parents or (2) the measure of antisocial behavior not being the same in the adoptee and either biological or adoptive parents. Twenty-three studies (33% of original pool) were rejected due to these criteria. An additional 13 studies (19%) were rejected because the studies did not include a measure that was specific to antisocial behavior (e.g., measure of general behavior problems) and an additional 6 studies (9%) were excluded because alcohol or drug abuse was taken as a sole indication of antisocial behavior in some cases. Nine studies (13%) were excluded because they reported analyses of a subset of a sample that was already included in our meta-analysis and four studies (6%) were excluded because they did not report sufficient information to calculate effect sizes.[5]

Twin studies employed samples ranging in size from 33 twin pairs to 975 pairs. Collectively, these studies involved 3795 twin pairs, with an average sample size of 211 twin pairs. Adoption studies employed samples ranging in size from 73 to 108 adoptees. These studies collectively involved 338 adoptees, with an average sample size of 85 adoptees. Therefore, the results of this meta-analysis are based on an effective sample size of 4133, which includes twin pairs and adoptees.

## Calculation of Effect Sizes

Our measure of effect size was the *d* statistic (Wolf, 1986). Calculation of *d* was designed (1) to have the effect size be an estimate of the degree of heritability in the antisocial measure and (2) to have analogous statistics for twin and adoption studies. For twin studies, *d* reflected the magnitude of the difference between the correlations (or concordance) of MZ and DZ twins on the antisocial measure. This procedure is analogous to the rationale behind Falconer's (1981) heritability coefficient ($h^2$), which assumes that greater similarities between MZ twins than DZ twins are due to the identical genetic makeup of MZ twins and, therefore, reflect greater heritability of the trait.[6] To calculate *d*, the correlations between MZ and

[5]A complete listing of the studies that formed the initial pool but were rejected due to our inclusionary criteria is available from the submitting author by request.

[6]There has been a long-standing debate over the use of the heritability coefficient as an estimate of genetic influences (see Plomin *et al.*, 1990). Many of the more recent behavioral genetic studies have used more sophisticated methods for testing the strength of genetic effects, such as by comparing the between- and within-pair variance in twin studies (e.g., Ghodsian-Carpey & Baker, 1987) or by using structural equation modeling to partition genetic effects in either twin or adoption studies (see Eaves *et al.*, 1993). It was apparent in our large initial study pool that there were too few studies currently available to conduct a meta-analysis using these more sophisticated measures of genetic effect. Therefore, we used the comparison between MZ and DZ twin concordance as a rough estimate of genetic effect because this was the only information available in the majority studies that would enable us

DZ twins were first converted to Fisher *z* scores and differences between the two coefficients (MZ–DZ) were used to obtain the *d* statistic (Wolf, 1986). Therefore, positive values of *d* reflected higher MZ than DZ concordance and negative *d* scores reflected lower MZ than DZ concordance. Calculation of *d* was necessary to combine results from twin and adoption studies. However, the absolute value of *d* is not directly translatable into the percentage of variance explained by heredity, and therefore, heritability coefficients ($h^2$) were calculated for all twin studies as well.

An analogous *d* statistic was calculated in adoption studies. To be included in the meta-analysis, a study had to provide some comparison between the correlation of adoptees' antisocial behavior with antisocial behavior in their biological parents and the correlation of an adoptees' behavior with antisocial behavior in the adoptive parents. The statistical test employed in this comparison was converted to *d* (Wolf, 1986), so that positive *d* values reflected greater correlations between adoptees and their biological parents antisocial behavior than between adoptees and their adoptive parents behavior. This approach leads to a somewhat conservative estimate of heritability, since the difference in correlations reflect the influence of genetics *relative* to the influence of rearing environment. However, we felt that this method provided an estimate of heredity that was most comparable to the estimate obtained from twin studies.

Multiple effect sizes were calculated within a single published study if *d* could be calculated for independent samples within the study (Wolf, 1986). For example, four studies reported separate concordance rates for males and females on the same measures of antisocial behavior. Therefore, separate effects sizes were calculated for the male sample and the female sample. However, to maintain the independence of effect sizes for statistical analysis, if the study reported multiple measures of antisocial behavior *within the same sample*, a composite measure was formed which was based on the average of each measure and this composite measure was converted to a single *d* statistic. In no case did this involve combining measures that differed on any of the moderator variables (e.g., type of antisocial behavior, severity of antisocial behavior).

## Moderator Variables

*Type of Antisocial Behavior.* Outcome measures of antisocial behavior were grouped into three categories: (1) aggression (verbal, physical, direct, and indirect), (2) criminal behavior (convictions), and (3) antisocial per-

to calculate an effect size estimate that was comparable across studies.

sonality. Eight measures of effect size were calculated from the six studies of aggression, all but one of these measures were obtained from twin studies. Nine measures of effect size were obtained from the six studies of antisocial personality, with only one of these measures coming from adoption samples. Three studies produced the four measures of effect size for the criminality variable, all but one measure coming from twin studies.

*Severity.* Severity of antisocial behavior was measured by placing the measure of antisocial behavior into a dichotomous category of "Severe" and "Nonsevere." Since all measures of criminality included actual convictions, all measures of criminality were considered Severe. Measures of aggression which included physical aggression to others and destruction of property or a mixture of the preceding two definitions were also considered Severe. A measure of antisocial personality was considered Severe if subjects had symptoms of or were diagnosed with antisocial personality disorder or with childhood conduct disorder. Nonsevere antisocial behaviors were measures of aggressive behavior that consisted of noncompliance, teasing others, or attacking an inanimate object. Also, measures of antisocial personality based on personality inventories were grouped in the Nonsevere category, unless only extreme scores were used.

*Demographic Variables.* Potential demographic moderators were the nationality, age, gender, and racial composition of the samples. Thirteen effect sizes (from 10 studies) used samples drawn from the United States. Four effect sizes were based on samples from Great Britain, three effect sizes were based on Danish samples, and one effect size was based on a Canadian sample.

Age of the sample on which the effect size was calculated was defined in several ways. Mean age of the sample was reported in 12 studies (16 effect sizes). The average age across all studies included in the meta-analysis was 18.7 years. However, only six studies (seven effect sizes) reported exact age ranges (ranging from 4 to 75 across all studies included). The one measure of age that could be computed for all studies was whether all subjects were younger than 18 years of age (10 effect sizes) or the subjects were all 18 years or older (9 effect sizes) or the subjects were mixed between children and adults (2 effect sizes).

Only one study (two measures of effect size) provided no information on the gender composition of the sample. Of the remaining 19 measures of effect size, 7 were obtained on all-male samples. Five measures of effect size were based upon all-female samples and the rest were based on mixed-sex samples. The racial composition of the sample was also coded, although only seven studies (10 measures of effect size), provided information on the racial composition of their samples.

## Methodological Variables

Several methodological variables were also measured as potential moderators. Use of blinding as a methodological variable was coded for each study. A study was considered to have "optimal" blinding if (1) the twin study ensured that the individual assessing antisocial behavior in one twin was blind both to antisocial behavior in the cotwin and to the zygosity of the twin or (2) the adoption study ensured that the individual assessing the adoptee for presence of a particular antisocial behavior was blind to the measure of antisocial behavior in both the adoptee's biological and the adoptee's adoptive parents. If such blinding procedures were clearly not conducted or insufficient information was given to determine the procedure, the study was considered in the "nonoptimal" blinding category.

The methods for determining zygosity in a twin studies was also included. A study was coded as using "Optimal" zygosity determination if blood typing or fingerprint comparisons on all twins in the sample were used to determine zygosity. "Nonoptimal" zygosity determination included blood typing for only a portion of the sample or making determinations based solely upon physical similarities. Three studies (five measures of effect size) were included in the optimal zygosity category.

Method of subject recruitment was divided into two categories, Clinic (convicted criminals or patients of a mental health facility) and Nonreferred (volunteers from a nonclinical population). Five studies (6 measures of effect size) used clinic samples and 10 studies (15 measures of effect size) employed volunteer samples.

## Coding Procedure

All effect sizes and moderator variables were coded independently by two raters (Wanous, Sullivan, & Malinak, 1989). The definitions of moderator variables were determined prior to the calculation of effect sizes and the actual coding of the moderator variables were always accomplished prior to the calculation effect size. Correlation coefficients between the codes of the two raters were generally quite high (mean $r$ = .87). All discrepancies were resolved through discussion between the two raters and the mutually agreed on resolution was the code used in all analyses.

## RESULTS

### Overall Effect Sizes

Table I lists each study and whether it was a twin or adoption study and by the type of antisocial behavior measured. It also provides the effect size and $h^2$ (twin studies only) for each study. In Table II, we present summary statistics of the effect sizes across all studies. The mean effect size across both twin and adoption studies was $d = .30$. Although this overall effect size was not statistically different from zero ($z = 1.4$, $p < .08$), this significance test is quite conservative given the limited sample size and subsequent limited power. As a guide based on effect sizes found in other areas of psychological research, an effect size of .10 is typically considered small, .30 as medium, and .50 as large (Cohen, 1977). Therefore, the overall effect of heredity would be considered a medium effect size.[7] Also, an average effect size may not be an appropriate summary statistic because of a skewed distribution of effect sizes. Over half ($n = 11$) of the effect sizes were medium to large (ranging from .45 to .68). In fact, the median effect size across studies was .45. The overall mean was dramatically reduced by two studies that produced negative effect sizes (Plomin *et al.,* 1981; Stevenson & Graham, 1988—girls only). To provide a frame a reference to these effect sizes, the heritability coefficients ($h^2$) for the twin studies were calculated and these results are also reported in Table II. The mean and median $h^2$ across twin studies was .48, which indicates that the percentage variance accounted for by genetics was estimated at 48%.

### Type and Severity of Antisocial Behavior

Table III presents the effect sizes by type of antisocial behavior. There were no significant differences among effect sizes obtained in studies measuring criminal behavior, antisocial personality, and aggression [$F(2,18) = .86$, $p$ = n.s.]. This remained true when measures of effect sizes were examined for twin studies alone [$F(2,15) = .93$, $p$ = n.s.].

Table III also presents the results of analyses comparing the effects sizes for measures of antisocial behavior divided by severity. The average effect size for studies that used measures of Severe antisocial behavior was significantly greater than studies that used measures of Nonsevere antiso-

[7]According to guidelines provided by Wolf (1986), all effect sizes were calculated twice: weighted by sample size ("unbiased") and unweighted ("biased") by sample size. However, only the unweighted effects sizes are reported because there was negligible differences in results using the weighted or unweighted statistics.

**Table I.** Summary of Effect Sizes Used in the Meta-Analysis

| Study | Sample size | Description of antisocial construct | Measurement technique | $H^2$ | $D$ |
|---|---|---|---|---|---|
| | | **Twin studies** | | | |
| | | Criminality measures | | | |
| Centerwall & Robinette, 1989 | 109 | Bad conduct and dishonorable discharge from U.S. military by court martial | Review of military records | .26 | .68 |
| Cloninger, Christiansen, Reich, & Gottesman, 1978 | 975 males | Minor offenses sanctioned by fines, felonies, and juvenile offenses after age 15 | Review of Danish police and penal registers | .67 | .47 |
| Cloninger *et al.*, 1978 | 938 females | Minor offenses sanctioned by fines, felonies, and juvenile offenses after age 15 | Review of Danish police and penal registers | .57 | .45 |
| | | Aggression measures | | | |
| O'Conner, Foch, Sherry, & Plomin, 1980 | 84 | Verbal and physical aggression; oppositional, noncompliant, and argumentative behaviors | Parent rating scale | .60 | .45 |
| Ghodsian-Carpey & Baker, 1987 | 38 | Aggression, lying, stealing, noncompliance, teasing, insulting others, and destruction of objects | Parent rating scale and behavioral observation | .77 | .54 |
| Rushton, Fulker, Neale, Nias, & Eysenck, 1986 | 136 males | Verbal and physical aggression, temper outbursts, and annoying others | Self-report questionnaire | .34 | .16 |
| Rushton *et al.*, 1986 | 339 females | Verbal and physical aggression, temper outbursts, and annoying others | Self-report questionnaire | .86 | .45 |
| Plomin, Foch, & Rowe, 1981 | 84 | Number and intensity of hits to a doll | Behavioral observation | .00 | −.38 |

**Table I.** Continued

| Study | Sample size | Description of antisocial construct | Measurement technique | $H^2$ | $D$ |
|---|---|---|---|---|---|
| Tellegen *et al.*, 1988 | 331 reared together | Pooled data from two measures: (1) Aggression Scale—Verbal and physical aggression, temper outbursts, and irritability & (2) Constraint scale—rejects conventional norms, sensation-seeking, and impulsive | Self-report questionnaire | .58 | .30 |
| Tellegen *et al.*, 1988 | 71 reared apart | Pooled data from two measures: (1) Aggression Scale—Verbal and physical aggression, temper outbursts, and irritability & (2) Constraint scale—rejects conventional norms, sensation-seeking, and impulsive | Self-report questionnaire | .80 | .41 |
| | | Antisocial personality | | | |
| Stevenson & Graham, 1988 | 94 males | Theft, lies, bullying, property destruction, and disobedience | Parent interview and parent rating scale | .42 | .48 |
| Stevenson & Graham, 1988 | 111 females | Theft, lies, bullying, property destruction, and disobedience | Parent interview and parent rating scale | .00 | −1.22 |
| Lytton, Watts, & Dunn, 1988 | 33 | Stealing, lying, vandalism, disobedience, and irritability | Teacher and parent rating scale | .39 | .52 |
| Dworkin, Burke, & Maher, 1976 | 42 under age 18 | Poor social conformity, problems with authority, sensation seeking, and unstable lifestyle | Self-report questionnaire | .40 | .23 |
| Dworkin, Burke, & Maher, 1976 | 42 over age 17 | Poor social conformity, problems with authority, sensation seeking, and unstable lifestyle | Self-report questionnaire | .32 | .21 |

| | | | | | |
|---|---|---|---|---|---|
| Baker & Daniels, 1990 | 104 | Unconventional lifestyle, defiance to authority, and impulsive behaviors | Self-report questionnaire | .84 | .46 |
| Rowe, 1983 | 166 females | Theft, fighting, vandalism, trespassing, lying about age, and causing a disturbance | Self-report questionnaire | .54 | .45 |
| Rowe, 1983 | 99 males | Theft, fighting, vandalism, trespassing, lying about age, and causing a disturbance | Self-report questionnaire | .36 | .26 |
| | | **Adoption studies** | | | |
| | | Criminality measures | | | |
| Hutchings & Mednick, 1975 | 108 | Convictions of felonies and misdemeanors | Review of Danish penal registry | | .38 |
| | | Aggression measures | | | |
| Mattes & Fink, 1990 | 41 | Discrete episodes of loss of control of aggressive impulses resulting in assault or destruction of property | Family interviews | | .67 |
| | | Antisocial personality | | | |
| Jarey & Stewart, 1985 | 74 | Symptoms of conduct disorder | Review of clinic records and parent/teacher rating scales | | .50 |

*Note:* $d$ = effect size, $h^2$ = average Falconer's (1981) heritability index.

**Table II.** Mean Overall Effect Sizes

| Effect size measure | *M*/Md (SD) | | |
|---|---|---|---|
| | All samples | Twin samples | Adoptee samples |
| *d* | .30/.45 (.41) ($n$ = 21) | .26/.48 (.43) ($n$ =18) | .52/.50 (.50) ($n$ = 3) |
| $h^2$ | — | .48/.48 (.26) ($n$ = 18) | — |

[a] *d* = effect size. $h^2$ = average Falconer's (1981) heritability index. Md = median.

cial behavior [$t(19) = 2.65$, $p < .01$]. Focusing on twin studies, significant effects of severity were also present [$t(16) = 2.24$, $p < .05$]. In fact the average effect size in the studies of Nonsevere antisocial behavior was 0. In the twin studies, the percentage of variance accounted for by genetic effects ($h^2$) averaged 58% for studies using measures of Severe measures of antisocial behavior and 34% for studies using Nonsevere measures of antisocial behavior.

**Table III.** Effect Sizes for Three Types of Antisocial Behavior and Groups Based on Severity

| | *M* (*SD*) *d* | |
|---|---|---|
| | Full sample | Twin only |
| | Type of antisocial behavior | |
| Criminality | .50 (.13) ($n$ = 4) | .53 (.13) ($n$ = 3) |
| Aggression | .33 (.32) ($n$ = 8) | .28 (.31) ($n$ = 7) |
| Antisocial personality | .18 (.54) ($n$ = 9) | .17 (.56) ($n$ = 8) |
| | $F(2,18) = .86$ ($p$ = ns) | $F(2,15) = 2.40$ ($p$ = ns) |
| | Severity of antisocial behavior | |
| Severe | .45 (.14) ($n$ = 14) | .42 (.14) ($n$ = 11) |
| Nonsevere | .00 (.61) ($n$ = 7) | .00 (.61) ($n$ = 7) |
| | $t(19) = 2.65$ $p < .01$ | $t(16) = 2.24$ $p < .05$ |

[a] *d*= effect size. *M* = mean.

## Demographic Moderators

Correlation coefficients were obtained for mean age of the samples and effect sizes. Age was not correlated with effect size in the full sample ($r = .22, p$ = n.s.), nor was it correlated with effect size ($r = .21, p$ = n.s.) in the twin studies alone. A comparison of the effect sizes from adult samples (>17 years) and child/adolescent samples is presented in Table IV. Three effect sizes were excluded from analysis because they came from mixed samples of adults and children. The effect sizes did not differ significantly between adult and child samples in either the full sample [$t(17) = 1.2, p$ = n.s.] or the twin sample [$t(15) = 1.4, p$ = n.s.], although the mean effect size was medium to large in the adult samples and small in the child samples.

Although not statistically significant, the pattern of results was contrary to predictions in that the younger sample exhibited lower mean effect sizes. These results seemed to be a function of the fact that 5 of the 11 effect sizes in child samples used Nonsevere measures of antisocial behavior. In contrast, only two of nine of the adult effect sizes were based on Nonsevere measures of antisocial behavior. Across the child samples, the

**Table IV.** Effect Size and Demographic Variables

| | Age, *M*(*SD*) | | | |
|---|---|---|---|---|
| | <18 yr | | >18 yr | *t* test |
| *d* full sample | .15 (.55) ($n = 10$) | | .41 (.15) ($n = 9$) | $t(17) = 1.2$ $p$ = n.s. |
| *d* twin only | .11 (.57) ($n = 9$) | | .41 (.16) ($n = 8$) | $t(15) = 1.6$ $p$ = n.s. |
| | Gender, *M* (*SD*) | | | |
| | All male | All female | Mixed | ANOVA |
| *d* full sample | .38 (.17) ($n = 7$) | .03 (.83) ($n = 4$) | .34 (.29) ($n = 10$) | $F(2,18) = 1.03$ $p$ = n.s. |
| *d* twin only | .37 (.19) ($n = 6$) | .03 (.84) ($n = 4$) | .25 (.30) ($n = 7$) | $F(2,14) = .72$ $p$ = n.s. |
| | Country of sample, *M*(*SD*) | | | |
| | United States | | Non-United States | *t* test |
| *d* full sample | .37 (.27) ($n = 13$) | | .17 (.57) ($n = 8$) | $t(19) = 1.1$ $p$ = n.s. |
| *d* twin only | .33 (.27) ($n = 11$) | | .14 (.61) ($n = 7$) | $t(16) = .88$ $p$ = n.s., |

[a] *d* = effect size.

mean effect size of studies using Severe measures ($n$ = 6) was .45, whereas the mean effect size of studies using Nonsevere measures ($n$ = 5) was –.13. In the adult samples, the mean effect size of studies using Severe measures ($n$ = 7) was .43, whereas the mean effect size for studies using Nonsevere measures ($n$ = 2) was .34. Therefore, for Severe measures alone there was no difference in the effect sizes between child and adult samples, with both estimates being in the medium to large range.

Neither the gender (percentage male) nor the racial (percentage European–Caucasian) composition of the sample was significantly associated with effect size either in the full sample (gender = .32, racial $r$ = –.03) or in the twin samples (gender $r$ = .28, racial $r$ = –.04, respectively). However, the number of measures of effect size for which the racial composition of the samples was reported was quite small (8 of 22). A comparison of the mean effect sizes from all-male samples, all-female samples, and mixed-sex samples is provided in Table IV. There was no significant effect for gender in these analyses, although the effect sizes in the all females samples ($n$ = 4) tended to be somewhat lower. We were concerned that, like age, there may have been a confound between gender and severity which could have influenced the results. Unlike age, however, the gender composition of the sample was not confounded with severity of antisocial behavior. There were comparable proportions of effect sizes in the male, female, and mixed samples based on severe measures of antisocial behavior (71, 75, and 70%, respectively). Also reported in Table IV are the results comparing the effect sizes from samples in the United Stats to samples from other countries. There were no differences in effect sizes based on the nationality of the sample.

## Methodological Variables

Sample size was not significantly correlated with effect size across all studies ($r$ = .13) or in twin studies alone ($r$ = .18). However, effect sizes from twin studies were higher for those using optimal blinding techniques ($n$ = 3) than from studies that did not use optimal blinding ($n$ = 19) [$t(16) = 2.4, p < .05$]. These results are summarized in Table V. There was also a significant effect for the type of sample recruitment, with Clinic samples showing larger effect sizes across all studies [$t(19) = 2.56, p < .05$] and in twin studies only [$t(20) = 2.4, p < .05$]. However, there were no differences in mean effect sizes based on the method for determining zygosity.

**Table V.** Effect Size and Methodological Variables

| | Blinding, *M*(*SD*) | | |
|---|---|---|---|
| | Optimal | Nonoptimal | *t* test |
| *d* full sample | .53 (.13) ($n$ = 3) | .25 (.43) ($n$ = 18) | $t(19) = 1.10$ $p$ = n.s |
| *d* twin only | .53 (.13) ($n$ = 3) | .20 (.45) ($n$ = 15) | $t(16) = 2.4$ $p < .05$ |
| | Sample recruitment, *M*(*SD*) | | |
| | Clinic | Volunteer | *t* test |
| *d* full sample | .53 (.12) ($n$ = 6) | .20 (.45) ($n$ = 15) | $t(19) = 2.56$ $p < .05$ |
| *d* twin only | .53 (.13) ($n$ = 3) | .20 (.45) ($n$ = 15) | $t(16) = 2.41$ $p < .05$ |
| | Zygosity determination, *M* (*SD*) | | |
| | Optimal | Nonoptimal | *t* test |
| *d* twin only | .30 (.38) ($n$ = 5) | .24 (.46) ($n$ = 13) | $t(16) = .24$ $p$ = n.s. |

[a]*d* = effect size.

## DISCUSSION

One of the more interesting findings of this meta-analytic review was that the appearance of a large literature on the heritability of antisocial behavior is quite deceptive. Although an initial screening yielded 70 published behavioral genetic studies between 1975 and 1991, only 15 studies were judged suitable for this review. We do not feel that our criteria for inclusion was too restrictive. The majority of the studies rejected was adoption studies. The rejection of these studies was because either they did not study both the biological and the adoptive parents of the adoptees or the same construct was not measured in adoptees and their adoptive or biological parents (e.g., Bohman, Cloninger, Sigvardsson, & von Knorring, 1987; Crowe, 1975). The latter methodology could be justifiable if different measures in parents and children were based on an explicit theory of age-dependent differences in the phenotypic expression of an antisocial trait. However, this was not the case in the studies excluded from analysis, and in fact, most of the studies were excluded because of different measures used to assess adoptive and biological parents. Therefore, without confidence that the cross generational comparisons were measuring the same trait in adoptive and biological parents or in parents and children, we did not feel that the effect size would provide an interpretable estimate of the

degree of hereditary influence. This methodology not only placed limitations on the overall number of effect sizes available for analysis, but it resulted in the meta-analysis being predominantly based on twin studies. Therefore, interpretation of results should be made in the context of the issues inherent in the twin methodology for studying genetic influences (Plomin *et al.,* 1991).

The next most common reason for a study being excluded from analysis was because antisocial behavior was not measured in isolation from other types of psychological dysfunction (e.g., Graham & Stevenson, 1985; Maurer, Cadoret, & Cain, 1980). Therefore, it would be impossible to obtain an effect size for antisocial behavior that was separate from other types of psychological dysfunction. Additionally, many studies published results using overlapping samples (e.g., Hutchings & Mednick, 1975; Mednick, Brennan, & Kandel, 1988; Mednick, Gabrielli, & Hutchings, 1984; Mednick, Moffit, Gabrielli, & Hutchings, 1984). This common practice of using overlapping samples led to the appearance of a much larger literature than that which actually exists.

Therefore, any conclusion on the heritability of antisocial behavior must be made in the context of a modest sized literature with numerous methodological problems. Even within the studies included in this meta-analysis, the issue of methodological rigor was important. Only two studies (Centerwall & Robinette, 1989; Cloninger *et al.,* 1978) reported using methods of assuring blind evaluations within twin pairs or in the assessment of biological and adoptive parents. This is unfortunate because these two studies produced significantly larger effects of heredity than studies that failed to use such methodology. Clearly this review points to the continuing need for well-designed behavioral genetic studies of antisocial behavior (Mednick & Kandel, 1988). Another point related to methodological rigor that is important in interpreting these data is the fact that our effect sizes were based on very gross indices of heredity and did not include some of the more recent advances in behavioral genetic research which allow one to test mechanisms of genetic influence (e.g., Rutter *et al.,* 1990; also, see footnote 6).

Within the constraints of these limitations, the findings of this quantitative review support the conclusions reached in many qualitative reviews (e.g., Dilalla & Gottesman, 1991; Mednick & Kandel, 1988; Plomin *et al.,* 1991; Rutter *et al.,* 1990) that heredity plays a significant role in the development of antisocial behavior. As stated in the Introduction, it would be ludicrous to assert that heredity could be the sole cause in the development of any psychological syndrome, including antisocial behavior. However, the overall effect size was moderate to large in comparison with other effect sizes found in psychological research. Across twin studies, an average

of approximately 50% of the variance in measures of antisocial behavior was accounted for by heredity. This estimate of genetic effects is much higher than that found in another recent meta-analysis on hereditary influences to criminality (Walters, 1992). However, the differences in findings are likely due to major differences in methodology (see footnote 3) between our meta-analysis and the one conducted by Walters (1992).

Our results further suggested that this overall effect size must be interpreted cautiously because of several influential moderators. Probably the strongest moderating influence to the effect size was the severity of antisocial behavior. Our results suggest that when severe manifestations of antisocial behavior are studied, there tends to be stronger genetic effects. In fact, the mean effect size for studies using less severe measures of antisocial behavior was zero, indicating that on the average, the influence of heredity was negligible in these studies. Severity likely plays a role in the second significant moderator found in this meta-analysis. Studies of clinic populations tended to show larger effects of heredity than did volunteer samples. Intuitively, one would expect that clinic populations were more likely to have individuals that show more extreme and maladaptive patterns of antisocial behavior.

The importance of severity is exemplified by the twin study of Ghodsian-Carpey and Baker (1987), which tested the relationship between severity and degree of genetic influence explicitly. In their study of 38 twin pairs between 4 and 7 years of age, biometrical genetic analysis revealed much lower heritability estimates when a measure of less severe aggression (e.g., negativism, noncompliance, teasing, yelling) was studied than when a measure of more severe antisocial behavior (e.g., cruelty to animals, firesetting, destroying others things) was studied.

These findings have intriguing theoretical implications. It is generally accepted that antisocial behavior is a heterogeneous category that likely has multiple causal trajectories (see Lahey, Loeber, Quay, Frick, & Grimm, 1992). One interpretation of these findings is that more severe antisocial behavior, which seems to have a unique developmental trajectory (Lahey *et al.,* 1992), may also have unique causal factors that are more strongly influenced by heredity. Alternatively, it is also possible that the association between antisocial behavior and genetic influences is continuous but studies using measures of severe antisocial behavior provide a more accurate measurement of the behavioral dimension. For example, one could argue that hits to a toy Bobo doll (Plomin, DeFries, & McClearn, 1981) and other of the less severe measures of "antisocial behavior" were not even measuring the same construct as studies employing measures of more severe behavior. Clearly, it will be important for future behavioral genetic studies to more explicitly test possible differences in the strength of genetic effects using

definitions that take into account the severity of antisocial behavior. It is especially important to test alternative indices of severity (e.g., violent/nonviolent, chronic/transient) to further clarify these results.

There were no significant differences in effect sizes across type of antisocial behavior (i.e., criminality, aggression, and antisocial personality). Therefore, it appears that these divisions are not as important in terms of the relative contribution of genetic effects, as are differences in severity, regardless of definition. Also, age, gender, and racial composition of the sample did not significantly moderate effect sizes, nor did the country from which the sample was drawn. The failure to find effects for age was somewhat surprising given consistent findings that earlier age-of-onset marks an especially severe and chronic pattern of antisocial behavior (Loeber, 1991; Robins, 1966). In fact, although not statistically significant, the correlations with age were in the direction of *older* samples showing greater heritability, which is consistent with past qualitative reviews of the literature (Plomin *et al.,* 1990).

There are several possible explanations for the positive correlation between age and degree of genetic contribution. First, the child samples were more likely to use less severe measures of antisocial behavior than were the adult samples. In fact, when the five effect sizes from child samples that used Nonsevere measures of antisocial behavior were eliminated, the effect size (.45) in child samples was indistinguishable from the mean effect size from adult samples using severe measures (.43). Second, the "child" samples included both child and adolescent samples up to the age of 18. This is crucial because the studies on "early-onset" antisocial behavior typically defined early onset as *prior to age 11* (e.g., Robins, 1966). Therefore, we could not test the possibility that the genetic contribution differed between preadolescent onset and adolescent onset antisocial behavior (i.e., there was a nonlinear age trend).

In summary, our review suggests that conclusions based on the behavioral genetic research on antisocial behavior must be made cautiously due to the deceptively small literature and due to the numerous methodological limitations in the existing studies. This is an unfortunate limitation because the more methodologically sound studies tended to have larger effect sizes. Therefore, it is possible that our findings, which support the contention that there is an inherited predisposition to antisocial behavior, might even be an underestimate of the magnitude of the effects of heredity. In addition, our overall finding must be qualified by the moderating effects severity. Significant effects of heredity were confined to the more severe manifestations of antisocial behavior. These results suggest that future behavioral genetic studies should employ more precise definitions of antisocial

behavior, especially ones that distinguish between more and less severe patterns of behavior.

## APPENDIX

### Studies Included in the Meta-Analysis

Baker, L. A., & Daniels, D. (1990). Nonshared environmental influences and personality differences in adult twins. *Journal of Personality and Social Psychology, 58*, 103, 110.

Centerwall, B. S., & Robinette, C. D. (1989). Twin concordance for dishonorable discharge from the military: With a review of the genetics of antisocial behavior. *Comprehensive Psychiatry, 30*, 442-446.

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