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Genetic and Environmental Influences on Antisocial Behavior: A Meta-Analysis of Twin and Adoption Studies

Soo Hyun Rhee and Irwin D. Waldman
Emory University

A meta-analysis of 51 twin and adoption studies was conducted to estimate the magnitude of genetic and environmental influences on antisocial behavior. The best fitting model included moderate proportions of variance due to additive genetic influences (.32), nonadditive genetic influences (.09), shared environmental influences (.16), and nonshared environmental influences (.43). The magnitude of familial influences (i.e., both genetic and shared environmental influences) was lower in parent–offspring adoption studies than in both twin studies and sibling adoption studies. Operationalization, assessment method, zygosity determination method, and age were significant moderators of the magnitude of genetic and environmental influences on antisocial behavior, but there were no significant differences in the magnitude of genetic and environmental influences for males and females.

Considerable research has focused on the goal of explaining the etiology of antisocial behavior. In particular, the role of familial influences on antisocial behavior has been studied extensively. Dysfunctional familial influences such as psychopathology in the parents (e.g., Robins, 1966), coercive parenting styles (e.g., Patterson, Reid, & Dishion, 1992), physical abuse (Dodge, Bates, & Pettit, 1990), and family conflict (e.g., Norland, Shover, Thornton, & James, 1979) have been shown to be significantly related to antisocial behavior. Often, these variables are considered environmental influences, and the possibility that they may also reflect genetic influences is not considered. This is unfortunate because disentangling the influences of nature and nurture is the first step toward the goal of eventually explaining the etiology of antisocial behavior. Also, estimating the relative magnitude of genetic and environmental influences on antisocial behavior is an important step toward the search for specific candidate genes and environmental risk factors underlying antisocial behavior. Although it is not possible to disentangle genetic from environmental influences in family studies because genetic and environmental influences are confounded in nuclear families, twin and adoption studies have the unique ability to disentangle genetic and environmental influences and to estimate the magnitude of both simultaneously.

More than a hundred twin and adoption studies of antisocial behavior have been published. Nonetheless, it is difficult to draw clear conclusions regarding the magnitude of genetic and environmental influences on antisocial behavior given the current literature. The main reason for this difficulty is the considerable heterogeneity of the results in this area of research, with published heritability estimates (i.e., the magnitude of genetic influences) ranging from very low (e.g., .00; Plomin, Foch, & Rowe, 1981) to very high (e.g., .71; Slutske, Heath, et al., 1997). Various hypotheses have been proposed to explain these heterogeneous results across studies, including differences in the age of the sample (e.g., Cloninger & Gottesman, 1987), the age of onset of antisocial behavior (e.g., Moffitt, 1993), and the measurement of antisocial behavior (e.g., Plomin, Nitz, & Rowe, 1990).

We conducted a meta-analysis of twin and adoption studies in order to provide a clearer and more comprehensive picture of the magnitude of genetic and environmental influences on antisocial behavior. Given previous hypotheses proposed to explain the heterogeneity in the results, we examined the possible moderating effects of three study characteristics (i.e., the operationalization of antisocial behavior, assessment method, and zygosity determination method) and two participant characteristics (i.e., the age and sex of the participants) on the magnitude of genetic and environmental influences on antisocial behavior. We examined the operationalization of antisocial behavior given the evidence that antisocial personality disorder (ASPD), conduct disorder (CD), criminality, and aggression are distinct but related constructs (e.g., Robins & Regier, 1991). We examined assessment method and zygosity determination because of evidence suggesting that these are potential methodological confounders (e.g., McCartney, Harris, & Bernieri, 1990; Plomin, 1981). Sex was examined given the consistent evidence that antisocial behavior is more prevalent in males than females (e.g., Hyde, 1984; J. Q. Wilson & Herrnstein, 1985). Age was examined because of the potential to test an interesting hypothesis regarding the development of antisocial

Soo Hyun Rhee and Irwin D. Waldman, Department of Psychology, Emory University.

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Correspondence concerning this article should be addressed to Soo Hyun Rhee, who is now at the Institute for Behavioral Genetics, University of Colorado at Boulder, Campus Box 447, Boulder, Colorado 80309. E-mail: soo.rhee@colorado.edu

behavior. L. F. DiLalla and Gottesman (1989) and Moffitt (1993) have suggested that individuals who engage in antisocial behavior can be divided into a smaller group whose antisocial behavior is persistent throughout the life course and influenced predominantly by genetics and a larger group whose antisocial behavior is limited to adolescence and influenced predominantly by environment. If their hypothesis is correct, the magnitude of genetic influences on antisocial behavior should be lower in adolescence than in childhood or adulthood.

Previous Reviews Examining Behavior Genetic Studies of Antisocial Behavior

A number of traditional literature reviews (e.g., Carey, 1994; Gottesman & Goldsmith, 1994; Plomin et al., 1990) of twin and adoption studies of antisocial behavior have been published, and most researchers in this area have concluded that both genetic and environmental influences are important contributors to individual differences in antisocial behavior. Although these reviews are informative, they did not provide a quantitative estimate of the genetic and environmental influences on individual differences in antisocial behavior across studies. Three previous meta-analyses have provided such an estimate. Walters (1992) examined 11 family studies, 14 twin studies, and 13 adoption studies of criminality and found genetic influences on crime that were low to moderate in magnitude (i.e., a mean unweighted phi coefficient of .25 and a mean weighted phi coefficient of .09). Mason and Frick (1994) examined 12 twin studies and 3 adoption studies of antisocial behavior and attributed approximately 50% of the variance in measures of antisocial behavior to genetic influences. They examined several moderating variables and found that effect sizes did not vary across the type of antisocial behavior (i.e., criminality, aggression, or antisocial personality), demographic variables (i.e., sex, age, and racial composition), and two methodological variables (i.e., sample size and zygosity determination), but they found larger estimates of genetic influences for severe antisocial behavior, antisocial behavior in clinic-referred samples, and studies with *optimal blinding* (i.e., assessment of antisocial behavior that is blind to the relatives' level of antisocial behavior). Miles and Carey (1997) examined 20 twin studies and 4 adoption studies of aggression and concluded that genetic influences account for up to 50% of the variance. They also tested several potential moderators of genetic and environmental influences on aggression, including sex, age, and assessment method. The heritability estimate for males was higher than that for females, and the heritability estimate for younger samples was lower than that for older samples. Studies using parent reports yielded a lower heritability estimate and a higher estimate for the magnitude of shared environmental influences than those using self-reports.

Walters's (1992) and Mason and Frick's (1994) meta-analyses have some methodological problems that make the interpretation of their results difficult. Mason and Frick (1994) provided a detailed description of the methodological problems (e.g., inclusion of nonindependent samples) in Walters's meta-analysis. A serious concern about Mason and Frick's meta-analysis is the effect size they chose to report. They used an effect size of *d* for both adoption studies and twin studies, subtracting the dizygotic (DZ) correlation from the monozygotic (MZ) correlation in twin studies and subtracting the adoptee–adoptive parent correlation from the adoptee–biological parent correlation in adoption studies. This effect size, *d,* is not appropriate because the difference between the MZ correlation and the DZ correlation is not comparable to the difference between the adoptee–biological parent correlation and the adoptee–adoptive parent correlation. Heritability is estimated in twin studies by doubling the difference between the MZ correlation and the DZ correlation, whereas heritability is estimated in adoption studies by doubling only the adoptee–biological parent correlation. Another methodological problem in Mason and Frick's study is that their effect size, *d,* included the difference between the concordances of MZ and DZ twins as well as the difference between the correlations of MZ and DZ twins. Concordances vary according to the base rate, such that the same concordances with different base rates are associated with different correlations (A. Heath, personal communication, March 1994).

There are several important differences between the present meta-analysis and the previous three meta-analyses. First, the present study is more comprehensive, examining 10 independent adoption samples and 42 independent twin samples from 51 studies (two separate samples were examined in Eley, Lichtenstein, & Stevenson, 1999). Second, we adopted a broader conceptualization of antisocial behavior, examining relevant diagnoses, criminality, aggression, and antisocial behavior (i.e., a composite index of delinquency and aggression). Third, as in Mason and Frick (1994), nonindependent samples were not treated as independent. Fourth, as in Miles and Carey (1997), direct analysis of the data was conducted. Fifth, more potential moderators were examined, including operationalization, assessment method, zygosity determination method, sex, and age. Sixth, the present meta-analysis entailed a direct comparison between the results of twin and adoption studies. Seventh, the present meta-analysis also addresses several issues that could not be examined quantitatively in the meta-analysis because not enough studies in the literature examined them. These issues include the role of genotype–environment interaction on antisocial behavior, longitudinal studies of antisocial behavior, and specific environmental influences on antisocial behavior.

Operationalization as a Moderator

The operationalizations of antisocial behavior can be divided into three major categories (Plomin et al., 1990). First, antisocial behavior has been examined in terms of psychiatric diagnoses, such as ASPD and CD. Second, antisocial behavior has been operationalized in terms of the violation of legal or social norms, that is, as criminality and delinquency. Third, antisocial behavior has been operationalized as aggressive behavior.

The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV;* American Psychiatric Association, 1994) described the essential features of ASPD as "a pervasive pattern of disregard for, and violation of, the rights of others that begins in childhood or early adolescence and continues into adulthood" (p. 645). A diagnosis of ASPD requires a history of CD before the age of 15 and three or more of the following criteria: failure to conform to social norms with respect to lawful behaviors (i.e., as indicated by repeatedly performing acts that are grounds for arrest), deceitfulness, impulsivity, irritability and aggressiveness, reckless disregard for safety, consistent irresponsibility, and lack of remorse. CD, a criterion for the diagnosis of ASPD, is described by the

DSM–IV as "a repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated" (American Psychiatric Association, 1994, p. 90). It usually occurs in childhood or early adolescence and is manifested as aggression toward people and animals, destruction of property, deceitfulness or theft, and serious violations of rules.

Criminality has been defined as an unlawful act that leads to arrest, conviction, or incarceration, whereas *delinquency* has been defined as unlawful acts committed as a juvenile. In addition to official records, past researchers also have assessed delinquency with anonymous self-reports of criminal activity that has not led to arrest, conviction, or incarceration. Aggression is usually studied as a personality characteristic and assessed with measures such as the Adjective Check List (Gough & Heilbrun, 1972) and the Multidimensional Personality Questionnaire (Tellegen, 1982, as cited in Tellegen et al., 1988). The operationalization of aggression has been very heterogeneous in the past, ranging from negative affect (Partanen, Bruun, & Markkanen, 1966) to the number of hits to a Bobo doll (Plomin et al., 1981). For the present review, the operationalization of aggression was restricted to the type of behavioral aggression described in the *DSM* criteria for CD (e.g., bullying, initiating physical fights, and using a weapon that can cause serious physical harm).

In deciding which studies to include in the present review, an important question had to be considered. Do diagnoses, criminality–delinquency, and aggression reflect the same construct? It is clear that the three operationalizations are related. CD and criminality are criteria for ASPD, whereas aggression and delinquency are criteria for CD. Past research shows moderate correlations between self-report measures of aggression and ASPD in a sample of individuals engaging in substance abuse (Mutaner et al., 1990) and a significant relation between criminality and ASPD in a sample of individuals engaging in criminal activity (e.g., number of prior arrests was significantly related to presence of antisocial disorder; Abram, 1989). In addition, childhood aggression was found to predict adult criminality (e.g., boys rated by peers and teachers as highly aggressive at age 8 had more than five arrests on average at age 26 compared with less than two arrests in comparison boys; Pulkkinen & Pitkänen, 1993).

On the other hand, the three operationalizations of antisocial behavior are not synonymous. The Epidemiologic Catchment Area study led by Robins and Regier (1991) reports that only 27% of boys and 21% of girls with three or more CD symptoms will be diagnosed with ASPD in adulthood, whereas 49% of boys and 33% of girls with six or more CD symptoms will be diagnosed with ASPD. Delinquency before age 15 predicted later ASPD in 29% of males and 13% of females. Also, whereas 40% of male criminals and 18% of female criminals qualify for an ASPD diagnosis, 55% of males with ASPD and 17% of females with ASPD are criminals.

Although there are no definitive conclusions regarding the relations among the diagnoses of ASPD and CD, criminality–delinquency, and aggression other than that they are moderately overlapping constructs, studies examining all three operationalizations of antisocial behavior were included in the present review for the following reasons. First, past reviews have focused on only one operationalization (e.g., aggression in Miles & Carey, 1997) or reviewed the results of studies using different operationalizations separately (e.g., Plomin et al., 1990). This is understandable, as conclusive evidence showing that the different operationalizations reflect the same construct is lacking, and the magnitude of genetic and environmental influences on antisocial behavior may differ across operationalizations (Plomin et al., 1990). Thus, in the present meta-analysis, studies examining all three operationalizations were included to conduct a quantitative test of this issue. Second, age was examined as a possible moderator to examine potential developmental shifts in the relative magnitudes of genetic and environmental influences on antisocial behavior. In order to do so, studies using different operationalizations of antisocial behavior had to be included because antisocial behavior is expressed differently by children and adults and therefore is defined differently for them. Third, adopting broader inclusion criteria increases the power of the meta-analysis, given that it is based on a greater number of studies.

In addition to clinical diagnoses, criminality, and aggression, "antisocial behavior," an omnibus operationalization that includes aggression and delinquency items, was examined. Some researchers (e.g., Rowe, 1983) conducted twin studies of delinquency, although the measures used in these studies also included aggression items. Also, some studies used measures including both aggression and delinquency items, such as the externalizing scale from the Child Behavior Checklist (Achenbach & Edelbrock, 1983). In addition to the general moderator analyses of operationalization, we examined possible differences between ASPD and CD in a more focal analysis.

Assessment Method as a Moderator

Researchers have shown that assessment method can influence the results of behavior genetic studies. For example, McCartney et al. (1990) compared parent and self-reports of sociability and found that parent reports resulted in higher correlations than self-reports in MZ twins but resulted in lower correlations than self-reports in DZ twins. They also found that for activity–impulsivity, parent reports resulted in higher correlations than self-reports in both MZ and DZ twins. In contrast, Miles and Carey (1997) found that behavior genetic studies of aggression using parent reports resulted in a lower heritability estimate when compared with those using self-reports.

Researchers studying temperament have found that parent reports tend to yield DZ correlations that are very low or even negative. This may be the result of parents' exaggerating the differences between their DZ twins, which has been described as a *rater contrast effect* (Loehlin, 1992a). One example of such a finding emerged from the MacArthur longitudinal twin study (Emde et al., 1992). No resemblance of DZ twins on measures of behavioral inhibition and shyness was found using parent reports, but significant DZ resemblance was found using observational measures of the same constructs. Plomin's (1981) review of twin studies examining personality concluded that objectively assessed behavior yielded lower heritabilities than self-reports and parent reports. Similarly, Miles and Carey's (1997) meta-analysis of behavior genetic studies of aggression concluded that two studies using an objective method found little evidence of genetic influences on aggression, in contrast to studies using self-report or parent report.

In addition to self-report, report by others (i.e., parent and teacher report), and objective measures, antisocial behavior has been assessed by two other methods. Criminality has been assessed with official records, and aggression has been assessed by examining reactions to aggressive material (e.g., whether one finds aggressive humor to be funny or not; G. D. Wilson, Rust, & Kasriel, 1977). In the present review, assessment method was examined as a moderator, comparing self-report, report by others (i.e., parent and teacher report), official records, objective measures, and reactions to aggressive material.

Zygosity Determination Method as a Moderator

Zygosity determination method was examined as a possible moderator of genetic and environmental influences on individual differences in antisocial behavior. Zygosity determination methods used in twin studies of antisocial behavior include blood grouping, questionnaires, and a combination of the two methods. The inaccuracy of blood grouping in determining the zygosity of twin pairs is less than 1% (e.g., Smith & Penrose, 1955). Questionnaire methods of determining zygosity, which involve asking about the physical similarity of the twin pairs, have been found to agree highly with zygosity diagnosis by blood grouping. For example, Kasriel and Eaves (1976) found that if all twin pairs who agree that they were confused in childhood and are alike in appearance are determined to be MZ, only 3.9% of the sample would be diagnosed incorrectly. Nevertheless, estimates of the magnitude of genetic and environmental influences may be affected by the zygosity determination method. McCartney et al. (1990) predicted that studies that used blood grouping would have higher effect sizes for MZ twins and lower effect sizes for DZ twins because use of blood grouping in zygosity determination would purify the MZ and DZ samples. They found that studies using blood grouping did have higher effect sizes for MZ twins, but that the zygosity determination method did not moderate effect sizes for DZ twins. In the present review, studies using blood groupings, questionnaires, and a combination of the two methods (i.e., studies using the questionnaire method for the whole sample and the blood grouping method for a subset of the sample) are compared.

Age as a Moderator

It is important to investigate age as a possible moderator of genetic and environmental influences on human behavior in general and on antisocial behavior in particular. In the behavior genetics literature, there is a general finding for a variety of traits that as age increases, the magnitude of genetic and nonshared environmental influences increases, whereas the magnitude of shared environmental influences decreases (Loehlin, 1992a; Plomin, 1986). One example of such a finding is Matheny's (1989) longitudinal study of temperament. Over 12 to 30 months of age, MZ twins became more concordant than DZ twins for age-to-age changes in temperament measures of emotional tone, fearfulness, and approach.

McCartney et al. (1990) conducted a meta-analysis of developmental changes in genetic and environmental influences on intelligence and several personality variables. They reported correlations between the components of variance (i.e., heritability, shared environment, and nonshared environment) and age for the three variables examined in the most studies (i.e., intelligence, sociability, and activity–impulsivity). In general, the correlations for both MZ and DZ twin pairs decreased as age increased, and this finding also applied to the eight studies examining aggression. The results were inconsistent, and the authors cautioned that they may not be reliable because they are based on few data points. Also, it should be noted that researchers conducting another meta-analysis that examined genetic and environmental influences on intelligence (Devlin, Daniels, & Roeder, 1997) concluded that an age-effects model, which allowed the heritability of IQ to increase with age, failed to fit the data better than a simpler model. In Miles and Carey's (1997) meta-analysis of behavior genetic studies examining aggression, the magnitude of shared environmental influences decreased and the magnitude of genetic influences increased from childhood to adulthood.

There appears to be conflicting evidence regarding age as a moderator of genetic and environmental influences on criminality. In five early twin studies examining juvenile delinquency, the weighted average of concordance rates for MZ and DZ twins was .87 and .72, respectively (Cloninger & Gottesman, 1987). In comparison, in seven early twin studies examining adult criminality, the weighted average of concordance rates for MZ and DZ twins was .51 and .23, respectively (Cloninger & Gottesman, 1987). These results suggest that juvenile delinquency during adolescence, unlike criminality during adulthood, is only moderately affected by genetic influences but is very strongly affected by shared environmental influences. Given these results, researchers have theorized that genetic influences on individual differences in delinquency may be minimal because the base rate for delinquency is very high (L. F. DiLalla & Gottesman, 1989) or because environmental influences such as peer pressure are particularly strong in adolescence (Raine & Venables, 1992). Pertinent to these hypotheses, Lyons et al. (1995) assessed juvenile and adult ASPD symptoms in the same participants using retrospective self-report. They found that the heritability for the adult antisocial traits (h^2 = .43) was higher than that for the juvenile antisocial traits (h^2 = .07), supporting Cloninger and Gottesman's conclusions.

In contrast, Rowe (1983) examined anonymous self-reports of delinquent acts and found that both genetic and environmental influences are substantial for juvenile delinquency. Some reviewers have attributed Rowe's contradictory finding to his use of self-report and have suggested that the finding of genetic influences is reflecting the response to questionnaires rather than the construct of juvenile delinquency (e.g., L. F. DiLalla & Gottesman, 1991). They have also noted that the finding of genetic influences may be a function of including items that assess aggression rather than delinquency. Other limitations of this study include a low response rate, which raises issues regarding sampling biases, and the use of a mailed questionnaire, which raises the possibility of nonindependent responses. On the other hand, Rowe and Rodgers (1989) asserted that it is premature to conclude that genetic influences are not important for delinquency, as the early twin studies had many methodological problems (e.g., haphazard sampling, small sample size, and variance in zygosity determination method). Carey (1994) admitted that the methodology of the early twin studies was generally poor but noted that similar methodological problems did not prevent finding genetic influences on adult criminality.

It is not possible to conclude from the information provided by the traditional literature reviews (e.g., Cloninger & Gottesman, 1987; L. F. DiLalla & Gottesman, 1989) whether age is an important moderator of genetic and environmental influences on antisocial behavior or criminality. In the present meta-analysis, we used participants' age as a moderator in order to examine this issue, comparing results for children (below age 13), adolescents (ages 13–18), and adults (above age 18).

The significance of age of onset and the continuity of antisocial behavior is discussed in several traditional literature reviews (e.g., Cloninger & Reich, 1983; L. F. DiLalla & Gottesman, 1989; Gottesman & Goldsmith, 1994). In particular, L. F. DiLalla and Gottesman (1989) hypothesized that there are three different types of offenders: continuous antisocials (i.e., those are who are delinquent as youths and continue to be criminal as adults), transitory delinquents (i.e., youths who are delinquent but not criminal as adults), and late bloomers (i.e., adults who are criminal but were not delinquent as adolescents). They accepted the conclusion of the early twin studies (e.g., Cloninger & Gottesman, 1987) that genetic influences are minimal for juvenile delinquency, and they hypothesized that delinquency is in many cases transitory and primarily affected by peer pressure.

A review by Moffitt (1993) concurs with L. F. DiLalla and Gottesman's (1989) hypothesis. Moffitt noted that although antisocial behavior shows impressive continuity over age, the prevalence of antisocial behavior increases almost 10-fold during adolescence. She also suggested a subtype hypothesis for antisocial behavior, with the first subtype comprising a small group of members who are antisocial from an early age and who continue to be antisocial during adulthood, and the second subtype being a much larger group whose members have a later age of onset for antisocial behavior and are only antisocial during adolescence. She hypothesized that the correlates and causes of persistent crime or antisocial psychopathology (e.g., genetic influences) may not be found in those who engage in juvenile delinquency.

Two recent twin studies have yielded data that are relevant to the issues of age of onset and continuity of antisocial behavior. First, Slutske, Lyons, et al. (1997) found that antisocial behavior that is earlier in onset is no more heritable than later-onset antisocial behaviors, but they also found that antisocial behavior that is persistent across the life span is more heritable than antisocial behavior that is limited to either childhood or adulthood. Slutske, Lyons, et al. cautioned that the use of retrospective reports may be a limitation of their study. Second, Waldman, Levy, and Hay (1997) examined the etiology of four types of antisocial behavior (i.e., oppositionality, aggression, property violations, and status violations) that vary monotonically in their median age of onset from 6 years old (oppositionality) to 9 years old (status violations). They found that antisocial behavior with an earlier age of onset is more heritable and shows a lesser magnitude of shared environmental influences than antisocial behavior with a later age of onset.

Given that so few twin studies have addressed the issue of age of onset or continuity of antisocial behavior, the present review cannot provide conclusive evidence for or against L. F. DiLalla and Gottesman's (1989) hypothesis. If one assumes, however, that antisocial behavior in adolescents is more transitory in general (although adolescents with continuous and transitory antisocial behavior are not distinguished), the results should indicate that the magnitude of genetic influences on antisocial behavior should be lowest in adolescence.

Sex as a Moderator

No matter how antisocial behavior is operationalized or assessed, it is more prevalent in males than females (e.g., Hyde, 1984; J. Q. Wilson & Herrnstein, 1985). Given this sex difference in prevalence, it is important to consider whether the magnitude of genetic and environmental influences differs in males and females. Therefore, the present meta-analysis examined whether sex is a significant moderator of the results of behavior genetic studies of antisocial behavior by comparing the results for males, females, and both sexes (i.e., studies reporting results for a combined sample of males and females or studies reporting results for opposite-sex twin pairs). Past literature reviews (e.g., Widom & Ames, 1988) have suggested that the magnitude of genetic and environmental influences on antisocial behavior is equal for the two sexes, whereas Miles and Carey (1997) found that the magnitude of genetic influences on aggression was slightly higher for males than for females.

One confusion in this area has to be addressed. The polygenic multiple threshold model attempts to explain the sex difference in prevalence by suggesting that the less affected sex needs a greater liability to manifest the disorder. There has been substantial support for this model in the area of antisocial behavior (e.g., Mednick, Gabrielli, & Hutchings, 1983; Sigvardsson, Cloninger, Bohman, & von Knorring, 1982). Raine and Venables (1992) suggested that such support for the polygenic multiple threshold model conflicts with the evidence that heritability is equal in males and females (Widom & Ames, 1988). The polygenic multiple threshold model makes a prediction about the degree of liability (both genetic and environmental) needed to express a disorder, rather than the magnitude of genetic or environmental influences on within-sex individual differences in antisocial behavior, however. The fact that females may need more liability (either genetic or environmental) to express antisocial behavior does not mean that genetic influences are of greater magnitude in females than males.

Confounding Among Moderators

In examining age, operationalization, and assessment method as moderators, the potential confounding among these variables must be investigated. Antisocial behavior is operationalized and assessed differently for children, adolescents, and adults (e.g., CD assessed by means of parent report in children vs. ASPD assessed by means of self-report in adults). Also, certain operationalizations of antisocial behavior are most frequently or readily assessed using certain methods (e.g., criminality by means of official records). Therefore, the age of the participants, the operationalization, and the assessment method may be all highly correlated across the studies of antisocial behavior.

Given these concerns, we assessed the potential confounding among these moderators in the studies included in the present meta-analysis. Tables 1 and 2, which show the number of studies at each level of the moderators, demonstrate the problem of confounding between the following pairs of moderators: age and operationalization, age and assessment method, and operational-

Table 1
Confounding Between Assessment Method and Operationalization: Number of Samples at Each Level of Moderator

	Assessment method				
Operationalization	Self	Others	Records	Reaction	Objective
Diagnosis	11	2			
Criminality			5		
Aggression	7	4		2	1
Antisocial behavior	4	8			

ization and assessment method. If there was no confounding between the potential moderators, the numbers of studies in these tables would be distributed equally throughout the tables. For example, males and females were nearly equally distributed across the four types of operationalization. In an extreme example, all of the studies using the assessment method of official records were studies examining the operationalization of criminality. In a less extreme example, the studies using the assessment method of report by others tended to be those examining antisocial behavior in childhood.

This type of confounding can make the interpretation of results difficult in two ways. First, if two confounded moderators are both found to be significant, it is possible that the second moderator is significant only because of its confounding with the first moderator. Fortunately, this problem can be assessed in the present meta-analysis. Each of the three moderators in question, operationalization, assessment, and age, was tested for significance after the other two moderators were controlled for statistically. Second, if one level of a moderator is completely confounded with a level of another moderator (e.g., all studies examining criminality being assessed by records), it is unclear whether the results reflect the first or second moderator. Unfortunately, we cannot resolve this problem in the present review. This problem can be addressed in future research, however, by diversifying the pairings among operationalization, assessment method, and age (e.g., by conducting more studies of criminality using a variety of assessment methods, rather than criminal records alone). Tables 1–2, and the corresponding tests of moderators in the meta-analysis, thus serve as a guide to fruitful directions for future behavior genetic studies of antisocial behavior.

Table 2
Confounding Between Age and Operationalization–Assessment Method: Number of Samples at Each Level of Moderator

	Age		
Moderator	Children	Adolescents	Adults
Operationalization			
Diagnosis	2	5	4
Criminality			3
Aggression	6		7
Antisocial behavior	7	5	2
Assessment method			
Self	1	7	12
Others	12	2	
Records			3
Reactive	1		1
Objective	1		

Comparisons Between Twin and Adoption Studies

The results of twin and adoption studies were directly compared in the present meta-analysis. Twin and adoption studies have unique assumptions or biases that can make interpretations of their results difficult. Comparing the results of twin and adoption studies can help determine whether the results of behavior genetic studies have been influenced by these unique assumptions or biases. To the degree that the results of twin and adoption studies are similar, it is more likely that the results reflect the true magnitude of genetic and environmental influences. One cannot rule out the possibility, however, that the results of twin and adoption studies are similar because they share similar biases to some extent that influence their results in the same direction. Therefore, the following assumptions and biases always should be considered when interpreting the results of behavior genetic studies.

In twin studies comparing the correlations between MZ and DZ twin pairs, one has to make the equal environments assumption, or the assumption that the environmental influences on the trait being examined are no more or less similar for MZ twins than for DZ twins. It is possible that the environmental influences on MZ twins are more similar because they are treated more similarly given their similar appearance. This bias could result in the overestimation of genetic influences. Another factor to consider in the equal environments assumption is that approximately two thirds of MZ twin pairs are monochorionic (i.e., share the same chorion), whereas one third of MZ twin pairs and all DZ twin pairs are dichorionic (Melnick, Myrianthopoulos, & Christian, 1978). Failure to account for the effect of sharing a chorion may bias estimates of genetic and environmental influences if prenatal environment influences the trait being examined (Prescott, Johnson, & McArdle, 1999). Several studies have found that monochorionic MZ twins are more similar than dichorionic MZ twins in personality (e.g., Reed, Carmelli, & Rosenman, 1991; Sokol et al., 1995) and cognitive ability (e.g., Rose, Uchida, & Christian, 1981), although others have failed to find significant differences between the two types of MZ twins (e.g., in temperament, Riese, 1999; in cognitive ability, Sokol et al., 1995). Also, sharing a chorion actually may lead to decreased similarity in monochorionic MZ twin pairs because of competition for resources within a twin pair as evidenced by greater similarity in birth weight for dichorionic MZ twins than for monochorionic MZ twins (Corey, Nance, Kang, & Christian, 1979; Vlietinck et al., 1989).

Another assumption of studies examining twins reared together is the assumption that the genetic variance is primarily additive and that there is no epistasis (i.e., interaction between alleles in different loci). The violation of this assumption may lead to overestimation of heritability and underestimation of the magnitude of shared environmental influences (Grayson, 1989). Although twin studies examine models including dominance (i.e., interaction between alleles in the same locus), they do not examine models including epistasis. The coefficient for genetic relationship for epistatic interactions depends on the number of loci involved and the type of interaction (Falconer & Mackay, 1996). The coefficient for genetic relationship for dominance is equal to the coefficient for genetic relationship for epistatic interactions only for Additive × Additive interactions between two loci. Eaves (1988) pointed out that in many behavior genetic studies, the difference between MZ and DZ correlations is much bigger than that predicted under an additive genetic model or a model including dominance (i.e., interaction between alleles in the same locus) alone. He also demonstrated that duplicate gene interactions between pairs of moderately frequent alleles at polygenic loci produce very small genetic correlations (approximately .12) between siblings compared with a genetic correlation of .50 for additive genetic influences and .25 for dominance genetic influences.

Another issue to consider when interpreting the results of twin studies is the generalizability of the findings. First, volunteers in social science studies tend to be above average in socioeconomic status (SES), and this would pertain to twin studies just as it does for other studies. Second, pre- and perinatal complications are more common in twin pairs than in singletons. Twins are born 3 to 4 weeks premature on average, are 30% lighter at birth, and tend to have delayed language development (Plomin, DeFries, McClearn, & Rutter, 1997). Given this concern, several researchers have compared the prevalence of antisocial behavior in twins and singletons and reached differing conclusions. Gjone and Nøvik (1995; Norwegian twins) and van den Oord, Koot, Boomsma, Verhulst, and Orlebeke (1995; Dutch twins) found that the level of antisocial behavior in twins is similar to that of singletons. On the other hand, Gau, Silberg, Erickson, and Hewitt (1992; Virginia twins) found small but consistent differences between the level of antisocial behavior in twins and singletons. Twins had higher levels of antisocial behavior than singletons in both older and younger children. They also found tentative support for the relation between increased perinatal complications and increased childhood behavior problems in twins. If the range of environmental influences is restricted in twin samples for any reason (e.g., higher SES in volunteers; more pre- and perinatal complications), the magnitude of genetic influences may be overestimated.

Adoption studies also have several selection or sampling biases that make interpretation of their results difficult. First, it may be difficult to generalize results of adoption studies because adoptees have a higher rate of antisocial behavior compared with the general population. This finding has been replicated in adoptees in several countries—for example, New Zealand (Fergusson, Lynskey, & Horwood, 1995), the Netherlands (Verhulst, Versluis-den Bieman, van der Ende, Berden, & Sanders-Woudstra, 1990), and the United States (Sharma, McGue, & Benson, 1998). Second, the range of the adoptee's adoptive home environment is restricted. For example, Fergusson et al. (1995) found that adoptees had several advantages over children in the general population in family stability, educational opportunities, standards of health care, material living standards, and mother–child interactions. Although one can ensure that the SES of the adoptive families is similar to that of the control families (e.g., Scarr & Weinberg, 1978), genetic influences may be overestimated and shared environmental influences may be underestimated when the sample's range of environments is restricted (Stoolmiller, 1999). Third, selective placement (viz., matching the environmental characteristics of the biological parents' home and the adoptive parents' home) often occurs in adoptions. Clerget-Darpoux, Goldin, and Gershon (1986) demonstrated how a genetic effect is simulated in adoption studies when there is a positive correlation between the adoptive and biological parents for an etiologic environmental variable.

Two types of adoption studies were included in the present meta-analysis: (a) parent–offspring adoption studies (i.e., comparing the correlation between adoptees and their adoptive parents with the correlation between adoptees and their biological parents) and (b) sibling adoption studies (i.e., comparing the correlation between adoptive siblings with the correlation between biological siblings). When parent–offspring data are interpreted, it is important to consider the possibility that the correlations between the parents and the offspring may be reduced by the age difference between the two generations and that the magnitude of familial (i.e., genetic and shared environmental) influences may be underestimated. Genetic influences on a trait may differ from one generation to another because the genes affecting the same trait may differ in their expression across age because of genotype–environment interaction. For example, genetic influences in the younger generation may be increased because of environmental facilitation of antisocial behavior—for example, by means of secular increases in substance use and less stringent parenting practices (e.g., Lykken, 1997). Also, there may be cohort-specific shared environmental influences other than the cultural transmission from parents to offspring. Unfortunately, the parent–offspring adoption studies included in the present meta-analysis did not provide enough information to address these possibilities. Therefore, each type of adoption study was compared with the twin studies separately, and the parent–offspring adoption studies were compared with the rest of the studies combined (i.e., twin studies and sibling adoption studies).

Behavior genetic studies also make the assumption of random mating. The studies included in the present meta-analysis did not control for the effects of assortative, or nonrandom, mating. A significant correlation between the phenotypes of couples is evidence of assortative mating, and Krueger, Moffitt, Caspi, Bleske, and Silva (1998) found evidence of substantial positive assortative mating for antisocial behavior in their sample of 360 couples from New Zealand. Although assortative mating for personality traits related to antisocial behavior was low ($r = .15$), assortative mating for self-reports of antisocial behavior and tendency to associate with peers who engage in antisocial behavior was high ($r = .54$). Positive assortative mating leads h^2 estimates to be biased downward and c^2 estimates to be biased upward in twin studies because the genetic resemblance between DZ twins is increased. In adoption studies, the h^2 estimate is biased upward because the genetic resemblance between the adopted away offspring and the biological parent, as well as between biological siblings, is increased in the presence of positive assortative mating. As Krueger et al.

suggested, future behavior genetic studies examining antisocial behavior should attempt to control for the effects of assortative mating in order to obtain unbiased estimates of the magnitude of genetic and environmental influences.

Method

Search Strategy

We began our search for twin and adoption studies of antisocial behavior by examining the PsycINFO and *Medline* databases. Appendix A shows the search terms used in this process. The references from the research studies and review articles found through this method were examined for any additional studies that might have been missed or published before the databases were established. Also, information about relevant unpublished manuscripts or manuscripts in press was obtained by examining pertinent reviews and the abstracts of the 1995, 1996, 1997, and 1998 Behavior Genetics Association meetings and searching the *Dissertation Abstracts International* and *Educational Resources Information Center* databases. Authors of 14 manuscripts provided unpublished data; four of these manuscripts were published subsequently.

One hundred forty-one twin and adoption studies examining antisocial behavior were identified. After we excluded unsuitable studies according to the criteria described below (i.e., construct validity, inability to calculate tetrachoric or intraclass correlations, and assessment of related disorders), 96 studies remained. After we addressed the problem of nonindependence in these studies, 51 studies (i.e., 10 independent adoption samples and 42 independent twin samples [two separate samples were examined in Eley et al., 1999]) remained.

Tables 3 and 4 list the 26 adoption studies and 70 twin studies that met the first three inclusion criteria, respectively. The tables are grouped by the 10 independent adoption samples and the 42 independent twin samples in the meta-analysis, and the inclusion–exclusion column indicates which studies were included and which studies were excluded. Tables 3 and 4 also indicate the operationalization examined in the study, the method of assessment, the method of zygosity determination, the mean or midpoint age, the sex of the sample, the number of pairs, the relationship of the pairs, and the effect sizes.

Inclusion Criteria for Studies in the Meta-Analysis

Construct Validity

General issues. Thirteen studies were excluded from the meta-analysis because of inadequate construct validity. The validity of the measures used in the studies considered for the meta-analysis was an important issue in deciding whether to include or exclude a study. Only studies examining antisocial behavior were included, and those examining related constructs such as anger and hostility were excluded. The included studies met one of the following qualifications. First, a study was included if it was clearly evident that it examined ASPD, CD, criminality, or aggression. Examples include studies assessing criminality with official records and ASPD with *DSM* criteria. Second, a study was included if there was empirical evidence that the measure of antisocial behavior used successfully discriminated between an antisocial group and a control group or if the measure was significantly related to a more established operationalization of antisocial behavior. We discuss the validity issues in more detail below for each operationalization of antisocial behavior.

Clinical diagnoses. As mentioned above, studies that used *DSM* criteria to assess ASPD or CD were included. It was not as clear whether studies examining psychopathy should be included in the operationalization of clinical diagnoses. The *DSM–IV* (American Psychiatric Association, 1994) states,

> [The pattern of ASPD] has also been referred to as psychopathy, sociopathy, or dyssocial personality disorder. Because deceit and manipulation are central features of ASPD, it may be especially helpful to integrate information acquired from systematic clinical assessment with information collected from collateral sources. (pp. 645–646)

However, some researchers have emphasized the difference between the *DSM* criteria and the traditional concept of psychopathy, noting that the *DSM* criteria for ASPD focus on antisocial behavior whereas the traditional concept of psychopathy focuses on personality traits (e.g., Hare, Hart, & Harpur, 1991).

The two personality measures used most often in assessing psychopathy are the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1942) Psychopathic Deviate (*Pd*) scale and the California Psychological Inventory (CPI; Gough, 1969) Socialization (*So*) scale. The MMPI was constructed empirically to distinguish nonpsychopathological from psychopathological populations, whereas the CPI was justified theoretically to describe variation within the general population. Approximately one third of the items on the CPI were derived from the MMPI, however. Given the evidence that psychopathy measures and the *DSM* criteria are related (e.g., Cooney, Kadden, & Litt, 1990), psychopathy measures were included as an operationalization of diagnosis. Nonetheless, given the concern that psychopathy and ASPD are not synonymous (e.g., Hare et al., 1991), the meta-analysis was repeated after excluding studies examining psychopathy (eight samples; Brandon & Rose, 1995; D. L. DiLalla, Carey, Gottesman, & Bouchard, 1996; Gottesman, 1963, 1965; Loehlin & Nichols, 1976; Loehlin, Willerman, & Horn, 1987; Taylor, McGue, Iacono, & Lykken, 2000; Torgersen, Skre, Onstad, Edvardsen, & Kringlen, 1993) to examine the sensitivity of the results to such studies.

Criminality and delinquency. All studies examining criminality used the assessment method of official records of arrests or convictions and were therefore included in the meta-analysis.

Aggression. A study examining aggression was included if it examined behavioral aggression (e.g., physical fighting, cruelty to animals, and bullying). For studies that did not meet this criterion, several issues regarding validity had to be resolved. First, 12 studies that examined other related variables such as anger, hostility, or impulsivity were not included. These studies were excluded because it was not clear whether they examined aggression or some related but distinct trait. Second, Partanen et al. (1966) was excluded because although it reported that it examined aggression, the aggression items examined in this study (e.g., "Are you readily insulted?" and "Do you easily become unhappy about even small things?") suggest that negative affect or anger, rather than aggression, was being assessed. Third, some studies examining aggression used measures with questionable validity (i.e., lack of evidence or inconclusive evidence regarding validity). For example, the Missouri Children's Picture Series (Sines, Pauker, & Sines, 1966, as cited in Owen & Sines, 1970) used by Owen and Sines distinguished institutionalized aggressive boys from boys from the general population (Defilippis, 1979) but did not distinguish teacher-referred children with behavior problems versus learning problems (Ollendick & Woodward, 1982). The meta-analysis was repeated after excluding the studies using measures with questionable validity (2 samples; Owen & Sines, 1970; G. D. Wilson et al., 1977) to assess the sensitivity of the obtained results to inclusion of such measures.

Antisocial behavior. A fourth operationalization, antisocial behavior, was included because several studies clearly examined antisocial behavior without specifically examining ASPD, CD, criminality, delinquency, or aggression (e.g., Rowe, 1983; Stevenson & Graham, 1988; Waldman, McGue, Pickens, & Svikis, in press). All of these studies examined a combination of delinquency and aggression items (e.g., the externalizing scale from the Child Behavior Checklist; Achenbach & Edelbrock, 1983). Many of the individual items examined in these studies are criteria for CD,

(*text continues on page 508*)

Table 3
Effect Sizes for Adoption Studies of Antisocial Behavior

Study	Operationalization	Assessment	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Texas adoptees								
Loehlin et al. (1985)	Psychopathy	Self-report		both-m	52	a-bf	.16	Excluded
				both-fm	53	a-bm	.06	Excluded
				both-m	241	a-af	−.03	Excluded
				both-fm	253	a-am	−.02	Included—adoption
Loehlin et al. (1987)	Psychopathy	Self-report		both-m	81	a-bf	.12	Included—averaged
				both-fm	81	a-bm	.07	Included—averaged
				both-m	180	a-af	.07	Excluded
				both-fm	177	a-am	.01	Excluded
St. Louis adoptees								
Cadoret et al. (1975)	ASB	Records–parent report		both-both	76	a-bp	.23	Excluded
Cunningham et al. (1975)	ASB	Records–parent report		both-both	77	a-bp	.16	Included—largest
Dutch adoptees								
van den Oord et al. (1994)	ASB	Parent report	12.40	fm-fm	35	b sibs	.42	Included—independent
					48	a sibs	.37	Included—independent
				m-m	30	b sibs	.46	Included—independent
					44	a sibs	.19	Included—independent
				m-f	46	b sibs	.52	Included—independent
					129	a sibs	.11	Included—independent
U.S. adoptees (CO, IL, MN, WI)								
McGue et al. (1996)	ASB (externalizing)	Self-report	15.00	both-both	194	a sibs (a-a)	.17	Included—averaged
					72	a sibs (a-b)	.06	Included—averaged
	ASB (antisocial)				194	a sibs (a-a)	.05	Included—averaged
					72	a sibs (a-b)	.34	Included—averaged
Iowa adoptees								
Cadoret (1978)	ASB/criminality–ASP/CD	Records–self- or parent report		m-both	81	a-bp	.10	Excluded
				fm-both	56	a-bp	.42	Excluded
Cadoret et al. (1985)	ASB–ASP	Records–self- or parent report		m-both	127	a-br	.43	Excluded
				fm-both	87	a-br	.20	Excluded
Cadoret et al. (1986)	ASB–ASP	Records–self-report		both-both	443	a-bp	.40	Included—largest
Cadoret et al. (1987)	ASB–ASP	Records–self-report		m-both	160	a-bp	.37	Excluded
Cadoret et al. (1990)	Criminality/delinquency-ASP	Records–self- or parent report		m-both	286	a-bp	.47	Excluded
Cadoret & Stewart (1991)	Criminality/delinquency–ASP	Records–self-report		m-both	283	a-bp	.47	Excluded
Iowa adoptees (1990s—males)								
Cadoret et al. (1995)	ASPD–aggressivity	Records–parent report		m-both	95	a-bp	.53	Included—independent
Iowa adoptees (1990s—females)								
Cadoret et al. (1996)	ASP–CD	Records–self-report		fm-both	102	a-bp	.49	Included—independent
Danish adoptees								
Hutchings & Mednick (1971)	Criminality	Records		m-m	971	a-bf	.19	Excluded
Mednick et al. (1983)	Criminality	Records		m-both	3,722	a-bp	.20	Excluded
Gabrielli & Mednick (1984)	Criminality	Records		m-both	2,592	a-bp	.17	Excluded
Baker (1986)	Criminality	Records		m-fm	2,532	a-bm	.17	Excluded
				m-m		a-bf	.04	Excluded
				m-fm		a-am	−.06	Excluded
				m-m		a-af	.06	Excluded

Table 3 (*continued*)

Study	Operationalization	Assessment	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Danish adoptees (*continued*)								
Baker et al. (1989)	Criminality	Records		fm-fm	7,065	a-bm	.15	
				fm-m		a-bf	.12	
				fm-both		a-bp	.14	Included—largest fm
				fm-fm		a-am	−.02	
				fm-m		a-af	.05	
				fm-both		a-ap	.01	Included—adoption
				m-fm	6,129	a-bm	.20	
				m-m		a-bf	.14	
				m-both		a-bp	.17	Included—largest m
				m-fm		a-am	.06	
				m-m		a-af	.11	
				m-both		a-ap	.09	Included—adoption
Swedish adoptees								
Bohman (1978)	Criminality	Records		m-m	892	a-bf	.01	Excluded
				m-f	1,077	a-bm	.00	Included—largest m
				fm-both	1,988	a-bp	.12	Included—largest fm
Bohman et al. (1982)	Criminality	Records		m-both	702	a-bp	.11	Excluded
Sigvardsson et al. (1982)	Criminality	Records		fm-both	407	a-bp	.34	Excluded
					457	a-ap	−.23	Excluded
Colorado adoptees								
Deater-Deckard & Plomin (1999)	Aggression	Parent report	9.5	both-both	78	a sibs	.26	Excluded
					94	b sibs	.39	Excluded
		Teacher report			78	a sibs	−.06	Excluded
					94	b sibs	.25	Excluded
	Delinquency	Parent report			78	a sibs	.22	Excluded
					94	b sibs	.43	Excluded
		Teacher report			78	a sibs	.14	Excluded
					94	b sibs	.24	Excluded
Young et al. (1997, personal communication)	CD–dimensional (adoptive sample)	Self-report	18.60	both-both	56	a sibs	.10	Excluded
	CD–dimensional (control sample)		18.10		42	b sibs	.17	Excluded
	CD–dimensional (treatment sample)		17.05		144	b sibs	.04	Excluded
	CD–threshold (adoptive sample)		18.60		56	a sibs	.00	Included—largest
	CD–threshold (control + treatment)		17.58		186	b sibs	.31	Included—largest
Young et al. (1996, personal communication)	CD (adoptive sample)	Self-report		m-m	43	a-af	.15	Excluded
				m-fm	57	a-am	−.02	Excluded
	CD (control sample)			m-m	96	b-bf	.17	Excluded
				m-fm	87	b-bm	.16	Excluded
	CD (treatment sample)			m-m	34	b-bf	.21	Excluded
				m-fm	86	b-bm	.28	Excluded
Parker et al., (1989, as cited in Carey, 1994)	Aggression	Parent report	4.00	both-both	66	b sibs	.42	Excluded
					45	a sibs	.54	Excluded
			7.00		19	b sibs	.55	Excluded
					17	a sibs	.28	Excluded

Note. Information within parentheses indicates whether data were obtained from personal communication or another publication. both = both male and female; m = male; a-bf = adoptee-biological father; Excluded = excluded from the meta-analysis because another study examined the same sample that was larger, more unique in assessment method, or better described; fm = female; a-bm = adoptee-biological mother; a-af = adoptee-adoptive father; a-am = adoptee-adoptive mother; Included—adoption = included only for analysis comparing parent–offspring studies with other types of studies; Included—averaged = included after using the averaging method of dealing with nonindependence; a-bp = adoptee–biological parent; ASB = antisocial behavior; Included—largest = included after using the largest sample method of dealing with nonindependence; b sibs = biological siblings; Included—independent = included because the study did not have a nonindependence problem; a sibs = adoptive siblings; CO = Colorado; IL = Illinois; MN = Minnesota; WI = Wisconsin; a-a = adoptive—adoptive sibling pair; a-b = adoptive—biological sibling pair; ASP = antisocial personality; CD = conduct disorder; a-br = adoptee–biological relative; ASPD = antisocial personality disorder; a-ap = adoptee–adoptive parent; b-bf = biological child–biological father; b-bm = biological child–biological mother.

Table 4
Effect Sizes for Twin Studies of Antisocial Behavior

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Midwest twins									
Cates et al. (1993)	Aggression (assault)	Self-report	Blood grouping–questionnaire	42.50	fm-fm	77	MZ	.07	Included—averaged
						21	DZ	.41	Included—averaged
	Aggression (verbal)					77	MZ	.41	Included—averaged
						21	DZ	.06	Included—averaged
	Aggression (indirect)					77	MZ	.40	Included—averaged
						21	DZ	.01	Included—averaged
NAS-NRC twins									
Centerwall & Robinette (1989)	Criminality	Records	Blood grouping–questionnaire–fingerprinting	36.50	m-m	5,933	MZ	.74	Included—largest
						7,554	DZ	.29	Included—largest
Horn et al. (1976)	Psychopathy	Self-report	Blood grouping		m-m	99	MZ	.43	Excluded
						99	DZ	.25	Excluded
Maudsley twins (psychiatric sample)									
Coid et al. (1993)	Criminality	Records	Blood grouping–questionnaire	45.90	both-both	92	MZ	.70	Included—independent
						109	DZ	.80	Included—independent
California twins									
Ghodsian-Carpey & Baker (1987)	Aggression	Parent report (CBCL–A)	Questionnaire	5.20	both-both	21	MZ	.78	Included—largest
						17	DZ	.31	Included—largest
		Aggression (MOCL)				17	MZ	.65	Excluded
						15	DZ	.35	Excluded
Danish twins									
Carey (1992)	Criminality	Records	Blood grouping–questionnaire	Lifetime	m-m	365	MZ	.74	Included—largest
						700	DZ	.47	Included—largest
					fm-fm	347	MZ	.74	Included—largest
						690	DZ	.46	Included—largest
					m-fm	2,073	DZ	.23	Included—largest
Christiansen (1973)	Criminality	Records	Blood grouping–questionnaire	Lifetime	m-m	325	MZ	.70	Excluded
						604	DZ	.29	Excluded
Christiansen (1974)	Criminality	Records	Blood grouping–questionnaire	Lifetime	m-m	132	MZ	.02	Excluded
						191	DZ	−.41	Excluded
						132	MZ	.45	Excluded
						191	DZ	−.03	Excluded
						132	MZ	.48	Excluded
						191	DZ	−.18	Excluded
Christiansen (1977a)	Criminality	Records	Blood grouping–questionnaire	Lifetime	m-m	325	MZ	.70	Excluded
						611	DZ	.28	Excluded
					fm-fm	328	MZ	.72	Excluded
						593	DZ	.41	Excluded
					m-fm	1,547	DZ	−.01	Excluded
Cloninger et al. (1978)	Criminality	Records	Blood grouping–questionnaire	Lifetime	m-m	338	MZ	.69	Excluded
						637	DZ	.36	Excluded
					fm-fm	323	MZ	.72	Excluded
						615	DZ	.43	Excluded
					m-fm	2,053	DZ	.24	Excluded

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
London twins (children)									
Stevenson & Graham (1988)	ASB	Parent report	Blood grouping–questionnaire–fingerprinting	13.00	m-m	46	MZ	.61	Included—independent
						48	DZ	.40	Included—independent
					fm-fm	53	MZ	.29	Included—independent
						58	DZ	.49	Included—independent
London twins (adults—1970s)									
G. D. Wilson et al. (1977)	Aggression	Reaction to stimuli		30.50	both-both	49	MZ	.59	Included—independent
						52	DZ	.34	Included—independent
London twins (adults—1980s)									
Rushton et al. (1986)	Aggression	Self-report	Blood grouping–questionnaire	30.00	m-m	90	MZ	.33	Included—independent
						46	DZ	.16	Included—independent
					fm-fm	206	MZ	.43	Included—independent
						133	DZ	.00	Included—independent
					m-fm	98	DZ	.12	Included—independent
Rushton (1996)	Delinquency	Self-report		50.00	m-m	42	MZ	.77	Excluded
						27	DZ	.44	Excluded
					fm-fm	126	MZ	.73	Excluded
						79	DZ	.47	Excluded
	Violence				m-m	42	MZ	.53	Excluded
						27	DZ	.06	Excluded
					fm-fm	126	MZ	.27	Excluded
						79	DZ	.05	Excluded
Minnesota twins (reared apart)									
D. L. DiLalla et al. (1996)	Psychopathy	Self-report	Blood grouping	40.40	both-both	66	MZ ra	.62	Included—largest
				45.10		54	DZ ra	.14	Included—largest
Grove et al. (1990)	Adult ASP	Self-report	Blood grouping	43.00	both-both	32	MZ ra	.41	Excluded
	Child ASP							.28	Excluded
Bouchard & McGue (1990)	Psychopathy	Self-report	Blood grouping	41.50	both-both	45	MZ ra	.53	Excluded
						26	DZ ra	.39	Excluded
Gottesman et al. (1984, as cited in Carey, 1994)	Psychopathy	Self-report			both-both	51	MZ ra	.64	Excluded
						25	DZ ra	.34	Excluded
Tellegen et al. (1988)	Aggression	Self-report	Blood grouping	40.90	both-both	44	MZ ra	.46	Excluded
						27	DZ ra	.06	Excluded
Minnesota twins (reared together—1970s)									
Tellegen et al. (1988)	Aggression	Self-report	Blood grouping	21.65	both-both	217	MZ	.43	Included—exception
						114	DZ	.14	Included—exception
Lykken et al. (1978)	Aggression	Self-report			m-m	88	MZ	.66	Excluded
						46	DZ	−.06	Excluded
					fm-fm	174	MZ	.43	Excluded
						92	DZ	.24	Excluded
McGue et al. (1993)	Aggression	Self-report	Blood grouping	19.80	both-both	79	MZ	.61	Excluded
						48	DZ	−.09	Excluded
	Aggression—follow-up			29.60		79	MZ	.58	Excluded
						48	DZ	−.14	Excluded

(*table continues*)

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Minnesota twins (1960s—high school sample)									
Gottesman (1963, as cited in Gottesman & Goldsmith, 1994)	Psychopathy	Self-report	Blood grouping	16.00	both-both	34	MZ	.57	Included—largest
						34	DZ	.18	Included—largest
Dworkin et al. (1976)	Psychopathy (MMPI)	Self-report	Blood grouping	15.90	both-both	25	MZ	.49	Excluded
						17	DZ	.30	Excluded
	Psychopathy (CPI)					25	MZ	.51	Excluded
						17	DZ	.31	Excluded
	Psychopathy—follow-up (MMPI)			27.90		25	MZ	.44	Excluded
						17	DZ	.34	Excluded
	Psychopathy—follow-up (CPI)					25	MZ	.69	Excluded
						17	DZ	.46	Excluded
Minnesota twins (1990s—adolescents)									
Taylor et al. (2000, personal communication)	Psychopathy	Self-report	Blood grouping–questionnaire	17.00	m-m	145	MZ	.52	Included—averaged
						77	DZ	.15	Included—averaged
					fm-fm	107	MZ	.48	Included—averaged
						52	DZ	.37	Included—averaged
	Delinquency				m-m	145	MZ	.51	Included—averaged
						77	DZ	.29	Included—averaged
					fm-fm	107	MZ	.60	Included—averaged
						52	DZ	.38	Included—averaged
Hershberger et al. (1995, personal communication)	CD	Self-report	Blood grouping–questionnaire	17.00	m-m	138	MZ	.72	Excluded
						67	DZ	.38	Excluded
Minnesota twins (1990s—adults)									
Finkel & McGue (1997)	Aggression	Self-report	Blood grouping–questionnaire	37.76	m-m	220	MZ	.37	Included—independent
						165	DZ	.12	Included—independent
					fm-fm	406	MZ	.39	Included—independent
						352	DZ	.14	Included—independent
					m-fm	114	DZ	.12	Included—independent
Minnesota twins (sample with alcoholism)									
Waldman et al. (in press, personal communication)	ASB	Self-report	Blood grouping–questionnaire	34.50	m-m	92	MZ	.37	Included—independent
						104	DZ	.31	Included—independent
					fm-fm	50	MZ	.50	Included—independent
						46	DZ	.34	Included—independent
					m-fm	74	DZ	.30	Included—independent
					fm-m	26	DZ	.43	Included—independent
Boston twins (adolescents)									
Gottesman (1965, as cited in Gottesman & Goldsmith, 1994)	Psychopathy	Self-report	Blood grouping	16.00	both-both	80	MZ	.46	Included—largest
						68	DZ	.25	Included—largest
Gottesman (1966, as cited in Carey, 1994)	Psychopathy	Self-report	Blood grouping	16.00	m-m	34	MZ	.32	Excluded
						32	DZ	.06	Excluded
					fm-fm	45	MZ	.52	Excluded
						36	DZ	.26	Excluded
Boston twins (children)									
Scarr (1966)	Aggression	Parent report	Blood grouping	8.08	fm-fm	24	MZ	.35	Included—independent
						28	DZ	−.08	Included—independent

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Vancouver twins									
Livesley et al. (1993)	ASB	Self-report	Questionnaire	28.68	both-both	90	MZ	.52	Included—independent
						85	DZ	.52	Included—independent
National Merit Scholarship twins									
Loehlin & Nichols (1976)	Psychopathy	Self-report	Questionnaire	18.00	m-m	202	MZ	.52	Included—independent
						124	DZ	.15	Included—independent
					fm-fm	288	MZ	.55	Included—independent
						193	DZ	.48	Included—independent
Calgary twins									
Lytton et al. (1988)	ASB	Teacher report		9.50	m-m	15	MZ	.85	Included—largest
						22	DZ	.47	Included—largest
		Mother report				13	MZ	.87	Excluded
						22	DZ	.67	Excluded
		Father report				12	MZ	.96	Excluded
						16	DZ	.96	Excluded
Philadelphia twins									
Meininger et al. (1988)	Aggression	Teacher report	Blood grouping	8.50	both-both	61	MZ	.67	Included—independent
						34	DZ	.11	Included—independent
Missouri twins									
Owen & Sines (1970)	Aggression	Reaction to stimuli	Blood grouping	10.00	m-m	10	MZ	.09	Included—independent
						11	DZ	−.24	Included—independent
					fm-fm	11	MZ	.58	Included—independent
						13	DZ	.22	Included—independent
Colorado twins (1980s)									
M. O'Connor et al. (1980)	Aggression	Parent report	Questionnaire	7.60	both-both	54	MZ	.72	Excluded
						33	DZ	.42	Excluded
Plomin & Foch (1980)	Aggression (no. of hits)	Objective test	Questionnaire	7.60	both-both	42	MZ	.44	Excluded
						29	DZ	.42	Excluded
	Aggression (intensity of hits)					43	MZ	.38	Excluded
						28	DZ	.48	Excluded
	Aggression (no. of quadrants)					40	MZ	.22	Excluded
						28	DZ	.44	Excluded
Plomin (1981)	Aggression (no. of hits)	Objective test	Questionnaire	7.60	both-both	53	MZ	.42	Included—exception
						32	DZ	.42	Included—exception
	Aggression (intensity of hits)					53	MZ	.39	Excluded
						31	DZ	.47	Excluded
	Aggression (no. of quadrants)					53	MZ	.23	Excluded
						31	DZ	.41	Excluded
Colorado twins (1990s)									
Zahn-Waxler et al. (1996)	ASB	Mother report	Blood typing–questionnaire	5.00	both-both	<200	MZ	.87	Excluded
						<200	DZ	.59	Excluded
		Father report			both-both	<200	MZ	.85	Excluded
						<200	DZ	.60	Excluded
Schmitz et al. (1994)	ASB	Parent report	Questionnaire	2.75	m-m	32	MZ	.55	Excluded
						26	DZ	.46	Excluded
					fm-fm	37	MZ	.71	Excluded
						45	DZ	.39	Excluded
					m-fm	89	DZ	.45	Excluded

(*table continues*)

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Colorado twins (1990s) (*continued*)									
Schmitz et al. (1995)	ASB	Parent report	Questionnaire	2.83	both-both	154	MZ	.70	Included—largest
						366	DZ	.44	Included—largest
Colorado twins (sample with LD and control sample)									
Willcutt et al. (1995, personal communication)	CD–sample with LD	Parent report		13.00	both-both	80	MZ	.76	Included—independent
						118	DZ	.57	Included—independent
	CD–control sample					40	MZ	.63	Included—independent
						45	DZ	.46	Included—independent
Ohio twins									
Rowe (1983)	ASB	Self-report	Blood grouping–questionnaire	17.50	m-m	61	MZ	.66	Included—independent
						38	DZ	.48	Included—independent
					fm-fm	107	MZ	.74	Included—independent
						59	DZ	.47	Included—independent
Norwegian twins (psychiatric sample)									
Torgersen et al. (1993)	Psychopathy	Self-report			both-both	24	MZ	.22	Included—independent
						28	DZ	.20	Included—independent
California twins									
Rahe et al. (1978)	Aggression	Self-report	Blood grouping	48.00	m-m	82	MZ	.31	Included—independent
						79	DZ	.21	Included—independent
Virginia twins									
Eaves et al. (1997, only largest sample reported)	CD	Self-report	Blood grouping–questionnaire	12.00	m-m	289	MZ	.36	Included—largest
						177	DZ	.13	Included—largest
					fm-fm	380	MZ	.24	Included—largest
						185	DZ	.19	Included—largest
					m-fm	283	DZ	.10	Included—largest
Silberg et al. (1994)	ASB	Parent report	Questionnaire	12.00	m-m	242	MZ	.85	Excluded
						253	DZ	.65	Excluded
					fm-fm	272	MZ	.78	Excluded
						233	DZ	.64	Excluded
					m-fm	271	DZ	.70	Excluded
Silberg et al. (1996)	ASB	Parent report	Blood grouping–questionnaire	14.00	m-m	106	MZ	.68	Excluded
						82	DZ	.36	Excluded
					fm-fm	162	MZ	.68	Excluded
						77	DZ	.53	Excluded
					m-fm	130	DZ	.41	Excluded
				9.5	m-m	159	MZ	.70	Excluded
						81	DZ	.33	Excluded
					fm-fm	185	MZ	.69	Excluded
						83	DZ	.49	Excluded
					m-fm	132	DZ	.40	Excluded
Simonoff et al. (1995)	Aggression–ASB	Mother report	Blood grouping–questionnaire		m-m	169	MZ	.81	Excluded
						113	DZ	.68	Excluded
		Father report				169	MZ	.77	Excluded
						113	DZ	.58	Excluded
		Self-report				169	MZ	.60	Excluded
						113	DZ	.41	Excluded

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Virginia twins (*continued*)									
Simonoff et al. (1995) (*continued*)									
	ODD and CD	Mother report				169	MZ	.65	Excluded
						113	DZ	.37	Excluded
		Father report				169	MZ	.75	Excluded
						113	DZ	.28	Excluded
		Self-report				169	MZ	.42	Excluded
						113	DZ	.11	Excluded
Indiana twins									
Pogue-Geile & Rose (1985)	Psychopathy	Self-report	Blood grouping	20.20	both-both	101	MZ	.47	Excluded
						102	DZ	.15	Excluded
				24.55	both-both	71	MZ	.23	Excluded
						62	DZ	.20	Excluded
Rose (1988, as cited in Gottesman & Goldsmith, 1994)	Psychopathy	Self-report	Blood grouping	24.00	both-both	228	MZ	.47	Excluded
						182	DZ	.23	Excluded
Brandon & Rose (1995, personal communication)	Psychopathy	Self-report	Blood grouping–questionnaire	20.35	both-both	289	MZ	.48	Included—largest
						228	DZ	.27	Included—largest
Western Reserve twins									
Edelbrock et al. (1995)	ASB	Parent report	Blood grouping–questionnaire	11.00	both-both	99	MZ	.79	Included—independent
						82	DZ	.53	Included—independent
British Columbia twins									
Blanchard et al. (1995, personal communication)	Aggression	Self-report		36.18	both-both	96	MZ	.59	Included—independent
						48	DZ	.34	Included—independent
Australian twins (children)									
Waldman et al. (1995, personal communication)	CD	Parent report	Questionnaire	8.66	m-m	437	MZ	.84	Included—independent
						278	DZ	.66	Included—independent
					fm-fm	495	MZ	.89	Included—independent
						225	DZ	.61	Included—independent
					m-fm	437	DZ	.56	Included—independent
Australian twins (adults)									
Slutske, Heath, et al. (1997)	CD	Self-report	Questionnaire	43.70	m-m	401	MZ	.71	Included—independent
						236	DZ	.35	Included—independent
					fm-fm	940	MZ	.68	Included—independent
						540	DZ	.47	Included—independent
					m-fm	604	DZ	.32	Included—independent
Dutch twins									
van den Oord et al. (1996)	Aggression	Parent report	Blood grouping–questionnaire	3.00	m-m	210	MZ	.81	Included—independent
						265	DZ	.49	Included—independent
					fm-fm	236	MZ	.83	Included—independent
						238	DZ	.49	Included—independent
					m-fm	409	DZ	.45	Included—independent
VET twins									
Lyons et al. (1995)	ASPD (juvenile traits)	Self-report	Blood grouping–questionnaire	44.60	m-m	1,788	MZ	.39	Included—averaged
						1,438	DZ	.33	Included—averaged
	ASPD (adult traits)					1,788	MZ	.47	Included—averaged
						1,438	DZ	.27	Included—averaged

(*table continues*)

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
VET twins (*continued*)									
Coccaro et al. (1997)	Aggression (direct)	Self-report	Blood grouping–questionnaire	44.13	m-m	182	MZ	.50	Excluded
						118	DZ	.19	Excluded
	Aggression (indirect)					182	MZ	.42	Excluded
						118	DZ	.02	Excluded
	Aggression (verbal)					182	MZ	.28	Excluded
						118	DZ	.07	Excluded
NEAD twins									
Neiderhiser et al. (1998; results—personal communication)	ASB	Multiple methods	Questionnaire	13.50	m-m	43	MZ	.77	Excluded
						46	DZ	.71	Excluded
					fm-fm	46	MZ	.87	Excluded
						46	DZ	.57	Excluded
T. G. O'Connor, McGuire, et al. (1998)	ASB	Multiple methods	Questionnaire	13.70	both-both	93	MZ	.81	Included—averaged
						99	DZ	.68	Included—averaged
T. G. O'Connor, Neiderhiser, et al. (1998)	ASB	Multiple methods	Questionnaire	16.70	both-both	63	MZ	.75	Excluded
						75	DZ	.36	Excluded
Deater-Deckard & Dodge (1997)	ASB	Mother report (Wave 1)	Questionnaire	13.70	both-both	93	MZ	.84	Included—averaged
						99	DZ	.59	Included—averaged
		Father report (Wave 1)				93	MZ	.77	Included—averaged
						99	DZ	.59	Included—averaged
		Mother report (Wave 2)		16.70		63	MZ	.66	Excluded
						73	DZ	.43	Excluded
		Father report (Wave 2)				63	MZ	.80	Excluded
						73	DZ	.47	Excluded
Pike et al. (1996)	ASB	Multiple methods	Questionnaire	14.50	both-both	93	MZ	.80	Excluded
						98	DZ	.65	Excluded
Indian twins									
Nathawat & Puri (1995)	Socialization	Self-report		24.00	both-both	15	MZ	.67	Included—averaged
						15	DZ	.47	Included—averaged
	Indirect aggression					15	MZ	.56	Included—averaged
						15	DZ	.01	Included—averaged
	Verbal aggression					15	MZ	.10	Included—averaged
						15	DZ	.03	Included—averaged
Cardiff twins									
Thapar & McGuffin (1996)	ASB	Parent report	Questionnaire	12.00	m-m	44	MZ	.62	Included—independent
						45	DZ	.60	Included—independent
					fm-fm	69	MZ	.62	Included—independent
						40	DZ	.60	Included—independent
Swedish twins (adults)									
Gustavsson et al. (1996)	Aggression (indirect)	Self-report			both-both	15	MZ ra	.22	Included—averaged
						26	MZ	.41	Included—averaged
						29	DZ	.27	Included—averaged
	Aggression (verbal)					15	MZ ra	−.03	Included—averaged
						26	MZ	.22	Included—averaged
						29	DZ	.23	Included—averaged

Table 4 (*continued*)

Study	Operationalization	Assessment	Zygosity	Age	Sex	*N*	Relationship	Effect size	Inclusion–exclusion
Swedish twins (children)									
Eley et al. (1999)	Aggression	Parent report	Questionnaire	8.50	m-m	176	MZ	.72	Included—averaged
						182	DZ	.41	Included—averaged
					fm-fm	160	MZ	.82	Included—averaged
						194	DZ	.45	Included—averaged
					m-fm	310	DZ	.41	Included—averaged
	Delinquency				m-m	176	MZ	.71	Included—averaged
						182	DZ	.59	Included—averaged
					fm-fm	160	MZ	.78	Included—averaged
						194	DZ	.60	Included—averaged
					m-fm	310	DZ	.46	Included—averaged
British twins									
Eley et al. (1999)	Aggression	Parent report	Questionnaire	12.00	m-m	99	MZ	.68	Included—averaged
						93	DZ	.45	Included—averaged
					fm-fm	124	MZ	.77	Included—averaged
						80	DZ	.44	Included—averaged
					m-fm	95	DZ	.27	Included—averaged
	Delinquency				m-m	99	MZ	.65	Included—averaged
						93	DZ	.66	Included—averaged
					fm-fm	124	MZ	.75	Included—averaged
						80	DZ	.57	Included—averaged
					m-fm	95	DZ	.32	Included—averaged
New York twins									
Seelig & Brandon (1997, personal communication)	ASB	Mother report		15.00	both-both	45	MZ	.75	Included—averaged
						38	DZ/b sibs	.31	Included—averaged
		Father report				45	MZ	.79	Included—averaged
						38	DZ/b sibs	.75	Included—averaged
		Self-report				45	MZ	.44	Included—averaged
						38	DZ/b sibs	.62	Included—averaged

Note. Information within parentheses indicates whether data were obtained from personal communication or another publication. fm = female; MZ = monozygotic twin pairs; Included—averaged = included after using the averaging method of dealing with nonindependence; DZ = dizygotic twin pairs; m = male; NAS-NRC = National Academy of Sciences–National Research Council; Included—largest = included after using the largest sample method of dealing with nonindependence; Excluded = excluded from the meta-analysis because another study examined the same sample that was larger, more unique in assessment method, or better described; both = both male and female; Included—independent = included because the study does not have a nonindependence problem; CBCL–A = Child Behavior Check List—Aggression; MOCL = Mothers' Observations Check List; ASB = antisocial behavior; ra = reared apart; ASP = antisocial personality; Included—exception = one of two exceptions in which the largest sample was not included; MMPI = Minnesota Multiphasic Personality Inventory; CPI = California Psychological Inventory; CD = conduct disorder; LD = learning disability; ODD = oppositional defiant disorder; VET = Vietnam era twins; ASPD = antisocial personality disorder; NEAD = nonshared environment in adolescent development; DZ/b = dizygotic twin pairs/biological sibling pairs; sibs = siblings.

which involves delinquency and aggression, but the operationalization of CD was reserved for studies that assessed the actual *DSM* criteria.

Inability to Calculate Tetrachoric or Intraclass Correlations

The effect sizes used in this meta-analysis were the Pearson product–moment or intraclass correlations that were reported in the studies or the tetrachoric correlations that were estimated from the concordances or percentages reported in the studies. These effect sizes were analyzed in model-fitting programs that estimate the relative contribution of genetic and environmental influences and test alternative etiologic models. Twenty-six studies were excluded from the meta-analysis because effect sizes were not reported or because there was not enough information reported to calculate the effect sizes.

Four frequently cited adoption studies were excluded for this reason. First, Crowe (1972, 1974, 1975) found that adopted away offspring of female criminal offenders were more likely to be criminal and have antisocial personality than adopted away offspring of controls, thus yielding evidence for genetic influences on antisocial behavior. Crowe counted the 52 adoptees of 41 biological mothers as individual cases rather than counting the 41 biological mothers as individual cases, creating a problem of nonindependence in mother–adopted away offspring pairs. Second, Jary and Stewart (1985) found that biological fathers of children with aggressive CD were more likely to have antisocial personality than adoptive fathers of children with aggressive CDs. They did not report similar information regarding the parents of a control group without aggressive CD, however. A comparison between the aggressive group and the control group is necessary for the estimation of a tetrachoric correlation, as the control group would provide the base rate of antisocial behavior in this sample.

Three early twin studies were excluded because they did not permit adequate effect size estimation (Dalgard & Kringlen, 1976; Hayashi, 1967; Rosanoff, Handy, & Rosanoff, 1934). These studies located twin pairs with at least one affected member (i.e., the proband), then compared the risk to the cotwin in MZ twin pairs and DZ twin pairs. The risk was estimated using either the pairwise concordance or the probandwise concordance. Studies using this method do not include the base rate of the variable of interest (i.e., how prevalent the condition is in the sample being studied), which is necessary for the estimation of tetrachoric correlations. In other words, these studies reported the number of twin pairs that are concordant for being affected and the number of twin pairs that are discordant but did not report the number of twin pairs that are concordant for being unaffected. There are two related problems that this entails. First, the estimation of the effect sizes used in this meta-analysis, tetrachoric or intraclass correlations, is impossible without the base rate. Second, concordances themselves may be misleading because their interpretation varies according to the base rate (A. Heath, personal communication, March 1991).

One possible way to include these early studies in the meta-analysis would be to use the base rate for antisocial behavior in the country from which the sample was drawn and include this information to estimate tetrachoric correlations. One unpublished meta-analysis of antisocial behavior (Ridenour & Heath's, 1997, meta-analysis of categorically defined antisocial behavior) took such an approach. We decided against this approach for the following reason. Even if the base rate for antisocial behavior were found for the specific countries of interest, it may not be appropriate for the specific operationalization used by the studies, the year the studies were published, and many other specific factors that can make the sample examined by the study quite different from a random sample from the population for which the base rate was derived.

The early twin studies excluded from this meta-analysis because of failure to provide the appropriate base rate have been discussed in many traditional literature reviews (e.g., Christiansen, 1977b; Cloninger & Gottesman, 1987). With the exception of Dalgard and Kringlen (1976), who found only slightly higher concordances for criminality in MZ twins than in DZ twins, the early twin studies found genetic influences to be of substantial magnitude for criminality, but not for juvenile delinquency.

Nineteen recent twin and adoption studies examining antisocial behavior also were excluded because they did not provide enough information for the calculation of effect sizes. For all of these studies, the information needed for the meta-analysis was found in other publications that analyzed data from the same sample. These excluded studies usually examined more complex issues (e.g., Cadoret, Cain, & Crowe, 1983, genotype–environment interaction; Langbehn, Cadoret, Yates, Troughton, & Stewart, 1998, relationship between CD and oppositional defiant disorder symptoms and adult antisocial behavior; Reiss et al., 1995, parenting style).

Assessment of Related Disorders

In several studies, another variable related to antisocial behavior (e.g., alcoholism, somatization disorder, or other personality disorders) was studied in addition to antisocial behavior. For example, one adoption study (Schulsinger, 1972) examined the aggregate risk for psychopathy, criminality, alcoholism, drug abuse, or mental illness in adoptees of biological parents with psychopathy and biological parents who did not have psychopathy. This means that some adoptees who do not engage in antisocial behavior could have been counted as "affected" because of their problems with alcohol or drug abuse (i.e., variables outside the scope of this meta-analysis). Such studies were not included because the assessment of other disorders interfered with the assessment of antisocial behavior (e.g., alcoholism or drug abuse being counted as antisocial behavior). Six studies were excluded from the meta-analysis because of assessment of related disorders.

Nonindependent Samples

Another justification for exclusion from the meta-analysis was nonindependent sampling. Several effect sizes from studies in the original reference list were from nonindependent samples as a result of several factors. Some authors published the same data in two different sources (e.g., Mednick et al., 1983; Mednick, Gabrielli, & Hutchings, 1984). In such cases, we only considered one of the studies for the meta-analysis. The other three factors leading to nonindependent samples are more complicated. First, some authors of a single publication examined more than one dependent measure of antisocial behavior in their sample (e.g., Ghodsian-Carpey & Baker, 1987). Second, several publications were a collection of follow-up data of the same sample (e.g., Cadoret, 1978; Cadoret, Troughton, O'Gorman, & Heywood, 1986). Third, several authors (in different publications) examined different dependent measures in the same sample (e.g., Grove et al., 1990; Tellegen et al., 1988).

Experts on meta-analysis have several suggestions for dealing with nonindependent samples (Mullen, 1989; Rosenthal, 1991). For example, Mullen gave four options for dealing with this problem: choosing the best dependent measure, averaging the effect sizes of the different dependent measures, conducting separate meta-analyses for each of the dependent measures, or using nonindependent samples as if they were independent samples (the least recommended approach). We did not follow the option of choosing the best dependent measure, unless one of the dependent measures did not fulfill the inclusion criteria described above, making the decision easy. This option was not taken in order to avoid making subjective choices, because we were aware of the effect sizes associated with each of the dependent measures. The option of conducting separate meta-analyses for each of the dependent measures was not chosen simply as a practical matter, because there were a large number of effect sizes from nonindependent samples. Therefore, the most viable option was to average the effect sizes from nonindependent samples.

In model-fitting analyses, the sample size must be indicated. Therefore, the option of averaging multiple effect sizes was used in cases in which the

sample size was identical across the nonindependent samples. If the sample size was not identical across the nonindependent samples, the effect size from the largest sample was used. More specifically, in cases of nonindependence in which the same dependent measure was used in the same sample multiple times (e.g., in follow-up analyses), the effect size estimated from the largest sample was chosen. In cases of nonindependence in which different dependent measures were used in the same sample (e.g., the author of one publication examining more than one dependent measure or authors of different publications examining different dependent measures in one sample), the effect sizes were averaged if the sample size was the same across the nonindependent samples, and the effect size from the largest sample was used if the sample size differed across the nonindependent samples.

When choosing the effect size from the largest sample, we made this decision without regard to other factors with two exceptions. M. O'Connor, Foch, Sherry, and Plomin (1980) and Plomin et al. (1981) studied the same Colorado twin sample. Although O'Connor et al.'s (1980) sample was larger by 2 more twin pairs, Plomin et al. (1981) was included in the meta-analysis instead. Plomin et al.'s (1981) study is the only study to examine an objective measure of aggression (except for Plomin and Foch, 1980, which also used the same sample), so it was important to include the study in the examination of the potential moderating effect of the assessment method. Tellegen et al. (1988) and Lykken, Tellegen, and DeRubeis (1978) reported results for the same aggression measure on the same sample. Although Lykken et al.'s sample was larger, Tellegen et al. was included instead, as Lykken et al. focused on the methodological issue of volunteer sampling and did not report information regarding two potential moderating variables (i.e., zygosity determination method and age), whereas this information was included in Tellegen et al.

In several cases, it was unclear whether several studies reported results from the same sample (e.g., the Minnesota Twin Family Study). Several pieces of information, including the year of the publication, the age of the sample, and the description of the sample were used to decide whether two studies actually used the same sample. In some studies (e.g., Parker, 1989, as cited in Carey, 1994), this decision was impossible to make because a description of the sample was not reported. The assumption of nonindependent sampling was made for these studies.

In Tables 3 and 4, the studies using the same samples are grouped together. The Inclusion–exclusion column indicates whether the study's effect sizes were included or excluded. "Included—averaged" indicates an effect size that was included in the meta-analysis after using the averaging method (i.e., averaging effect sizes with the same associated sample size) of dealing with nonindependence. "Included—largest" indicates an effect size that was included in the meta-analysis after using the largest sample method (i.e., simply choosing the effect size associated with the largest sample size) of dealing with nonindependence. "Included—independent" indicates an effect size that was included in the meta-analysis because the study does not have a nonindependence problem. "Excluded" indicates an effect size that was excluded from the meta-analysis because the same sample was examined in another study that was larger, more unique in assessment method, or better described.

Analyses

Determination of the Effect Size

Some adoption and twin studies used a continuous variable to measure antisocial behavior and reported either Pearson product–moment or intraclass correlations, which were the effect sizes used from these studies in the meta-analysis. In other studies, a dichotomous variable was used, and concordances, percentages, or a contingency table (including the number of twin pairs with both members affected, one member affected, and neither member affected) was reported. The information from the concordances or percentages was transformed into a contingency table, which was then used to estimate the tetrachoric correlation (i.e., the correlation between the latent continuous variables that are assumed to underlie the observed dichotomous variables). For these studies, the tetrachoric correlation was the effect size used in the meta-analysis.

For some studies, we directly estimated the tetrachoric correlation from the raw data either because we had access to the data (Slutske, Heath, et al., 1997; Waldman, Levy, & Hay, 1995; Waldman et al., in press) or because the tetrachoric correlation had to be estimated from contingency tables. For these studies, we were also able to estimate the weight matrix (i.e., the asymptotic covariance matrix of the correlation matrix). If the weight matrix can be estimated, it is possible to use weighted least squares (WLS) estimation, which is more appropriate for non-normally distributed variables like diagnoses of CD or ASPD, rather than maximum-likelihood (ML) estimation, in the model-fitting analyses.

One assumption of model-fitting analyses is that the variable being analyzed is normally distributed. Although we do not have access to the distributions of the variables being examined in the studies included in the meta-analysis, violation of the normal distribution assumption in studies examining antisocial behavior is often a problem. Typically, the distribution is positively skewed (i.e., inverse J-shaped) because the majority of the population exhibits little or no antisocial behavior. WLS estimation is preferable to ML estimation for obtaining asymptotically correct standard errors of parameter estimates and chi-square goodness-of-fit tests when the normal distribution assumption cannot be met or when correlations (rather than covariances) are analyzed (Neale & Cardon, 1992). For most of the studies included in the meta-analysis, however, we did not have access to the raw data and were limited to the published information. This meant that for these studies, we were limited to analyzing Pearson product–moment or intraclass correlations, and ML rather than WLS estimation had to be used.

Model-Fitting Analyses

The magnitude of additive genetic influences (a^2) and that of nonadditive genetic influences (d^2) constitute the amount of variance in the liability for antisocial behavior that is due to genetic differences among individuals. If genetic influences are additive, this means that the effects of alleles from different loci are independent and "add up" to influence the liability for a trait. If genetic influences are nonadditive, this means that alleles interact with each other to influence the liability for a trait, either at a single genetic locus (i.e., dominance) or at different loci (i.e., epistasis). Shared environmental influences (c^2) represent the amount of liability variance that is due to environmental influences that are experienced in common and make family members similar to one another, whereas nonshared environmental influences (e^2) represent the amount of liability variance that is due to environmental influences that are experienced uniquely and make family members different from one another.

It is customary in contemporary behavior genetic analyses to compare alternative models, containing different sets of causal influences, for their fit to the observed data (i.e., twin or familial correlations or covariances). These models posit that antisocial behavior is affected by the types of influences described above: additive genetic influences (A), shared environmental influences (C), nonadditive genetic influences (D), and nonshared environmental influences (E). In the present meta-analysis, the ACE model, the AE model, the CE model, and the ADE model were compared. It is not possible to estimate c^2 and d^2 simultaneously or test an ACDE model with data only from twin pairs reared together because the estimation of c^2 and d^2 both rely on the same information (i.e., the difference between the MZ and DZ twin correlations). If the DZ correlation is greater than half of the MZ correlation, the ACE model is the correct model and the estimate of d^2 in the ADE model is always zero. If the DZ correlation is less than half of the MZ correlation, the ADE model is the correct model and the estimate of c^2 in the ACE model is always zero. If another type of data, such as the correlations between adoptees and their adoptive and biological parents, also is included in the analyses, this provides another source of information for the estimation of c^2 and the ACDE model can be

tested. Given that the ACDE model can be tested only when both twin and adoption studies are included in the analysis, it was only possible to estimate c^2 and d^2 simultaneously when analyzing all of the data included in the meta-analysis. For other analyses (i.e., the comparison of including and excluding weight matrices, the comparison between twin and adoption studies, and the tests of moderators), both twin and adoption studies were not always available across different types of studies. Therefore, we were limited to comparing the ACE, AE, CE, and ADE models for analyses other than those that included all data included in the meta-analysis.

Two types of adoption studies and two types of twin studies were included in the meta-analysis. The adoption studies provided data on the comparison of the correlation between adoptees and their adoptive parents versus the correlation between adoptees and their biological parents (i.e., parent–offspring adoption studies) and the comparison of the correlation between adoptive siblings and the correlation between biological siblings (i.e., sibling adoption studies). Data from both studies of twin pairs reared together and twin pairs reared apart were included. The effect sizes (i.e., Pearson or intraclass correlations or the tetrachoric correlations plus the weight matrices) from each study were entered in separate groups in the model-fitting program Mx (Neale, 1995). Stem and leaf plots of the effect sizes from the adoption studies and the twin studies are shown in Tables 5 and 6, respectively. In the model-fitting program, the correlations between pairs of relatives are explained in terms of the components of variance that are shared between the relatives. These can include A, or additive genetic influences; C, or shared environmental influences; and D, or nonadditive genetic influences. Nonshared environmental influences, or E, do not explain any part of the correlation between the pairs of relatives because, by definition, nonshared environmental influences are not shared between relatives. The correlation between different types of relatives is explained by different sets of influences and their appropriate weights as shown in Appendix B. These weights reflect the genetic or environmental similarity between pairs of relatives. For example, the correlation between an adoptee and his or her adoptive parent is explained only by shared environmental influences (1*C), whereas the correlation between an adoptee and his or her biological parent is explained only by additive genetic influences (.5*A).

The example Mx script in Appendix C shows how an analysis was set up to test an ACDE model, and Figure 1 shows the path diagram for the ACDE model. In Appendix C, Group 1 defines the parameters of the model: a^2 (additive genetic influences), c^2 (shared environmental influences), d^2 (nonadditive genetic influences), and e^2 (nonshared environmental influences). Groups 2 to 9 show how the correlation matrix for each type of relative pair (adoptee and biological parent, adoptee and adoptive parent, biological siblings, adoptive siblings, MZ twins reared together, DZ twins reared together, MZ twins reared apart, and DZ twins reared apart) is defined in the Mx script according to the information shown in Appendix B. For each study, the effect size, or the correlation matrix for each type of relative pair (e.g., MZ twin pairs and DZ twin pairs), is listed in a separate group. If a study listed separate correlation matrices for independent groups (e.g., males and females, younger children and older children), these correlation matrices were listed in separate groups.

In analyzing behavior genetic data for two generations, as in the parent–offspring adoption studies, it is important to consider the possibility of estimating separate a^2 and c^2 values for children and parents because a^2 and c^2 estimates may differ across the generations. Unfortunately, the adoptee–adoptive parent and adoptee–biological parent correlations do not provide enough information for such analyses.

In the parent–offspring adoption studies, a problem of nonindependence exists because the same adoptees are in the adoptee–adoptive parent groups and the adoptee–biological parent groups. Therefore, the adoptee–adoptive parent data were included only in comparisons between the twin studies and the two types of adoption studies.

Analyses of All Data

The analyses were first conducted for all data, including the two types of twin studies and the two types of adoption studies. The ACDE model, the ACE model, the AE model, the CE model, and the ADE model were compared. The fit of each model, as well as of competing models, was assessed using both the chi-square statistic and the Akaike information criterion (AIC), a fit index that reflects both the fit of the model and its parsimony (Loehlin, 1992b). The AIC has been used extensively in both the structural equation modeling and behavior genetics literatures. Among competing models, that with the lowest AIC and the lowest chi-square value relative to its degrees of freedom is considered to be the best fitting model.

Assessment of Possible Outliers and High-Influence Studies

We examined the possibility that certain studies may be outliers or exert undue influence on the results by analyzing the data both including and excluding these studies. Specifically, we reanalyzed the data both including and excluding studies with construct validity concerns—that is, studies examining psychopathy (eight samples) or using measures with questionable validity (two samples)—to examine the sensitivity of the results to the effects from these studies. The data also were analyzed both including and excluding the Centerwall and Robinette (1989) study for three reasons. First, there was a much larger difference between the MZ and DZ correlations as compared with other studies included in the meta-analysis, thus raising the possibility that the study represented an outlier. Second, with almost 10,000 participants, this study was far larger than any other study, which meant that it could exert undue influence on the results. Third, this study used an unusual operationalization for criminality (i.e., dishonorable discharge from the military).

Table 5
Stem and Leaf Plot of the Effect Sizes (Correlations) in Adoption Studies

Adoptee–biological parent		Adoptee–adoptive parent		Biological siblings		Adoptive siblings	
Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf
.5	3	.5		.5	2	.5	
.4	0 9	.4		.4	2 6	.4	
.3		.3		.3	1	.3	7
.2		.2		.2		.2	
.1	0 2 4 6 7	.1		.1		.1	1 1 9
.0	0	.0	1 9	.0		.0	0
−.0		−.0	2	−.0		−.0	

Table 6
Stem and Leaf Plot of the Effect Sizes (Correlations) in Twin Studies

MZ twin pairs		DZ twin pairs		MZ twin pairs reared apart		DZ twin pairs reared apart	
Stem	Leaf	Stem	Leaf	Stem	Leaf	Stem	Leaf
.9	0	.9		.9		.9	
.8	0 1 1 4 3 5	.8	0	.8		.8	
.7	0 0 0 2 4 4 4 4 6 6 8 9	.7		.7		.7	
.6	1 2 2 3 6 6 6 7 7 8	.6	0 0 2 2 2 4	.6	2	.6	
.5	2 2 2 4 5 7 8 9 9	.5	0 0 1 2 2 3 6 6 7 9	.5		.5	
.4	2 3 3 3 4 5 6 8	.4	0 2 2 2 4 4 5 6 6 7 7 7 8 8 8 9 9 9	.4		.4	
.3	1 2 3 5 6 7 9	.3	0 0 1 4 4 4 6 7 8	.3		.3	
.2	2 4 9 9	.2	0 1 2 2 3 5 5 7 9	.2		.2	
.1		.1	0 1 2 2 2 3 4 4 5 6 6 7 8 9	.1	0	.1	4
.0	9	.0	0	.0		.0	
−.0		−.0	8	−.0		−.0	
−.1		−.1		−.1		−.1	
−.2		−.2	4	−.2		−.2	

Note. MZ = monozygotic; DZ = dizygotic.

Assessment of Potential Moderators

We examined whether operationalization (i.e., diagnoses, criminality, aggression, and antisocial behavior), assessment method (i.e., self-report, report by others, objective test, reaction to aggressive material, and records), zygosity determination method (i.e., blood typing, questionnaire, and a combination of the two), sex (i.e., male, female, and both or opposite sex), and age (i.e., children, adolescents, and adults) were significant moderators by contrasting the fit of a model in which the parameter estimates are constrained to be equal across levels of the relevant variables to the fit of a model in which the parameter estimates are free to vary across levels of the relevant variables on the same dataset. If the fit of the two models is significantly different, this indicates the significance of the moderator. It is possible that a nonsignificant result may be due to lack of power, especially if there is little variability in the levels of a moderator.

Assessment of Confounding Among Moderators

When testing a moderator for significance, one tests whether estimating separate parameter estimates (e.g., a^2, c^2, and e^2) for studies at each level of the moderator leads to a better fit than when the parameter estimates are constrained to be equal across the different levels of the moderator. When testing for one moderator's significance after another moderator has been statistically controlled for, one tests whether estimating separate parameter estimates for studies at each level of both moderators leads to a better fit than estimating separate parameter estimates for studies at each level of only one of the moderators. For example, when examining whether assessment method is a significant moderator after the effects of operationalization have been statistically controlled for, one compares two models. In the first model, parameter estimates are allowed to vary only across the four operationalizations. This model is compared with a second, less restrictive model, in which parameter estimates are allowed to vary across both the four operationalizations and the five assessment methods conjointly. If the fit of the second model (i.e., with both operationalization and assessment method as moderators) is significantly better than the fit of the first model (i.e., with only operationalization as a moderator), this indicates that assessment method is a significant moderator even after the effects of operationalization as a moderator are statistically controlled.

Comparisons Between Twin and Adoption Studies

Three comparisons between the twin studies and the two types of adoption studies were conducted. Twin studies were not divided into the two types (i.e., twin pairs reared together and twin pairs reared apart), given that there were only two samples of twin pairs reared apart. First, twin studies were compared with all adoption studies. Second, twin studies were compared with adoption studies examining adoptees and their adoptive or biological parents (i.e., parent–offspring adoption studies). Third, twin studies were compared with adoption studies examining adoptive and biological siblings (i.e., sibling adoption studies). These comparisons were made by contrasting the fit of two models: (a) a model in which the parameter estimates are constrained to be equal across the twin and adoption studies and (b) a model in which the parameter estimates are free to vary across the twin and adoption studies. If the fit of the two models is significantly different, this would indicate that the estimates of genetic and environmental influences from twin and adoption studies are significantly different. Note that this is the same procedure used for the assessment of potential moderators that was described above.

Effect of Excluding Weight Matrices

As discussed above, we did not have access to the raw data for most of the studies and were limited to analyzing the data published in the studies (i.e., Pearson product–moment correlations or intraclass correlations), which meant that ML estimation had to be used rather than the preferred WLS estimation. Although there was no other option, this is a limitation of the meta-analysis, given that WLS estimation is more appropriate than ML estimation when the normal distribution assumption is violated and when correlations rather than covariances are analyzed (Neale & Cardon, 1992). In order to examine the potential effects of using ML estimation rather than WLS estimation on the results, we examined the effects of excluding the weight matrices in two ways. First, in the studies with estimated weight matrices, the data were analyzed both including and excluding the weight matrices. Second, we contrasted the results from studies with the estimated weight matrices with those from studies for which the estimation of weight matrices was not possible. This contrast was tested by comparing the fit of the model in which the parameter estimates were constrained across the two types of studies with the fit of the model in which the parameter estimates were free to vary across the two types of studies.

Results

Analyses of All Data

In this section, the number of *samples* refers to the number of independent studies in the analyses. The number of *groups* refers

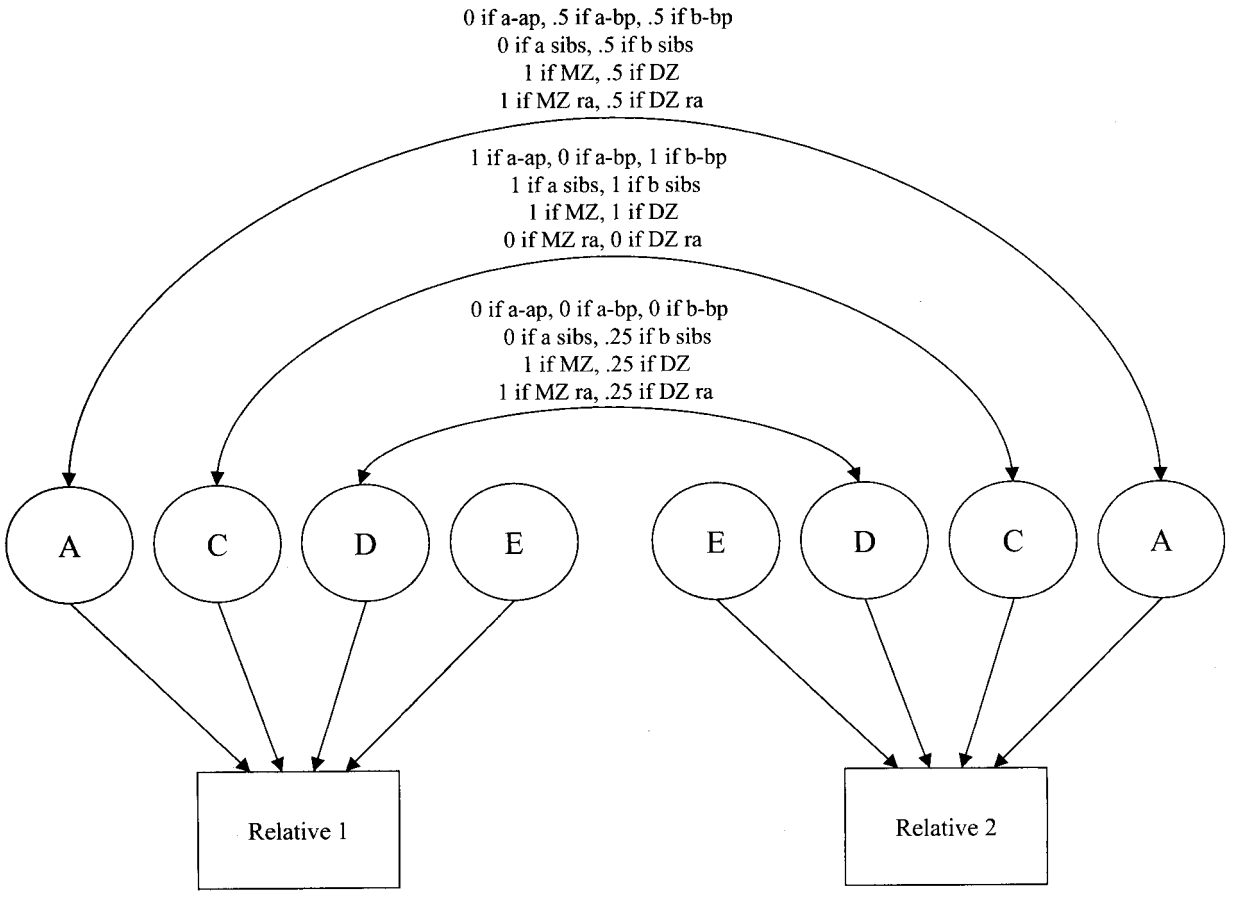

Figure 1. ACDE model. A = additive genetic influences; C = shared environmental influences; D = nonadditive genetic influences; E = nonshared environmental influences; a-ap = adoptee–adoptive parent pairs; a-bp = adoptee–biological parent pairs; b-bp = biological child–biological parent pairs; a sibs = adoptive sibling pairs; b sibs = biological sibling pairs; MZ = monozygotic twin pairs reared together; DZ = dizygotic twin pairs reared together; MZ ra = monozygotic twin pairs reared apart; DZ ra = dizygotic twin pairs reared apart.

to the total number of independently analyzed units in the samples. For example, Slutske, Heath, et al. (1997) and Torgersen, Skre, Onstad, Edvardsen, and Kringlen (1993) examined two independent samples, the Australian adult twins and the Norwegian twins. There are five groups (male–male MZ twin pairs, male–male DZ twin pairs, female–female MZ twin pairs, female–female DZ twin pairs, and male–female DZ twin pairs) in Slutske, Heath, et al. and two groups (MZ twin pairs and DZ twin pairs) in Torgersen et al. Therefore, if an analysis is conducted using data from Slutske, Heath, et al. and Torgersen et al., there would be two samples and seven groups in the analysis.

The results of analyses of the data from all of the samples meeting the inclusion criteria (N = 52 samples, 149 groups, 55,525 pairs of participants) are presented in Table 7. The full ACDE model fit best as compared with the other, more restrictive models. Excluding possible outliers—that is, studies that examined psychopathy (8 samples), and studies using measures with questionable validity (2 samples), and the Centerwall and Robinette (1989) study—did not alter the results of the meta-analysis, as parameter estimates did not differ after excluding these studies. (The specific results can be obtained from the authors.)

Assessment of Potential Moderators

Table 8 shows the results of analyses examining operationalization, assessment method, zygosity determination method, sex, and age as moderators of the magnitude of genetic and environmental influences on antisocial behavior. The chi-square difference between a model in which the parameter estimates are constrained to be equal and a model in which the parameter estimates are free to vary across the different levels of the moderator is shown for each moderator.

Operationalization

The chi-square difference test is significant for operationalization, indicating significant differences in the magnitude of genetic and environmental influences on diagnosis (14 samples, 40 groups, 11,681 pairs of participants), criminality (5 samples, 13 groups, 34,122 pairs of participants), aggression (14 samples, 40 groups, 4,408 pairs of participants), and antisocial behavior (15 samples, 48 groups, 4,365 pairs of participants), $\Delta\chi^2(9, N = 54{,}576) = 339.87$, $p < .01$. The ACE model was the best fitting model for diagnosis (a^2 = .44, c^2 = .11, e^2 = .45),

Table 7
Standardized Parameter Estimates and Fit Statistics—Inclusion of All Data

	Parameter estimate				Fit statistic			
Model	a^2	c^2	e^2	d^2	χ^2	*df*	*p*	AIC
ACE	.38	.18	.44	—	1,420.38	147	<.001	1,126.38
AE	.55	—	.45	—	1,707.89	148	<.001	1,411.89
CE	—	.45	.55	—	2,364.90	148	<.001	2,068.90
ADE	.41	—	.42	.17	1,590.58	147	<.001	1,296.58
ACDE	.32	.16	.43	.09	1,394.46	146	<.001	1,102.46

Note. Dashes indicate that data are not applicable. a^2 = the magnitude of additive genetic influences (A); c^2 = the magnitude of shared environmental influences (C); e^2 = the magnitude of nonshared environmental influences (E); d^2 = the magnitude of nonadditive genetic influences (D); AIC = Akaike information criterion.

aggression (a^2 = .44, c^2 = .06, e^2 = .50), and antisocial behavior (a^2 = .47, c^2 = .22, e^2 = .31), whereas the ADE model was the best fitting model for criminality (a^2 = .33, d^2 = .42, e^2 = .25). Within the operationalization of diagnosis, significant differences were found between studies examining ASPD (8 samples, 17 groups, 5,019 pairs of participants) and CD (5 samples, 22 groups, 6,560 pairs of participants). Although the magnitude of shared environmental influences was similar, the a^2 estimate was higher in studies examining CD (a^2 = .50, c^2 = .11, e^2 = .39), whereas the e^2 estimate was higher in studies examining ASPD (a^2 = .36, c^2 = .10, e^2 = .54).

The possible effects of confounding between operationalization and assessment method and between operationalization and age should be considered when interpreting these results (see Tables 1 and 2). Parent report was more frequently used in studies examining antisocial behavior than in studies examining diagnosis or

Table 8
Standardized Parameter Estimates and Fit Statistics for the Best Fitting Models—Test of Moderators

	Fit statistic			
Moderator	χ^2	*df*	*p*	AIC
Operationalization				
Parameters constrained to be equal	1,406.50	139	<.001	1,128.50
Parameters free to vary	1,066.63	130	<.001	806.63
Chi-square difference test	339.87	9	<.001	321.87
Assessment method				
Parameters constrained to be equal	1,361.73	139	<.001	1,083.73
Parameters free to vary	530.47	128	<.001	274.47
Chi-square difference test	831.26	11	<.001	809.26
Zygosity determination method				
Parameters constrained to be equal	1,305.79	110	<.001	1,085.79
Parameters free to vary	945.65	104	<.001	737.65
Chi-square difference test	360.14	6	<.001	348.14
Age				
Parameters constrained to be equal	1,351.30	133	<.001	1,085.30
Parameters free to vary	1,107.35	127	<.001	853.35
Chi-square difference test	243.95	6	<.001	231.95
Sex (studies examining one sex or both sexes: males, females, and both)				
Parameters constrained to be equal	1,420.38	147	<.001	1,126.38
Parameters free to vary	1,383.43	141	<.001	1,101.43
Chi-square difference test	36.95	6	<.001	24.95
Sex (studies examining one sex or both sexes: males and females)				
Parameters constrained to be equal	1,057.03	76	<.001	905.03
Parameters free to vary	1,037.67	73	<.001	891.67
Chi-square difference test	19.36	3	<.001	13.36
Sex (studies examining both sexes: males and females)				
Parameters constrained to be equal	870.61	66	<.001	738.61
Parameters free to vary	869.07	63	<.001	743.07
Chi-square difference test	1.53	3	.68	−4.47

Note. AIC = Akaike information criterion.

aggression, and there were more studies examining antisocial behavior in children and adolescents than studies examining antisocial behavior in adults. Also, all of the behavior genetic studies of criminality were those examining adults using the assessment method of official records. The specific comparison between studies examining the diagnoses of ASPD and CD showed that the magnitude of genetic influences was higher for CD, whereas the magnitude of nonshared environmental influences was higher for ASPD. These results may be explained by age differences (ASPD being assessed in adulthood and CD being assessed in childhood) or differences in assessment method (self-report being used more often to assess ASPD and parent report being used more often to assess CD).

Assessment Method

The chi-square difference test indicates that assessment method is a moderator of the magnitude of genetic and environmental influences on antisocial behavior, $\Delta\chi^2(11, N = 54{,}533) = 831.26$, $p < .01$. Self-report (23 samples, 69 groups, 13,329 pairs of participants), report by others (14 samples, 51 groups, 6,851 pairs of participants), records (5 samples, 13 groups, 34,122 pairs of participants), reaction to stimuli (2 samples, 6 groups, 146 pairs of participants), and objective assessment (1 sample, 2 groups, 85 pairs of participants) were compared. The ACE model was the best fitting model for self-report ($a^2 = .39$, $c^2 = .06$, $e^2 = .55$) and report by others ($a^2 = .53$, $c^2 = .22$, $e^2 = .25$), whereas the AE model was the best fitting model for reaction to aggressive stimuli ($a^2 = .52$, $e^2 = .48$). All of the studies using the assessment method of records were also studies examining criminality, and the ADE model was the best fitting model ($a^2 = .33$, $d^2 = .42$, $e^2 = .25$). Model fitting could not be conducted for the assessment method of objective test because of lack of information (i.e., only one study used an objective test).

Caution is recommended in interpreting these results, given that only one study (Plomin et al., 1981) used an objective test, and only two studies (Owen & Sines, 1970; G. D. Wilson, Rust, & Kasriel, 1977) used reaction to aggressive material. Also, all of the studies using the assessment method of records were studies examining the operationalization of criminality. When the assessment methods of self-report and report by others were compared, the magnitude of familial influences (a^2 and c^2) was higher for report by others than for self-report. These results differ slightly from the conclusions of Miles and Carey (1997), who found lower a^2 and higher c^2 estimates for parent reports than for self-reports of aggression. Again, the possibility of confounding between moderators should be considered. Studies using the assessment method of self-report were more likely to be those examining the operationalization of diagnosis in adults or adolescents, whereas studies using the assessment method of parent report were more likely to be those examining the operationalization of antisocial behavior in children.

Zygosity Determination Method

The chi-square difference test indicates that zygosity determination method is a significant moderator, as the magnitude of genetic and environmental influences differed significantly for studies using blood grouping (8 samples, 18 groups, 1,020 pairs of participants), a combination of blood grouping and the questionnaire method (15 samples, 55 groups, 27,631 pairs of participants), and the questionnaire method (11 samples, 39 groups, 8,249 pairs of participants), $\Delta\chi^2(6, N = 36{,}900) = 360.14$, $p < .01$. The ADE model was the best fitting model for studies using blood grouping ($a^2 = .14$, $d^2 = .33$, $e^2 = .53$), whereas the ACE model was the best fitting model for studies using the questionnaire method ($a^2 = .43$, $c^2 = .27$, $e^2 = .30$) and a combination of the two methods ($a^2 = .39$, $c^2 = .11$, $e^2 = .50$).

These parameters estimates are difficult to interpret, given that studies using the most stringent method of zygosity determination (i.e., blood grouping) and the least stringent method of zygosity determination (i.e., questionnaire) yielded higher estimates of genetic influences (broad $h^2 = .43$ to $.47$) than studies using a combination of the two methods (broad $h^2 = .39$).

Age

The chi-square difference test indicates that age is a significant moderator and that the magnitude of genetic and environmental influences on antisocial behavior in children (15 samples, 54 groups, 7,807 pairs of participants), adolescents (11 samples, 31 groups, 2,868 pairs of participants), and adults (17 samples, 50 groups, 27,671 pairs of participants) is significantly different, $\Delta\chi^2(6, N = 38{,}346) = 243.95$, $p < .01$. The ACE model was the best fitting model for children ($a^2 = .46$, $c^2 = .20$, $e^2 = .34$), adolescents ($a^2 = .43$, $c^2 = .16$, $e^2 = .41$), and adults ($a^2 = .41$, $c^2 = .09$, $e^2 = .50$). The magnitude of familial influences (a^2 and c^2) decreased with age, whereas the magnitude of nonfamilial influences (e^2) increased with age.

These results should be interpreted with caution for two reasons. First, although many studies examined a wide age range, either the mean or the midpoint age had to represent this age range, given that access to the raw data for each study was not possible. Second, age was simplified into a categorical variable (i.e., children, adolescents, and adults) in our meta-analysis, given the limitations of including continuous moderators in model-fitting analyses. As age increased, the magnitude of familial influences (i.e., both a^2 and c^2) decreased. These findings for behavior genetic studies of antisocial behavior differ somewhat from the general finding in the behavior genetics literature (Loehlin, 1992a; Plomin, 1986) that a^2 and e^2 estimates increase and c^2 estimates decrease with increasing age. These findings also differ from Miles and Carey's (1997) conclusion that a^2 estimates increase and c^2 estimates decrease with age. The confounding among moderators should again be considered in interpreting our results. The same pattern of results found for age was found for assessment method, with studies using report by others (viz., used more with children) yielding higher estimates of familial influences than those using self-report (viz., used more with adolescents and adults), and for operationalization, with studies examining antisocial behavior (viz., assessed more in children) yielding higher estimates of familial influences than those examining diagnosis (viz., assessed more in adults and adolescents).

The results were not consistent with L. F. DiLalla and Gottesman's (1989) hypothesis given that the magnitude of genetic influences was lower in both adolescence and adulthood than in childhood, but again, the presence of confounding among the moderators should be considered. Unfortunately, it is difficult to

interpret the results of analyses examining age as a moderator after statistically controlling for assessment method because only one study examining children used self-report and only two studies examining adolescents used parent report.

Sex

The chi-square difference test examining the differences among studies examining males (21 samples, 42 groups, 22,521 pairs of participants), females (19 samples, 38 groups, 7,375 pairs of participants), and both sexes or opposite-sex pairs (41 samples, 69 groups, 25,629 pairs of participants) was significant, $\Delta\chi^2(6, N = 55{,}525) = 36.95$, $p < .01$. The ACE model was the best fitting model for males ($a^2 = .38$, $c^2 = .17$, $e^2 = .45$), females ($a^2 = .41$, $c^2 = .19$, $e^2 = .40$), and both sexes/opposite-sex pairs ($a^2 = .35$, $c^2 = .17$, $e^2 = .48$). The magnitude of familial influences (a^2 and c^2) was higher in same-sex twin pairs ($a^2 = .39$, $c^2 = .18$, $e^2 = .43$) than in data including both sexes or opposite-sex twin pairs ($a^2 = .35$, $c^2 = .17$, $e^2 = .48$). These results support Cloninger, Christiansen, Reich, and Gottesman's (1978) conclusion that although many of the etiologic factors that influence antisocial behavior in males and females are shared in common, they are not fully identical. The difference between males and females also was significant, $\Delta\chi^2(3, N = 29{,}896) = 19.36$, $p < .01$, indicating that the a^2 and the c^2 estimates are higher in females. These results are not consistent with those of Miles and Carey (1997), who found higher heritability estimates for aggression in males.

Given the fact that several studies examined only one sex and the fact that these studies varied a great deal in the operationalization examined (e.g., dishonorable discharge for males and aggression for females) and the assessment method used (e.g., official records for males and parent report for females), the comparison of results for males and females was repeated after excluding these studies (see Tables 3 and 4 for studies including only one sex). When the analyses were limited to studies that examined antisocial behavior in both males (17 samples, 34 groups, 5,610 pairs of participants) and females (17 samples, 34 groups, 7,225 pairs of participants)—that is, when studies examining antisocial behavior in only one sex were excluded—the difference between males ($a^2 = .43$, $c^2 = .19$, $e^2 = .38$) and females ($a^2 = .41$, $c^2 = .20$, $e^2 = .39$) was no longer significant, $\Delta\chi^2(3, N = 12{,}835) = 1.53$, $p = .68$. This result is consistent with those of traditional literature reviews (e.g., Widom & Ames, 1988) in which the authors have concluded that the magnitude of genetic and environmental influences on antisocial behavior in males and females is similar.

Assessment of Confounding Among Moderators

The possibility of confounding was assessed between the following pairs of moderators: operationalization and assessment method, age and operationalization, and age and assessment method. All analyses showed that each moderator is significant even after the effects of the possible confounding moderator are controlled for statistically. For example, the model estimating separate parameter estimates for each level of operationalization and each level of assessment method fit significantly better than the model estimating separate estimates for each level of operationalization only, $\Delta\chi^2(13, N = 54{,}122) = 633.67$, $p < .001$, and the model estimating separate estimates for each level of assessment method only, $\Delta\chi^2(12, N = 54{,}122) = 112.56$, $p < .01$. This result indicates that assessment method is a significant moderator after controlling statistically for the effects of operationalization as a moderator, and that operationalization is a significant moderator after controlling statistically for the effects of assessment method as a moderator. Similarly, assessment method was a significant moderator after controlling for age, $\Delta\chi^2(7, N = 38{,}071) = 676.28$, $p < .01$; operationalization was a significant moderator after controlling for age, $\Delta\chi^2(18, N = 37{,}935) = 410.52$, $p < .01$; and age was a significant moderator after controlling for operationalization, $\Delta\chi^2(15, N = 37{,}935) = 335.44$, $p < .01$, and after controlling for assessment method, $\Delta\chi^2(7, N = 38{,}071) = 102.73$, $p < .01$.

Comparisons Between Twin and Adoption Studies

Comparisons of the results from twin (42 samples, 131 groups, 37,700 pairs of participants) and adoption studies (10 samples, 21 groups, 31,272 pairs of participants) are presented in Table 9. Twin ($a^2 = .45$, $c^2 = .12$, $e^2 = .43$) and adoption ($a^2 = .32$, $c^2 = .05$, $e^2 = .63$) studies yielded different parameter estimates, as there was a significant chi-square difference between the model in which the parameter estimates were constrained to be equal across twin and adoption studies and the model in which the parameter estimates were free to vary for each type of study, $\Delta\chi^2(3, N = 68{,}972) = 119.68$, $p < .01$. Results from twin studies were next compared with results from adoption studies after dividing the adoption studies into two types: (a) studies comparing the correlations between adoptees and their adoptive parents with the correlations between adoptees and their biological parents (i.e., parent–offspring adoption studies; 7 samples, 12 groups, 30,504 pairs of participants) and (b) studies comparing the correlations between adoptive siblings with the correlations between biological siblings (i.e., sibling adoption studies; 3 samples, 9 groups, 768 pairs of participants). There was a significant difference between the results from twin studies ($a^2 = .45$, $c^2 = .12$, $e^2 = .43$) and parent–offspring adoption studies ($a^2 = .31$, $c^2 = .05$, $e^2 = .64$), $\Delta\chi^2(3, N = 68{,}204) = 130.81$, $p < .001$, but not between the results from twin studies and sibling adoption studies ($a^2 = .48$, $c^2 = .13$, $e^2 = .39$), $\Delta\chi^2(3, N = 38{,}468) = 0.75$, $p = .86$. Given the similar results of twin and sibling adoption studies, results from the parent–offspring adoption studies were compared with those from the twin and sibling adoption studies combined (45 samples, 140 groups, 38,468 pairs of participants). The results were found to differ, such that the twin and sibling adoption studies ($a^2 = .44$, $c^2 = .13$, $e^2 = .43$) yielded higher a^2 and c^2 estimates and lower e^2 estimates than the parent–offspring adoption studies ($a^2 = .31$, $c^2 = .05$, $e^2 = .64$), $\Delta\chi^2(3, N = 68{,}972) = 131.65$, $p < .01$.

Effect of Excluding Weight Matrices

Table 10 shows the results of two analyses assessing the effect of excluding the weight matrices. First, it shows the effect of excluding the weight matrices in the samples where the estimation of weight matrices was possible. When the weight matrices were included, the best fitting model was the ACE model ($a^2 = .54$, $c^2 = .28$, $e^2 = .18$), but when the weight matrices were omitted,

Table 9
Standardized Parameter Estimates and Fit Statistics for the Best Fitting Models—Comparison Between Twin and Adoption Studies

Models	Parameter estimate				Fit statistic			
	a^2	c^2	e^2	d^2	χ^2	*df*	*p*	AIC
Comparison between all twin studies and all adoption studies								
Parameters constrained to be equal	.46	.10	.44	—	1,541.06	150	<.001	1,241.06
Parameters free to vary					1,421.38	147	<.001	1,127.38
Twin studies	.45	.12	.43	—	1,355.28	129	<.001	1,097.28
Adoption studies	.32	.05	.63	—	66.10	19	<.001	28.10
Chi-square difference test					119.68	3	<.001	113.68
Comparison between all twin studies and parent–offspring adoption studies								
Parameters constrained to be equal	.46	.10	.44	—	1,531.82	141	<.001	1,249.82
Parameters free to vary					1,401.01	138	<.001	1,125.01
Twin studies	.45	.12	.43	—	1,355.28	129	<.001	1,097.28
Parent–offspring studies	.31	.05	.64	—	45.73	10	<.001	25.72
Chi-square difference test					130.81	3	<.001	124.81
Comparison between all twin studies and sibling adoption studies								
Parameters constrained to be equal	.44	.13	.43	—	1,363.69	138	<.001	1,087.69
Parameters free to vary					1,362.94	135	<.001	1,092.94
Twin studies	.45	.12	.43	—	1,355.28	129	<.001	1,097.28
Sibling adoption studies	.48	.13	.39	—	7.66	7	.36	−6.34
Chi-square difference test					0.75	3	.86	−5.25
Comparison between twin–sibling adoption studies and parent–offspring adoption studies								
Parameters constrained to be equal	.46	.10	.44	—	1,541.06	150	<.001	1,241.06
Parameters free to vary					1,409.41	147	<.001	1,115.41
Twin–sibling adoption studies	.44	.13	.43	—	1,363.69	138	<.001	1,087.69
Parent–offspring studies	.31	.05	.64	—	45.72	10	<.001	25.72
Chi-square difference test					131.65	3	<.001	125.65

Note. Dashes indicate that none of the best fitting models included nonadditive genetic influences. a^2 = the magnitude of additive genetic influences; c^2 = the magnitude of shared environmental influences; e^2 = the magnitude of nonshared environmental influences; d^2 = the magnitude of nonadditive genetic influences; AIC = Akaike information criterion.

the best fitting model was the ADE model (a^2 = .43, d^2 = .26, e^2 = .31). This analysis shows that excluding the weight matrices results in an overestimation of the magnitude of genetic influences and an underestimation of shared environmental influences, although a significance test is not possible (i.e., given that the same data were analyzed in this comparison). Second, Table 10 shows the comparison between studies with and without estimable weight matrices. There was a significant chi-square difference between a model in which all estimates were constrained to be equal and a model in which estimates were free to vary between studies with estimated weight matrices (10 samples, 27 groups, 22,584 pairs of participants) and studies without estimated weight matrices (42 samples, 122 groups, 32,941 pairs of participants), $\Delta\chi^2(3, N = 55{,}525) = 303.68$, $p < .01$. Studies with estimated weight matrices (a^2 = .54, c^2 = .28, e^2 = .18) had higher a^2 and c^2 estimates than studies without estimated weight matrices (a^2 = .35, c^2 = .17, e^2 = .48).

Discussion

Overview of the Results

When all available data from both twin and adoption studies were analyzed together and the magnitude of nonadditive genetic influences was estimated in addition to the magnitude of shared environmental influences, the best fitting model was the ACDE model. On the basis of this analysis, there were moderate additive genetic (a^2 = .32), nonadditive genetic (d^2 = .09), shared environmental (c^2 = .16), and nonshared environmental (e^2 = .43) influences on antisocial behavior.

Operationalization, assessment method, zygosity determination method, and age accounted for significant differences in the genetic and environmental influences on antisocial behavior. Although sex was a significant moderator when data from all studies were examined, there were no statistically significant sex differences in studies that examined both sexes. In the three pairs of moderators that are confounded in the literature (i.e., age and operationalization, age and assessment method, and operationalization and assessment method), each moderator was found to be significant even after the other potentially confounding moderator was controlled for statistically.

Parent–offspring adoption studies showed a lower magnitude of familial influences on antisocial behavior (i.e., lower a^2 and c^2 and higher e^2) than the twin and sibling adoption studies. There are several possible reasons for this result. First, the age difference between the children and their parents may lead to lower correla-

Table 10
Standardized Parameter Estimates and Fit Statistics for the Best Fitting Models—Effect of Excluding Weight Matrices

Model	Parameter estimate				Fit statistic			
	a^2	c^2	e^2	d^2	χ^2	*df*	*p*	AIC
Studies with estimable weight matrices: Weight matrices included and weight matrices omitted								
Weight matrices included	.54	.28	.18	—	66.03	25	<.001	16.03
Weight matrices omitted	.43	—	.26	.31	685.26	25	<.001	635.26
Direct comparison between studies with and without estimable weight matrices								
Parameters constrained to be equal	.38	.18	.44	—	1,420.38	147	<.001	1,126.38
Parameters free to vary					1,116.70	144	<.001	828.70
With weight matrices	.54	.28	.18	—	66.03	25	<.001	16.03
Without weight matrices	.35	.17	.48	—	1,050.67	120	<.001	810.67
Chi-square difference test					303.68	3	<.001	297.68

Note. Dashes indicate that data are not applicable. a^2 = the magnitude of additive genetic influences; c^2 = the magnitude of shared environmental influences; e^2 = the magnitude of nonshared environmental influences; d^2 = the magnitude of nonadditive genetic influences; AIC = Akaike information criterion.

tions, given that there may be age- or cohort-specific genetic and/or environmental influences. This age difference is absent in the twin studies and smaller in the sibling adoption studies. Second, because of the practical obstacles involved in conducting an adoption study, in several studies, different operationalizations and methods of assessment were used for the adoptees and their parents (e.g., criminality via official records for the parents and aggression via self-report for the adoptees).

There was not a statistically significant difference between the results of twin studies and sibling adoption studies. This result should be interpreted while considering the fact that 42 independent twin samples were compared with only 3 independent sibling adoption samples. Although the power to detect a statistically significant difference between the two types of studies may have been limited by the small number of sibling adoption studies, the parameter estimates for the twin studies (a^2 = .45, c^2 = .12, e^2 = .43) and the sibling adoption studies (a^2 = .48, c^2 = .13, e^2 = .39) were very similar.

When data from studies with estimated weight matrices were analyzed both including and excluding the weight matrices, we found that excluding the weight matrices led to an overestimation of the magnitude of genetic influences and an underestimation of the magnitude of shared environmental influences. This suggests that simply using ML estimation without a weight matrix to analyze covariances, as is typical of contemporary twin studies of antisocial behavior, may bias parameter estimates when analyzing data that do not meet the assumption of multivariate normality.

Limitations of the Present Meta-Analysis

Analyses of Correlations Without Weight Matrices

Most of the studies included in the meta-analysis simply reported Pearson or intraclass correlations in their publications, and we were limited to using this information in the meta-analysis. This leads to two major methodological limitations in the meta-analysis.

One assumption of model fitting is that the variances on the outcome measures are equal for the different groups of relatives examined. Given that only correlations are analyzed, there was no way to compare the variances of different types of relatives (e.g., MZ twins vs. DZ twins; twin studies vs. adoption studies) or across other variables such as gender or age. This is an important consideration because there may be genuine differences in the variances of outcome measures across the different groups of relatives. For example, the variance in the antisocial behavior of adoptees may be restricted because antisocial behavior is more common in adoptees than nonadoptees (e.g., Sharma et al., 1998) or because most adoptees are placed in middle-class homes (e.g., Fergusson et al., 1995). Also, given that we were not able to test for differences in variances between MZ and DZ twins, we were not able to examine sibling influences (i.e., cooperation or contrast effects), which have been found to be important in antisocial behavior (e.g., Carey, 1992).

Another significant limitation in analyzing only the correlations reported in the individual studies was the limitation of having to use ML estimation rather than WLS estimation. WLS estimation is preferable to ML estimation for obtaining asymptotically correct standard errors of parameter estimates and chi-square goodness-of-fit tests when the normal distribution assumption is violated or when correlations rather than covariances are analyzed. As stated above, in the present meta-analysis, we found that excluding the weight matrices and using ML estimation led to an overestimation of the magnitude of genetic influences and an underestimation of the magnitude of shared environmental influences. Again, this result suggests that using ML estimation without weight matrices may bias parameter estimates when analyzing data that do not meet the normality assumption.

Effects of Censored Variables

It is possible that many of the scales measuring antisocial behavior fail to distinguish differences among the majority of the population who do not show significant problems with antisocial

behavior. This failure can lead to a "floor effect"—that is, most of the sample having scores close to the lower end of the scale. This type of censoring may be the primary reason that the normality assumption is not met in many of the studies included in the meta-analysis. When variables are censored, correlations in the middle range (i.e., .50 to .60) are decreased more than correlations in the lower range. This means that if the uncensored MZ correlation is in the middle range, the magnitude of genetic influences is underestimated, and if the uncensored DZ correlation is in the middle range, the magnitude of genetic influences is overestimated (van den Oord & Rowe, 1997). Unfortunately, the possible effects of censoring on the results could not be assessed in the present meta-analysis.

Simultaneous Estimation of Shared Environmental Influences and Nonadditive Genetic Influences

The findings of this meta-analysis demonstrate the importance of comparing the results of twin and adoption studies, given the finding of significant differences between twin and parent–offspring adoption studies. Another reason for examining twin and adoption study results simultaneously is the ability to estimate the magnitude of shared environmental influences in the presence of nonadditive genetic influences, and vice versa. We found that the ACDE model, a model that includes both shared environmental influences and nonadditive genetic influences, was the best fitting model when analyzing all of the data included in the meta-analysis. Unfortunately, we were limited to comparing the more restrictive ACE, AE, CE, and ADE models when testing the significance of moderators because both twin and adoption study data were not available for each level of the moderators examined. Given that the ACE model was the best fitting model for most of these analyses, the results may give the false impression that nonadditive genetic influences are unimportant for antisocial behavior. The inability to estimate the magnitude of shared environmental influences and nonadditive genetic influences simultaneously is a limitation of both the twin study design and the adoption study design considered separately. The fact that the ACE model was the best fitting model for most of the analyses examining moderators does not mean that nonadditive genetic influences are unimportant for antisocial behavior.

Future Directions

Examination of Other Operationalizations

Although we were able to contrast the results from a number of different operationalizations of antisocial behavior, further meaningful distinctions in the operationalizations of antisocial behavior should be examined. The results of behavior genetic studies of violent versus nonviolent crime illustrate the importance of this issue. Two adoption studies and one twin study have contrasted violent and nonviolent crimes. Mednick et al. (1984) found that in Danish adopted males, the frequency of property crime was related to the number of convictions of the biological father, whereas the frequency of violent crime was not. Bohman, Cloninger, Sigvardsson, and von Knorring (1982) also found evidence that property crime and violent crime may differ in their etiology. Genetic influences were found to be significant for property crimes, but not for cases of violent crime associated with alcoholism. Cloninger and Gottesman (1987) analyzed the data from the Danish twin sample and found that the heritability for property crimes was .78, whereas the heritability for violent crime was .50. When cross-correlations were examined, they found that there was no genetic overlap between property crime and violent crime, suggesting a distinct and specific etiology for property crime and violent crime. In this meta-analysis, the data on violent and nonviolent crimes could not be analyzed separately because most studies reported results on crime in general.

In the past, researchers have disagreed about the role of genetic influences on delinquency, with some arguing that there are genetic influences on criminality but not on delinquency (e.g., L. F. DiLalla & Gottesman, 1991) and others arguing that there are genetic influences on delinquency as well (e.g., Rowe, 1983). This debate could not be resolved in the present meta-analysis. Many studies with child or adolescent samples did find genetic influences of substantial magnitude for antisocial behavior in general, but no study examined criminality or delinquency in children or adolescents without the inclusion of aggression items. In order to resolve the past debate, new studies on juvenile delinquency (i.e., studies without the inclusion of aggression items or the methodological problems of the early twin studies) are needed.

We were unable to examine another meaningful distinction between two different kinds of aggression, that of relational and overt aggression (Crick, Casa, & Mosher, 1997; Crick & Grotpeter, 1995), because there are no published twin or adoption studies of relational aggression. *Overt aggression* harms others through physical damage or threat of physical damage, whereas *relational aggression* harms others by damaging their peer relationships or reputation (e.g., spreading rumors, excluding them from the peer group). Although relational aggression does not lead to physical harm to the victims, it has serious consequences for both the aggressors (e.g., higher levels of loneliness, depression, and negative self-perceptions, as well as concurrent and future peer rejection; Crick & Grotpeter, 1995) and the victims (e.g., depression, anxiety; Crick & Grotpeter, 1996). The distinction between relational and overt aggression is an especially important consideration when examining sex differences in aggression and its causes, given that females are significantly more relationally aggressive and less overtly aggressive than males (Crick et al., 1997; Crick & Grotpeter, 1995). Given the evidence that overt and relational aggression are correlated but distinct (Crick et al., 1997), future behavior genetic studies of overt and relational aggression should examine the degree of genetic and environmental influences that are common to both types of aggression and specific to each type of aggression.

Validity of the Assessment Method

It was often difficult to make conclusive statements about the moderators examined in the present meta-analysis given concerns regarding the validity of the assessment method. Confounding between assessment method and other moderators was a serious problem, and in some cases, there is convincing evidence that the results reflect the assessment method rather than other moderators that have more conceptual importance, such as operationalization or age.

Given the current evidence, it is not possible to distinguish whether the behavior genetic results on criminality refer to the operationalization of criminality per se or the assessment method of official records, as all of the studies examining criminality used the assessment method of official records. Beyond the problem with confounding, official records also have a validity problem, given that many criminal activities escape detection and therefore do not appear in official records (J. Q. Wilson & Herrnstein, 1985). The additional use of self-reports may lessen this problem, given self-reports' potential to assess criminality in people who are able to escape arrest or incarceration because of intelligence or high social status (Raine & Venables, 1992). Use of self-report alone (e.g., Rowe, 1983), however, also has led to debate regarding the validity of the assessment method (e.g., L. F. DiLalla & Gottesman, 1991).

If the results of studies that examine the same operationalization but use different assessment methods do not agree, questions of validity of the assessment method are raised. In this meta-analysis, studies assessing aggression with parent and self-report found that genetic influences are important, but the one study (Plomin et al., 1981) that examined aggression using an objective test found no evidence for genetic influences. The objective test used by Plomin et al. (1981) has been validated against peer ratings and teacher ratings of aggression (Johnston, DeLuca, Murtaugh, & Diener, 1977), but the sample size in Plomin et al.'s (1981) study is small. Larger behavior genetic studies using different types of validated, objective tests of aggression are necessary to resolve this question. Given these conflicting findings, there is reason to suspect that one of the assessment methods does not validly assess the construct of aggression. Thus, the finding of genetic influences on antisocial behavior or the lack thereof may be influenced by the method used to assess antisocial behavior.

No matter which operationalization was being examined (i.e., diagnosis, aggression, or antisocial behavior), the magnitude of familial influences (a^2 and c^2) was lower in studies using the assessment method of self-report than in studies using the assessment method of report by others. The only exception occurred in studies examining antisocial behavior, where the a^2 estimate was .47 for both report by others and self-report. These results suggest the possibility that the lower h^2 and c^2 estimates may be more a function of the assessment method of self-report than a function of any of the operationalizations that were examined. Two separate raters are involved in the assessment method of self-report, whereas only one rater rates both twins or siblings when parent report is used. It is possible that this difference between the assessment methods led to lower familial correlations and a lower estimate of the magnitude of familial influences in studies using self-report.

The confounding between age and assessment method precluded our ability to test L. F. DiLalla and Gottesman's (1989) hypothesis regarding genetic influences on continuous versus transitory antisocial behavior. The assessment method of report by others was used only in children and adolescents, whereas the assessment method of self-report was used only in adolescents and adults. Given the fact that the pattern of results for age (i.e., familial influences decreasing and nonfamilial influences increasing as age increases) was identical to the pattern of results for assessment method (i.e., familial influences smaller and nonfamilial influences larger for self-report than for report by others) and that age and assessment method are confounded, it is impossible to conclude whether age moderates the magnitude of genetic and environmental influences on antisocial behavior.

The assessment methods used in future behavior genetic studies of antisocial behavior should be diversified given the common concerns regarding the validity of the assessment method. For example, a combination of official records and self-report should be used to assess criminality given the shortcomings of each assessment method. Larger behavior genetic studies using different types of validated, objective tests of aggression are needed. Most important, the limitations of the assessment method chosen for a behavior genetic study of antisocial behavior should be acknowledged and considered given the evidence that the assessment method can influence the results. If multiple assessment methods are used to assess antisocial behavior in a single twin study, the common pathways model (see Figure 2) can be used to estimate the magnitude of the genetic and environmental influences that are common to the latent construct being examined (i.e., antisocial behavior) and the genetic and environmental influences that are specific to each assessment method (e.g., Riemann, 1999).

Genotype–Environment Interaction

The adoption study is the ideal method for testing genotype–environment interactions because the genetic and environmental influences on a trait are disentangled and can be measured distinctly. In contrast, genotype–environment interactions may be more difficult to test in twin studies because the genetic and environmental influences on a trait are likely to be correlated.

Data from several adoption studies (Cadoret et al., 1983; Cloninger, Sigvardsson, Bohman, & von Knorring, 1982; Mednick et al., 1983) show evidence of genotype–environment interaction for antisocial behavior, although there were not enough relevant studies in the meta-analysis to conduct a quantitative review of this issue. Mednick et al. (1983) conducted a cross-fostering analysis of Danish adoptees. Among adoptees who had a criminal background in both their biological and adoptive parents, 24.5% became criminal themselves. This is in comparison to 20% of adoptees who have a criminal background only in their biological parents, 14.7% of adoptees who have a criminal background only in their adoptive parents, and 13.5% of adoptees with no criminal background. Cloninger et al. (1982) found similar results for petty criminality in Swedish adoptees when they considered both biological variables (i.e., criminality in biological parents) and environmental variables (i.e., negative rearing experiences and adoptive placement). Among adoptees with both biological and environmental risks, 40% were criminal. This is in comparison to 12.1% of those with only biological risk factors, 6.7% of those with only environmental risk factors, and 2.9% of those with neither biological nor environmental risk factors. Also, in a sample of adoptees from Iowa, Cadoret et al. (1983) found that when both genetic and environmental risk factors were present, they accounted for a greater number of antisocial behaviors than an additive combination of the two kinds of risk factors acting independently.

The genotype–environment interactions were not statistically significant in Cloninger et al. (1982) or Mednick et al. (1983). Unfortunately, the power to test the genotype–environment interaction term may be reduced in adoption studies of antisocial

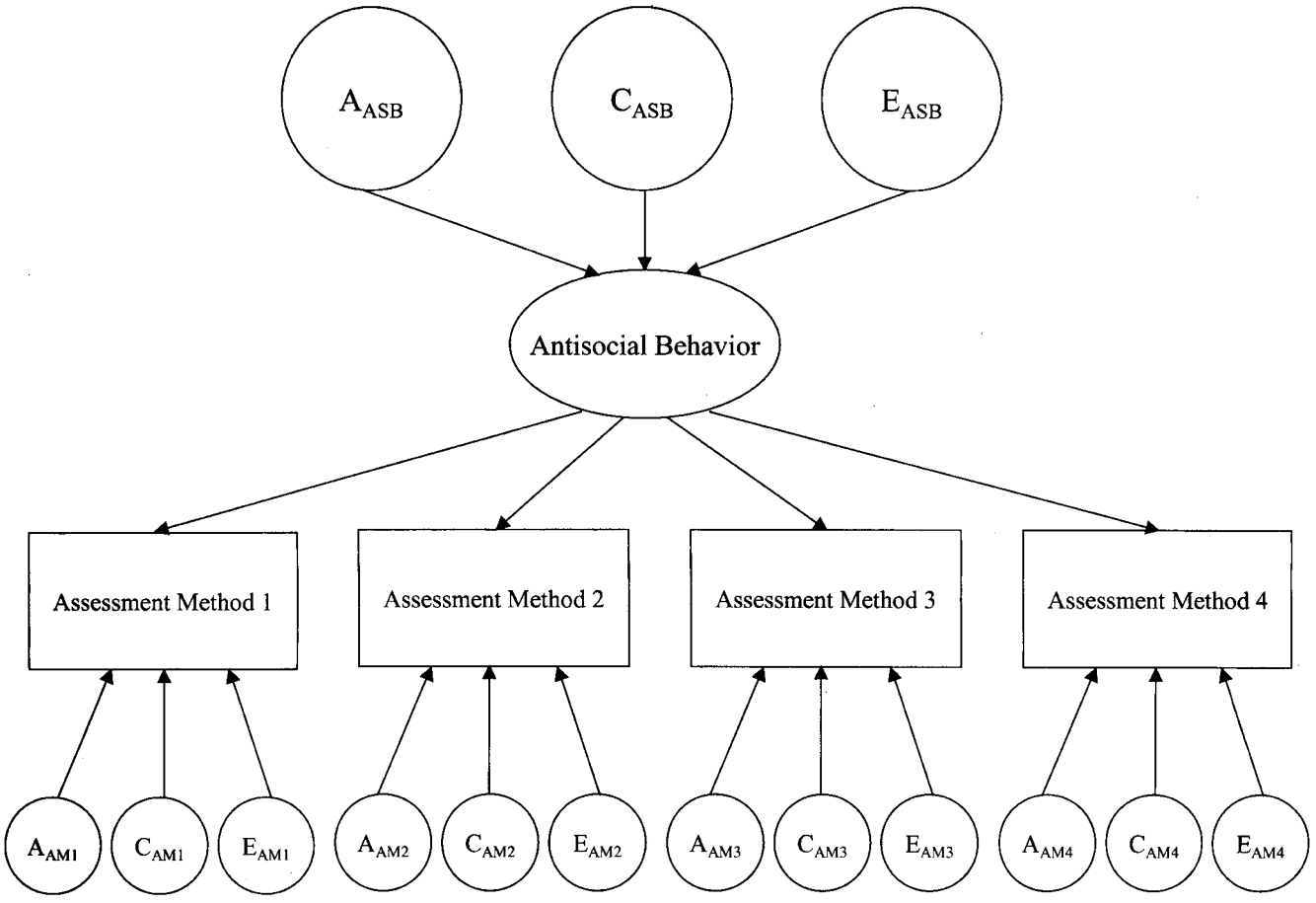

Figure 2. Common pathway model. A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences; ASB = antisocial behavior; AM = assessment method.

behavior because of range restriction in the variables used to indicate the environmental and/or genetic influences on antisocial behavior. McClelland and Judd (1993) demonstrated that restricting the range of the predictor variables reduces the residual variance of the product of the two predictors and, in turn, the statistical power to detect an interaction. The problem with range restriction is especially a concern in adoption studies of antisocial behavior because the chance of adoptees being placed in adoptive homes with criminal or antisocial adoptive parents is very low. For example, one reviewer of this article noted that in Cloninger et al. (1982), none of the 862 adoptees came from an adoptive family in which a parent had an arrest record. Therefore, the statistical difficulties of detecting interactions should be considered in interpreting adoption studies examining genotype–environment interactions. Also, future behavior genetic studies should consider alternative research design strategies, such as oversampling extreme observations (McClelland & Judd, 1993). For example, such studies may oversample children with a low genetic predisposition to antisocial behavior who are reared in environments that predispose them to antisocial behavior.

Multivariate Analyses

In the present meta-analysis, four operationalizations of antisocial behavior were studied: diagnosis, criminality, aggression, and antisocial behavior. Operationalization was a significant moderator, suggesting that the magnitude of genetic and environmental influences is different for the different operationalizations.

In order to determine the extent to which these operationalizations have common or specific genetic and environmental influences, multivariate behavior genetic analyses of two or more operationalizations should be conducted. One example of such an analysis is Cloninger and Gottesman's (1987) finding that there is little genetic overlap between violent and nonviolent crime.

According to several reviewers (e.g., Carey, 1994; L. F. DiLalla & Gottesman, 1991; Nigg & Goldsmith, 1994), the next important step in clarifying the role of genes and environment on antisocial behavior is multivariate behavior genetic research on personality and psychopathology. These researchers have suggested a number of personality variables that may share common genetic influences with antisocial behavior, including low harm avoidance, high novelty seeking, low reward dependence, overattribution of hostility, and many others. Gottesman and Goldsmith (1994) suggested that the statistical line of evidence that must be established is the documentation of the heritability of the personality variables, demonstration that the personality variables predict antisocial behavior, documentation that the patterns of antisocial behavior are heritable, and demonstration that the genetic influences underlying both the personality variables and antisocial behavior overlap.

Age of Onset and Developmentally Different Subtypes of Antisocial Behavior

L. F. DiLalla and Gottesman (1989) and Moffitt (1993) have suggested that in order to show conclusive evidence regarding their hypotheses, future studies of antisocial behavior should include longitudinal data of the same individuals. Five studies included in the current meta-analysis examined the same individuals at two time points, but none of these studies provide the kind of evidence needed to examine L. F. DiLalla and Gottesman's (1989) hypothesis. Two of these studies (Loehlin, Willerman, & Horn, 1987; Lytton, Watts, & Dunn, 1988) only assessed antisocial behavior at the second assessment. Dworkin, Burke, Maher, and Gottesman (1976) found that heritability for psychopathy (i.e., as measured by the MMPI Psychopathic Deviate scale and the CPI Socialization scale) was significant during adolescence (mean age = 15.9) but not during adulthood (mean age = 27.9 years); however, the sample size was very small (i.e., 27 MZ pairs and 17 DZ pairs). McGue, Bacon, and Lykken (1993) found that there are nonadditive genetic influences on aggression at both late adolescence (mean age = 20 years) and adulthood (mean age = 30 years), but the same sample size was small (79 MZ pairs and 48 DZ pairs). Deater-Deckard, Reiss, Hetherington, and Plomin (1997) also reported results on longitudinal data for the same individuals, but the two waves of assessments were only 3 years apart and both assessments occurred when the twins were adolescents.

Environmental Influences on Antisocial Behavior

The most frequently cited candidate for a specific environmental influence on antisocial behavior is parenting style. Patterson and his colleagues (e.g., Patterson et al., 1992) contended that inadequate parental supervision can lead to antisocial behavior in children. They have described *coercive cycles* during which a child responds to a mother's command with aggression or a temper tantrum, the mother responds in turn by backing off, and the aggression or temper tantrum is thus reinforced. Several experimental studies using random assignment show that parent management training, which attempts to alter these coercive cycles by training parents to reinforce prosocial behavior rather than aggressive behavior, is effective in improving parenting skills and reducing aggressive behavior in children (Brestan & Eyberg, 1998; Kazdin, 1987). Further evidence for coercion theory is provided by studies that show that the intervention's effect on the child's aggressive behavior is mediated by the improvement in parenting practices. For example, Forgatch and DeGarmo (1999) showed that parent training reduced coercive parenting, prevented decay in positive parenting, and improved effective parenting practices, and that these improvements in turn led to improvements in child adjustment, including reduced externalizing behavior. Similarly, Eddy and Chamberlain (2000) showed that the positive effects of multidimensional treatment foster care on severe antisocial behavior were mediated by improved family management skills. Also, parenting style may influence children's antisocial behavior indirectly through sibling influences (Bank, Patterson, & Reid, 1996) and peer influences (Forgatch & Stoolmiller, 1994). The results of these studies support the view that parenting styles and behavior represent important environmental influences on antisocial behavior and that they should be included as specific environmental indices in future behavior genetic studies.

In contrast to previous theories that emphasize the influence of parenting, Harris's (1995, 1998) group socialization theory of development emphasizes the importance of peer group influences on personality development. Harris's (1995) main criticism of the previous research emphasizing the influence of parenting styles is the failure to consider the possibility of genetic influences on children's behavior and the possibility that parents could be reacting to their children's behavior rather than causing it. Harris (1995) cited examples of significant peer group influences on several variables including smoking (Rowe, Chassin, Presson, Edwards, & Sherman, 1992, as cited in Harris, 1995) and motivation to do well in school (Kindermann, 1993; as cited in Harris, 1995) and suggested that neighborhood and peer group influences are also important environmental influences on antisocial behavior. According to the group socialization theory of development, delinquency is pervasive during adolescence (Moffitt, 1993) not because adolescents are aspiring to adult status but because adolescents are contrasting themselves from adults as a group by exhibiting delinquent behaviors that set them apart from adults. Harris's (1995, 1998) theory is consistent with previous studies that have reported a significant relationship between exposure to deviant peers and antisocial behavior (e.g., Keenan, Loeber, Zhang, Stouthamer-Loeber, & van Kammen, 1995).

On the other hand, Rowe, Woulbroun, and Gulley (1994) raised the possibility that the relationship between exposure to deviant peers and antisocial behavior may be due to peer selection (i.e., deviant children being more likely to select deviant peers than nondeviant children) rather than peer influence. For example, Rowe and Osgood (1984) found that children's antisocial behavior was significantly related to association with deviant peers, but the cross-correlation between the children's own antisocial behavior and association with deviant peers was higher in MZ twins than in DZ twins. This result suggests that there are genetic influences on the relation between antisocial behavior and association with deviant peers and supports peer selection as an explanation for this relationship.

Deater-Deckard and Dodge (1997) attempted to integrate the results of studies examining the influence of parenting and those of behavior genetic studies of childhood antisocial behavior. They concluded that there is a significant relation between harsh physical discipline and childhood antisocial behavior but that the magnitude of the effect depends on several variables. First, the association between harsh physical discipline and childhood aggression includes a nonlinear component, in that the degree of association should be larger for the upper end of the continuum of physical discipline (i.e., harsh discipline or abusive discipline). Stoolmiller, Patterson, and Snyder (1997) also found evidence for a nonlinear relation between harsh, abusive discipline and antisocial behavior but suggested that the causal effect may be limited to families with out-of-control children and unskilled parents. Second, the association varies across cultural groups, in that there is a positive correlation between physical discipline and childhood aggression for European American children, but not for African American children. Third, parental discipline effects vary according to the context of the broader parent–child relationship, such as parent–child warmth. Fourth, the relation between harsh physical discipline and childhood aggression is stronger for same-gender parent–child dyads.

Turkheimer and Gottesman (1996) discussed the lack of evidence for shared environmental influences from behavior genetic

studies and offered a possible explanation for this finding. They conducted a study simulating the dynamics of genes, environment, and development and concluded that environmental variation is only detectable when the genotype is held constant. Turkheimer and Gottesman explained that two siblings with different genotypes can both be affected by the same shared environmental influences but that the effect of the shared environmental influences may make them dissimilar rather than similar given the differences in their genotype. They also found that small changes in environment can result in large and sudden changes in phenotypic outcomes that would be difficult to capture with traditional linear models. In contrast, linear models fit the phenotypic variation associated with genotype well. In future studies examining shared environmental influences on antisocial behavior, researchers should consider the possibility of nonlinear relations.

Given the strong evidence of both shared and nonshared environmental influences on antisocial behavior, more studies examining specific shared and nonshared environmental influences within behavior genetic designs are needed. Behavior genetic studies are uniquely equipped to examine these issues, given their ability to estimate the true magnitude of parental and peer environmental influences on antisocial behavior while controlling for genetic influences, including those on peer selection. Although difficult to implement, the examination of specific environmental influences in a combined twin–adoption design is especially recommended given the ability to examine measured environmental variables, shared and nonshared environmental influences, and additive and nonadditive genetic influences simultaneously. There also are a number of genetically noninformative designs that can be used to evaluate the effects of the environment while controlling for genetic effects (see Rutter, Pickles, Murray, & Eaves, 2001, for a detailed review). These include migration designs (i.e., comparing the incidence of a disorder in a migrant population to that in the country of origin and the host country), time trends analyses (e.g., changes in marriage rates, secular trends in suicide), and intervention designs (e.g., the parent training studies discussed above).

Conclusion

In the current meta-analysis, we found that there were moderate additive genetic ($a^2 = .32$), nonadditive genetic ($d^2 = .09$), shared environmental ($c^2 = .16$), and nonshared environmental influences ($e^2 = .43$) on antisocial behavior. When twin and adoption studies were compared, there was a significant difference between twin studies and parent–offspring adoption studies, but not between twin studies and sibling adoption studies. There was a lower magnitude of familial influences (i.e., both a^2 and c^2) in the parent–offspring adoption studies as compared with the twin or sibling adoption studies. All of the potential moderators examined except for sex (i.e., operationalization, assessment method, zygosity determination method, and age) were found to account for significant differences in the genetic and environmental influences on antisocial behavior. Although there was a significant difference between studies examining both sexes simultaneously and studies examining the two sexes separately, there was no statistically significant difference in the results for males and females in studies that included both sexes. Several future directions were recommended for overcoming the limitations of the present meta-analysis and for improving our understanding of genetic and environmental influences on antisocial behavior. These include examining further distinctions in the operationalization of antisocial behavior, diversifying the assessment methods used to measure antisocial behavior, examining genotype–environment interactions, conducting multivariate behavior genetic analyses, conducting longitudinal studies to more effectively examine the effects of age of onset and developmentally different subtypes on the genetic and environmental influences underlying antisocial behavior, examining specific environmental influences, and controlling for the effects of assortative mating.

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Appendix A

Terms Used in PsycINFO and *Medline* Searches

We searched for each of the words in the left column in combination with any of the words in the right column:

aggressive	twin(s)
aggression	adoptee(s)
antisocial	adoptive
conduct	genetic
psychopathy	genetics
sociopathy	genes
crime	environmental
criminal	environment
criminality	
delinquent	
delinquency	
behavior problem(s)	
problem behavior(s)	

Appendix B

Correlations for Adoption and Twin Relationships

Relationship	Correlation
Adoption studies	
Adoptee–adoptive parent	1*C
Adoptee–biological parent	.5*A
Biological child–biological parent	.5*A + 1*C
Adoptive siblings	1*C
Biological siblings	.5*A + 1*C + .25*D
Twin studies	
MZ twin pairs reared together	1*A + 1*C + 1*D
DZ twin pairs reared together	.5*A + 1*C + .25*D
MZ twin pairs reared apart	1*A + 1*D
DZ twin pairs reared apart	.5*A + .25*D

Note. C = shared environmental influences; A = additive genetic influences; D = nonshared environmental influences; MZ = monozygotic; DZ = dizygotic.

(*Appendixes continue*)

Appendix C

Example of an Mx Script for a Model Testing an ACDE Model

```
G1: model parameters
Calculation Ngroups=9
Matrics
X Lower 1 1 Free        ! a: additive genetic parameter
Y Lower 1 1 Free        ! c: shared environmental parameter
Z Lower 1 1 Free        ! e: unique environmental parameter
W Lower 1 1 Free        ! d: non-additive genetic influence parameter
I Iden 22
H Full 1 1              ! scalar, .5
Q Full 1 1              ! scalar, .25
End Matrics;
Matrix H .5
Matrix Q .25
Begin Algebra;
A=X*X′;                 ! a^2: additive genetic variance
C=Y*Y′;                 ! c^2: shared environmental variance
E=Z*Z′;                 ! e^2: unique environmental variance
D=W*W′;                 ! d^2: non-additive genetic variance
V=A+C+E+D;              ! total variance
P=A|C|E|D;              ! put parameter estimates in one matrix
S=P@V~;                 ! standardized parameter estimates
End Algebra;
Labels Row X parest_a
Labels Row Y parest_c
Labels Row Z parest_e
Labels Row W parest_d
Labels Row A a^2
Labels Row C c^2
Labels Row E e^2
Labels Row D d^2
Labels Row V variance
Labels Row P estimate
Labels Col P a c e d
Labels Row S standest
Labels Col S a^2 c^2 e^2 d^2
End

Title G2: adoptee-biological parents - Loehlin 1987
Data NInput_vars=2 NObservations=81
KMatrix Symm
1
.095 1
Matrices = Group 1
Covariances A+C+E+D | H@A _
H@A | A+C+E+D /
Option Rsiduals
End

Title G3: adoptee-adoptive mother - Loehlin 1985
Data NInput_vars=2 NObservations=253
Kmatrix Symm
1
−.02 1
Matrices = Group 1
Covariances A+C+E+D | C _
C | A+C+E+D /
Option Rsiduals
End

Title G4: biological siblings - van den Oord 1994
Data NInput_vars=NObservations=35
KMatrix Symm
1
.42 1
Matrices = Group 1
Covariances A+C+E+D | H@A+C+Q@D _
H@A+C+Q@D | A+C+E+D /
Option Rsiduals
End
```

Appendix C (*continued*)

```
Title G5: adoptive siblings - van den Oord 1994
Data NInput_vars=2 NObservations=48
KMatrix Symm
1
.37 1
Matrices = Group 1
Covariances A+C+E+D | C _
C | A+C+E+D /
Option Rsiduals
End

Title G6: MZ twin pairs reared together - Cates 1993
Data NInput_vars=2 NObservations=77
KMatrix Symm
1
.29 1
Matrices = Group 1
Covariances A+C+E+D | A+C+D _
A+C+D | A+C+E+D /
Option Rsiduals
End

Title G7: DZ twin pairs reared together - Cates 1993
Data NInput_vars=2 NObservations=21
KMatrix Symm
1
.16 1
Matrices = Group 1
Covariances A+C+E+D | H@A+C+Q@D _
H@A+C+Q@D | A+C+E+D /
Option Rsiduals
End

Title G8: MZ twin pairs reared apart - DiLalla 1996
Data NInput_vars=2 NObservations=66
KMatrix Symm
1
.62 1
Matrices = Group 1
Covariances A+C+E+D | A+D _
A+D | A+C+E+D /
Option Rsiduals
End

Title G9: DZ twin pairs reared apart - DiLalla 1996
Data NInput_vars=2 NObservations=54
KMatrix Symm
1
.14 1
Matrices = Group 1
Covariances A+C+E+D | H@A+Q@D _
H@A+Q@D | A+C+E+D /
Option Rsiduals

Option NDecimals=4
Option DF=-15
Option Iterations=200
Option Check
End
```

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