Genetic and Environmental Influences on Aggression

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Author Note
This work was supported in part by NIDA DA-13956 to Soo Hyun Rhee and NIMH MH-01818 to Irwin D. Waldman. Earlier versions of this chapter were presented at the meeting of the American Society of Criminology in 1996 and the meeting of the Behavior Genetics Association in 1997, and a more extensive version has been published in Psychological Bulletin, 128(3), pp. 490–529.
Considerable research has focused on understanding the etiology of aggression and antisocial behavior and has utilized a myriad of theoretical and empirical approaches. Of these, behavior genetic methods have the advantages of testing competing alternative etiological models and of clearly distinguishing genetic from environmental influences and estimating their magnitude. More than a hundred twin and adoption studies of antisocial behavior have been published, which led us (Rhee & Waldman, 2002) to conduct a meta-analysis of twin and adoption studies of antisocial behavior and several specific operationalizations thereof, including aggression.

In this chapter, we address the specific question of the magnitude of genetic and environmental influences on aggression, and how the pattern and magnitude of these influences compare to those on antisocial behavior in general. In the twin and adoption studies reviewed here, aggression is usually studied as a personality characteristic and assessed with such measures as the Adjective Checklist (Gough & Heilbrun, 1972) and the Multidimensional Personality Questionnaire (Tellegen, 1982 as cited in Tellegen, Lykken, Bouchard, Wilcox, Segal, & Rich, 1988). The operationalization of aggression has been very heterogeneous in the past, ranging from reports of negative affect (Partanen, Bruun, & Markkanen, 1966) to observations of the number of times a child hits a Bobo doll (Plomin, Foch, & Rowe, 1981). For the present review, the operationalization of aggression was restricted to the type of behavioral aggression described in the DSM-IV criteria for conduct disorder (CD) (e.g., bullying, initiating physical fights, and using a weapon that can cause serious physical harm). Toward the end of the chapter, we qualitatively review the emerging behavior genetic literature on specific forms of aggression in children, including reactive, proactive, and relational aggression. We also briefly describe the burgeoning molecular genetic literature examining the association of relevant candidate genes with aggression.

The role of familial influences on antisocial behavior has been studied extensively. Dysfunctional familial influences, such as parental psychopathology (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 found 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 reaching the eventual goal of explaining the specific 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.

Twin studies can disentangle genetic and environmental influences by comparing the similarity between monozygotic twin pairs – who share 100% of their genes identical by descent – to the similarity between dizygotic twin pairs, who share 50% of their genes, on average. Traits with genetic influences will show greater similarity between monozygotic twins than between dizygotic twins. Adoption studies demonstrate genetic influences on a trait if there is a significant correlation between the trait in adoptees and their biological relatives, and that there are environmental influences on a trait if there is a significant correlation between the trait in adoptees and their adoptive parents or siblings.

**A Meta-Analysis of Behavior Genetic Studies of Aggression**

We began our search for twin and adoption studies of aggression by examining the PsycInfo and Medline databases. The search terms used in this process were aggressive, aggression, crime, criminality, delinquent, or delinquency in combination with the terms twin(s), adoptee(s), adoptive, genetic, genetics, genes, environmental, or environment. We examined the references from the research studies and review papers found through this method for any additional studies that might have been missed or published before the databases were established. After excluding unsuitable studies according to the criteria described below (i.e., construct validity, inability to calculate tetrachoric or intraclass correlations, and assessment of related disorders), and addressing the problem of nonindependence in these studies, 14 studies examining aggression remained. Table 1
lists the behavior genetic studies examining aggression included in the meta-analysis. Also listed are the method of assessment and method of zygosity determination (in twin studies) used in the study, the mean or median age, the sex of the sample, the number of pairs, the relationship of the pairs, and the effect sizes.

**INSERT TABLE 1 ABOUT HERE**

**Inclusion Criteria for Studies in the Meta-Analysis**

*construct validity.* Studies examining aggression were included if they examined behavioral aggression (e.g., physical fighting, cruelty to animals, and bullying). Studies that examined other related variables, such as anger, hostility, or impulsivity, were not included because it was not clear whether they examined aggression or some related but distinct trait. An additional study (Partanen, Brunn, & Markkanen, 1966) was excluded because the aggression items examined (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 per se, was being assessed.

*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 using model-fitting programs that estimate the relative contribution of genetic and environmental influences and test the fit of alternative etiological models.

*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, in which researchers examined more than one dependent measure of antisocial behavior in their sample or published follow-up data of the same sample in separate publications. Several suggestions for dealing with nonindependent samples have been offered in the meta-analytic literature (Mullen, 1989; Rosenthal, 1991). For example, Mullen gives 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. Taking this option would have required 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, given that 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.

Given that model-fitting analyses require specification of the sample size, we used the option of averaging multiple effect sizes in cases where the sample size was identical across the nonindependent samples. If the sample size was not identical across the nonindependent samples, we used the effect size from the largest sample. More specifically, in cases of nonindependence where the same dependent measure was used in the same sample multiple times (e.g., in follow-up analyses), we chose the effect size estimated from the largest sample. 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.

**Determination of Effect Sizes**

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) were 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) which was the effect size used in the meta-analysis. For some studies, we directly estimated the tetrachoric correlation from the raw data 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 co-variance matrix of the correlation matrix). If the weight matrix can be estimated, it is possible to use weighted least squares (WLS) estimation in the model-fitting analyses, which is more appropriate for non-normally distributed variables like diagnoses, rather than maximum likelihood (ML) estimation.

Model-Fitting Analyses

The magnitude of additive and nonadditive genetic influences \((a^2\) and \(d^2\)) constitutes the proportion of variance in the liability for aggression that is due to genetic differences among individuals. If genetic influences are additive, the effects of alleles from different loci are independent and “add up” to influence the liability underlying a trait. If genetic influences are nonadditive, the 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 proportion 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 proportion 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 co-variances). These models posit that aggression is caused by the types of influences described above: additive genetic influences \((A)\), nonadditive genetic influences \((D)\), shared environmental influences \((C)\), and nonshared environmental influences \((E)\). In the present meta-analysis, we contrasted the fit of the ACE model, the AE model, the CE model, and the ADE model. We assessed the fit of each model, as well as of competing models, using both the \(\chi^2\) statistic and the Akaike Information Criterion (AIC), a fit index that reflects both the fit of the model and its parsimony (Loehlin, 1992). The AIC has been used extensively in both the structural equation
modeling and behavior genetic literatures. Among competing models, that with the lowest AIC and the
lowest $\chi^2$ relative to its degrees of freedom is considered to be the best-fitting model.

Unfortunately, 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 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 the MZ correlation, however, the ADE model is the correct model, and the
estimate of $c^2$ in the ACE model is always zero.

Results and Discussion

Table 2 shows the model-fitting results for aggression and for antisocial behavior for purposes of
comparison. The ACE model was the best-fitting model for aggression ($a^2 = .44$, $c^2 = .06$, $e^2 = .50$), although the fit
of this model was very close to that of the AE model, and the magnitude of shared environmental influences on
aggression is modest. By comparison, in our omnibus meta-analysis of behavior genetic studies of antisocial
behavior there were moderate additive genetic ($a^2 = .32$), non-additive genetic ($d^2 = .09$), shared environmental
($c^2 = .16$), and non-shared environmental ($e^2 = .43$) influences. These results suggest that the magnitude of
genetic and non-shared environmental influences is slightly higher for aggression than for antisocial behavior in
general, and that evidence for the role of shared environmental influences on aggression is at best tentative
pending future studies.

Future Directions in Behavior Genetic Studies of Aggression

Unfortunately, we were unable to examine a meaningful distinction between relational and overt
aggression (Crick, Casa, & Mosher, 1997; Crick & Gropet, 1995) because there are no published twin or
adoption studies of relational aggression. Overt aggression harms others through physical damage or the threat
thereof, whereas relational aggression harms others by damaging their peer relationships or reputation (e.g.,
spreading rumors, excluding 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 especially important when examining sex differences in aggression and its causes, given that females are significantly more relationally aggressive and less overtly aggressive than males (Crick & Grotpeter, 1995; Crick et al., 1997). Given the evidence that overt and relational aggression are correlated but distinct (Crick et al., 1997), future behavior genetic studies are necessary to examine the degree of genetic and environmental influences that are common to both overt and relational aggression versus specific to each.

Similarly, few behavior genetic studies have distinguished between reactive and proactive aggression (Dodge, Lochman, Harnish, Bates, & Pettit, 1997; Vitaro, Brendgen, & Tremblay, 2002; Waschbusch, Willoughby, & Pelham, 1998). Reactive aggression is characterized by impulsive “hot-blooded” anger, appears to be a response to frustration or perceived threat, and is associated with a lack of self control, whereas proactive aggression is premeditated and “cold-blooded,” less emotional, and more likely driven by the expectation of reward (Dodge et al., 1997). Evidence suggests that reactively and proactively aggressive children differ in developmental histories, adjustment, and social information-processing patterns and that reactive and proactive aggression are distinct (Dodge et al., 1997; Vitaro et al., 2002). Future directions for behavior genetic studies of antisocial behavior should include the examination of the distinction between relational and overt aggression, and multivariate studies examining the etiology of the overlap between different operationalizations of antisocial behavior. Genetically informative studies of aggression recently have made important strides in these areas. In addition, several behavior genetic studies examining the development of aggression have been conducted.

The Overlap between Different Dimensions of Aggression

Two genetically informative studies have examined the common and unique etiologies between proactive and reactive aggression, with differing results. Brendgen et al. (2006) examined the association between proactive and reactive aggression in a sample of 172 6-year-old twin pairs from Quebec. The
magnitude of genetic influences was similar for proactive ($h^2 = .41, e^2 = .59$) and reactive aggression ($h^2 = .39, e^2 = .61$). The correlation between proactive and reactive aggression ranged from .51 to .60 in this sample, with the correlation between genetic influences on the two types of aggression being .87, and the correlation between nonshared environmental influences being .34. Brendgen et al. also found that most of the association between proactive and reactive aggression was due to common etiological factors influencing physical aggression. The second study examining proactive vs. reactive aggression is Baker et al. ’s (2008) study of 1219 9- to 10-year-old twins from southern California. Results were presented separately for child, mother, and teacher report, as the correlation between these sources was low (.18 to .26). They found a significant sex difference in the magnitude of genetic and environmental influences on aggression for child report (boy – $h^2 = .38$ and $e^2 = .62$ for reactive aggression and $h^2 = .50$ and $e^2 = .50$ for proactive aggression; girls – $c^2 = .36$ and $e^2 = .64$ for reactive aggression and $c^2 = .14$ and $e^2 = .86$ for proactive aggression), but not for mother report ($h^2 = .26, c^2 = .27, and e^2 = .46$ for reactive aggression and $h^2 = .32, c^2 = .21, and e^2 = .47$ for proactive aggression) or teacher report ($h^2 = .20, c^2 = .43, and e^2 = .37$ for reactive aggression and $h^2 = .45, c^2 = .14, and e^2 = .41$ for proactive aggression). The phenotypic correlation between proactive and reactive aggression ranged from .46 to .80. Results suggest that both common genetic and environmental influences are responsible for this correlation, but results varied across the three sources (child report – $r_g = .57$ and $r_e = .46$ for boys, $r_c = .53$ and $r_e = .53$ for girls; mother report – $r_g = .76, r_c = .76, r_e = .43$; teacher report – $r_g = 1.0, r_c = 1.0, r_e = .53$).

As stated above, another meaningful distinction in aggression is that between relational and overt aggression (Crick & Grotspeter, 1995; Crick, Casa, & Mosher, 1997). Two recent twin studies have examined the association between relational and overt aggression. Brendgen et al. (2005) examined the association between physical aggression (i.e., overt aggression) and social aggression (i.e., relational aggression) in 234 6-year-old twin pairs from Quebec. Teacher and peer reports of physical and social aggression were obtained, and the magnitude of genetic and environmental influences was similar for the two sources (teacher report – $h^2 = .63, e^2 = .37$ for physical aggression; $h^2 = .20, c^2 = .20, e^2 = .60$ for social aggression; peer report – $h^2 = .54, e^2 = .46$ for physical aggression; $h^2 = .23, c^2 = .23, e^2 = .54$ for social aggression). The phenotypic correlation between
physical and social aggression was .43 for teacher ratings and .41 for peer ratings, and there was evidence of
common genetic and nonshared environmental influences on the two types of aggression (teacher report – \( r_g = .79, r_e = .31 \); peer report – \( r_g = 1.0, r_e = .12 \)). Ligthart et al. (2005) examined relational vs. direct (i.e., overt)
aggression in 7449 7-year-old Dutch twin pairs. They found evidence of genetic, shared environmental, and
nonshared environmental influences on both relational and direct aggression, and found evidence of significant
sex differences on the magnitude of genetic and environmental influences for direct aggression (relational
aggression – \( h^2 = .66, c^2 = .16, e^2 = .18 \); direct aggression – \( h^2 = .53, c^2 = .23, e^2 = .24 \) for males; \( h^2 = .60, c^2 = .13, e^2 = .27 \) for females). The phenotypic correlation between relational and direction aggression was .58 for
boys and .47 for girls, and this correlation was due to both common genetic and shared environmental influences
(55% genetic, 33% shared environmental, and 12% nonshared environmental influences in boys and 58%
genetic, 30% shared environmental, and 12% nonshared environmental influences in girls).

The Overlap between Aggression and Other Types of Antisocial Behavior

Several studies have examined the potential differential etiology of aggressive and non-aggressive
antisocial behavior. Although the results from these studies are not uniform, there are several general
conclusions that can be drawn from them, which are summarized in Table 3. First, although most studies
reported similar, moderate heritabilities for both types of antisocial behavior, some studies (e.g., Eley,
Lichtenstein, & Stevenson, 1999) suggest that aggressive behavior is more heritable than non-aggressive
antisocial behavior. This conclusion is borne out by findings from a recent meta-analysis of 19 studies of
aggressive and 15 studies of non-aggressive antisocial behavior (Burt, 2009). In this meta-analysis, the etiology
of aggression included additive genetic and non-shared environmental influences but no evidence for shared
environmental influences (\( h^2 = .65, c^2 = .00, e^2 = .35 \)), whereas the etiology of non-aggressive antisocial behavior
also included shared environmental influences and showed a lower magnitude of genetic influences (\( h^2 = .48, c^2 = .18, e^2 = .34 \)).

Second, the phenotypic correlation between aggressive and non-aggressive antisocial behavior was
moderate in most studies (ranging from .32 in Gelhorn et al., 2006 and .48 to .76 in Bartels et al., 2003). Third,
most studies suggest that there are significant common genetic influences on the covariance between aggressive behavior and non-aggressive antisocial behavior. Fourth, there was phenotypic continuity between aggressive and non-aggressive antisocial behavior from childhood to adolescence ($r = .28$ to .61 in Eley, Lichtenstein, & Moffitt, 2003), which also had genetic influences. However, it is possible that some of this phenotypic continuity is due to shared method variance, as the correlation between aggressive behavior in childhood assessed via parent report and non-aggressive antisocial behavior in adolescent assessed via self report was lower ($r = .07$ to .15 in Tuvblad, Eley, & Lichtenstein, 2005).

**INSERT TABLE 3 ABOUT HERE**

*Behavior Genetic Studies of the Development of Aggression*

Several researchers have examined the stability of aggressive behavior in behavior genetic studies. The overall conclusion is that aggressive behavior is moderately stable from childhood to adolescence, and that genetic influences explain a larger percentage of the stability of aggressive behavior than shared or nonshared environmental influences. van der Valk, Verhulst, Neale, and Boomsma (1998) conducted a longitudinal study of aggressive behavior in 111 pairs of adopted biological siblings, 221 pairs of adopted nonbiological siblings, and 1484 adopted singletons from the Netherlands. Aggressive behavior was assessed via parent questionnaires at age 10-15 years, then again three years later. The correlation between the two time points ranged from .51 to .70, and the covariance between the two time points was due 69% to genetic influences, 14% to shared environmental influences, and 17% to nonshared environmental influences. van Beijsterveldt, Bartels, Hudziak, and Boomsma (2003) examined the stability of aggression at ages 3, 7, 10, and 12 in a large sample of Dutch twin pairs (ranging from 1509 pairs at age 12 to 6488 pairs at age 3). Aggressive behavior was assessed via parent questionnaires. The correlations for aggression assessed at different ages ranged from .41 to .77. Genetic influences explained approximately 65% of the total stability of aggression, whereas shared environmental influences accounted for approximately 25% of the total stability of aggression. A simplex model, wherein a dynamic developmental process consisting of transmission of existing influences interacting with new
influences, fit best for genetic influences. In contrast, there was a stable set of the same shared environmental influences and age-specific nonshared environmental influences.

Haberstick, Schmitz, Young, and Hewitt (2006) examined the development of aggressive behavior from childhood to early adolescence (parent report at age 7, 9, 10, 11, and 12 and teacher report at age 7, 8, 9, 10, 11, and 12), and reported conclusions slightly different from those of van Beijsterveldt et al. (2003). The contribution of common genetic influences to the stability of aggression ranged from 66% to 87% for parent report and 59% to 95% for teacher report, and the rest was attributable to common nonshared environmental influences. There was no evidence of significant common shared environmental influences across age. The authors noted that there is consistent evidence of common genetic influences on aggression assessed at different ages despite the fact that different teachers rated the children at each age. Eley, Lichtenstein, and Moffitt (2003) examined over 1000 twin pairs from the Swedish Twin Registry assessed at age 8-9 and 13-14 years. Aggressive behavior was assessed via parent report at both time points. Continuity in aggression between childhood and adolescence ranged from .53 to .69, and was due 84% to genetic influences, 8% to shared environmental influences, and 8% to nonshared environmental influences.

**Candidate Genes for Aggression and Antisocial Behavior**

Based on the evidence that aggression is heritable, researchers have initiated attempts at finding specific genes that contribute to its etiology using a candidate gene approach. In well-designed candidate gene studies, genes are selected based on the known or hypothesized involvement of their gene product in the etiology of the trait or disorder (i.e., its pathophysiological function and etiological relevance). Whereas genome scans may be thought of as exploratory searches for putative genes that underlie a disorder or trait, well-conducted candidate gene studies represent a targeted test of the role of specific genes in the etiology of a disorder or trait, as the location, function, and etiological relevance of candidate genes are most often known or strongly hypothesized *a priori*. Genes underlying various aspects of the dopaminergic, noradrenergic, and serotonergic neurotransmitter pathways represent viable candidates given the role of these neurotransmitter systems in the etiology and pathophysiology of aggression. For example, there is considerable overlap between antisocial behavior and
childhood Attention Deficit Hyperactivity Disorder (e.g., Lilienfeld & Waldman, 1990); thus candidate genes for ADHD may also be relevant candidates for aggression and antisocial behavior. Several genes within the dopamine system appear to be risk factors for ADHD (Waldman & Gizer, 2006). Dopamine genes are plausible candidates for ADHD, given that the stimulant medications that are the most frequent and effective treatments for ADHD appear to act primarily by regulating dopamine levels in the brain (Seeman & Madras, 1998; Solanto, 1984) although they also affect noradrenergic and serotonergic function (Solanto, 1998). In addition, “knock-out” gene studies in mice, which examine the behavioral effects of the deactivation of specific genes, have further demonstrated the potential relevance of genes within these neurotransmitter systems. Results of such studies have markedly strengthened the consideration as candidate genes for ADHD of dopaminergic genes, such as the dopamine transporter gene \( \text{DAT1} \); Giros, Jaber, Jones, Wightman, & Caron, 1996) and the dopamine receptor D3 and D4 genes \( \text{DRD3} \) and \( \text{DRD4} \); Accili et al., 1996; Dulawa, Grandy, Low, Paulus, & Geyer, 1999; Rubinstein et al., 1997), as well as genes within the serotonergic system, such as the serotonin 1\( \beta \) receptor gene \( \text{HTR1\( \beta \)} \); Saudou et al., 1994). Serotonergic genes also are plausible candidates for aggression, given the demonstrated relations between serotonergic function and aggression (Berman, Kavoussi, & Coccaro, 1997).

Although a comprehensive review of molecular genetic studies of aggression and antisocial behavior is beyond the scope of this review, several lines of research have implicated an association between serotonin and aggression. Several researchers have found lower cerebrospinal fluid levels of 5-hydroxyindoleacetic acid, a serotonin metabolite, in aggressive or violent individuals (e.g., Brown, Goodwin, Ballenger, Goyer, & Major, 1979; Linnoila et al., 1983). Mice lacking the \( \text{HTR1\( \beta \)} \) gene show enhanced aggressive behavior (Saudou et al., 1994), and a serotonin transporter \( \text{5HTT} \) polymorphism is associated with aggression in nonhuman primates who experienced insecure early attachment relationships (Suomi, 2003). Unfortunately, human studies examining the association between \( \text{5HTT} \) and violence or aggression have yielded conflicting results.

Candidate genes for neurotransmitter systems may include (1) \textit{precursor genes} that affect the rate at which neurotransmitters are produced from precursor amino acids (e.g., tyrosine hydroxylase for dopamine, tryptophan hydroxylase for serotonin); (2) \textit{receptor genes} that are involved in receiving neurotransmitter signals
(e.g., genes corresponding to the five dopamine receptors, DRD1, D2, D3, D4, and D5, and to the serotonin receptors, such as HTR1β and HTR2A); (3) transporter genes that are involved in the reuptake of neurotransmitters back into the presynaptic terminal (e.g., the dopamine and serotonin transporter genes, DAT1 and 5HTT); (4) metabolite genes that are involved in the metabolism or degradation of these neurotransmitters (e.g., the genes for catechol-o-methyl-transferase [COMT] and for monoamine oxidase A and B [i.e., MAOA and MAOB]); and (5) genes that are responsible for the conversion of one neurotransmitter into another (e.g., dopamine beta hydroxylase, or DβH, which converts dopamine into norepinephrine). We anticipate that there will be a steep increase in the number of studies of such candidate genes and aggression, violence, and antisocial behavior over the next decade.

In conclusion, the results of a meta-analysis suggest that there are moderate additive genetic and nonshared environmental influences and modest shared environmental influences on aggression. Behavior genetic studies examining relational versus overt aggression and reactive versus proactive aggression recently have begun to be conducted. The first set of these studies demonstrate moderate heritabilities and non-shared environmental influences on these aggression dimensions, with genetic influences contributing substantially to their overlap. Shared environmental influences also were found to underlie some of these dimensions of aggression, particularly social or relational aggression. An association between dopamine and serotonin and aggression has been implicated in some early candidate gene studies, although human studies examining the association between the serotonin transporter gene and violence or aggression have yielded conflicting results. Future molecular genetic studies will illuminate the specific genetic underpinnings of aggression and antisocial behavior.
References

Note: References marked with an asterisk indicate studies included in the meta-analysis.


Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-


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Table 1: Effect Sizes for Behavior Genetic Studies Examining Aggression

*Note.* Only the first author’s name was included in the table. Information within the parentheses indicates whether the data were obtained from personal communication or another publication. *m* = male; *fm* = female; *both* = both male and female; *MZ* = *MZ* twin pairs; *DZ* = *DZ* twin pairs; *MZ ra* = *MZ* twin pairs reared apart.
Table 2: Model-Fitting Results: Standardized Parameter Estimates and Fit Statistics

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