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Human Aggression Across the Lifespan: Genetic Propensities and Environmental Moderators

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Abstract

This chapter reviews the recent evidence of genetic and environmental influences on human aggression. Findings from a large selection of the twin and adoption studies that have investigated the genetic and environmental architecture of aggressive behavior are summarized. These studies together show that about half (50%) of the variance in aggressive behavior is explained by genetic influences in both males and females, with the remaining 50% of the variance being explained by environmental factors not shared by family members. Form of aggression (reactive, proactive, direct/physical, indirect/relational), method of assessment (laboratory observation, self-report, ratings by parents and teachers), and age of the subjects—all seem to be significant moderators of the magnitude of genetic and environmental influences on aggressive behavior. Neither study design (twin vs. sibling adoption design) nor sex (male vs. female) seems to impact the magnitude of the genetic and environmental influences on aggression. There is also some evidence of gene-environment interaction (G × E) from both twin/adoption studies and molecular genetic studies. Various measures of family adversity and social disadvantage have been found to moderate genetic influences on aggressive behavior. Findings from these G × E studies suggest that not all individuals will be affected to the same degree by experiences and exposures, and that genetic predispositions may have different effects depending on the environment.

Are all humans innately and equally capable of inflicting harm on others? Do we learn by our various experiences to manipulate and even harm others for our own personal gain; or conversely, to be kind and benevolent, offering help even at costs to ourselves? Although these fundamental questions pertaining to the nature of human aggression have plagued scientists and laypersons for centuries, some answers can be found in research spanning the last few decades.

The early experiments of Milgram (1963) made it clear that, under certain circumstances, individuals can be coaxed into aggression and violence. The presence of a strict authority and removal of personal responsibility for one's actions can result in aggressive behaviors that inflict harm on others. The infamous Stanford prison experiment (Haney *et al.*, 1973) also demonstrated that the propensity toward violence and aggression can be elicited—extremely and unexpectedly—in situations, where a legitimized ideology and a powerful authority can lead to impressionability and obedience.

Yet, while these powerful studies revealed the importance of social factors in inducing aggressive behaviors, not all individuals responded in an equally aggressive manner. In Milgram's (1963) first set of experiments, while 65% (26 of 40) of participants complied with the instruction to administer what they believed to be a final, massive 450-volt shock, the remaining 35% did not comply. Many of those who engaged in the aggressive behavior stated they were very uncomfortable doing so, and every participant reportedly questioned the experiment at some point or refused money promised for their study participation (Milgram, 1963). Although the studies by Milgram and Zimbardo provide clear evidence for the role of environment and social situations in affecting aggressive behavior, there are,

nonetheless, large individual differences in the propensity for violence and aggression, even under these extreme circumstances.

What factors contribute to individual differences in aggression? Behavioral genetic studies of family members' resemblance for aggressive behavior help shed light on the matter. Twin and adoption studies agree with the experimental literature on aggression, which shows that a large effect of environmental factors is evident, particularly of the nonshared variety. Yet, there is also plenty of evidence, based on a variety of definitions of aggressive behavior from children to adults, for genetic propensity toward aggression (see reviews by Burt, 2009; Miles and Carey, 1997; Rhee and Waldman, 2002). Although few behavioral genetic studies have explicitly examined the question of gene by environment (G × E) interactions, we contend that such interactions are likely to exist and that the genetic propensity for aggression should exert its effects more strongly in some situations than others. Consistent with the early findings of Milgram and Zimbardo, individual genetic predispositions should moderate the extent to which aggression can be elicited, even in extreme situations such as these infamous studies. Our view is that while many, if not most, humans may have the potential for aggression and violence under the right circumstances, not all individuals will succumb to these behaviors under the same circumstances.

This chapter will review recent evidence of genetic and environmental influences on human aggression, with particular attention to several key questions and issues. We first consider how estimates of the relative importance of genetic effects (i.e., heritability) may vary across forms of aggression and the way in which it is measured. As detailed in other chapters of this volume, there are numerous definitions of aggression. Some definitions distinguish between reactive and proactive forms (Dodge *et al.*, 1997; Raine *et al.*, 2006), and others consider direct and indirect forms of aggression (e.g., physical vs. relational; Lahey *et al.*, 2004; Tackett *et al.*, 2009). Some definitions may include extreme criminal violence, such as assault, rape, and murder, although these extreme behaviors are relatively rare and have not been studied extensively in genetically informative designs. Measures of aggression can include self-reporting, teacher and parent reports (particularly for young children), and official records from schools or the justice system. This review focuses on twin and sibling adoption studies of aggressive behavior measured as a trait within the wider population. We compare effect sizes (heritability) across these various definitions and ways of measuring aggression. We also consider how heritability estimates may vary across both age and gender. Given higher levels of aggression in males across the lifespan, one obvious question concerns whether genetic propensities are of greater importance in one sex and how these differences might vary across age. We consider a variety of measurable environmental factors that might moderate these genetic influences and which may thus lead to G × E interactions for aggressive behavior. Although direct tests of G × E interactions have been relatively rare in the behavioral genetic literature on human aggression, it is likely that such interactions exist, given their robust effects in other forms of antisocial behavior (e.g., property criminal offending; Cloninger *et al.*, 1982).

Finally, we briefly review evidence for specific genetic influences in aggression by summarizing some of the more recent findings from molecular genetic studies. These effects are reviewed in detail elsewhere in this volume, so our focus here is on how a few specific genes may be involved in G × E interactions.

I. Heritability of Aggression: Twin and Adoption Studies

Behavioral genetic research relies on the different levels of genetic relatedness between family members in order to estimate the relative contribution of heritable and environmental factors to individual differences in a phenotype of interest. Major research designs include:

(a) studies of twins raised together and (b) studies of adopted individuals and their biological and adoptive family members. Although designs combining both approaches are the most powerful for separating genetic and environmental effects in human behavior, such studies of twins separated at birth and raised apart are rare and have not studied aggressive behavior extensively. Nonetheless, there are a handful of adoption studies and over two dozen studies of twins raised together which have specifically examined the genetic and environmental influence in aggression in nonselected samples from Northern America and Europe that are reasonably representative of the general population.

In the classical twin design, monozygotic (identical) twins share their common environment and they are assumed to share 100% of their genes. Dizygotic (fraternal) twins also share their common environment and they are assumed to share on average 50% of their genes. By comparing the resemblance for aggressive behavior between monozygotic and dizygotic twins, the total phenotypic variance of aggression can be divided into additive genetic factors (or heritability, h^2), shared environmental factors (c^2), and nonshared environmental factors (e^2). Shared environmental factors refer to nongenetic influences that contribute to similarity within pairs of twins. Nonshared environmental factors are those individual experiences that cause siblings to differ in their levels of aggressive behavior. Heritability is the proportion of total phenotypic variance due to genetic variation (Neale and Cardon, 1992). Genetic influences may also be divided into those that are additive (i.e., allelic effects add up across loci) and those that are nonadditive (i.e., due to dominance or epistasis). In twin studies, however, it is not possible to estimate both additive and nonadditive genetic effects (d^2) simultaneously with shared twin environment effects. The twin correlations summarized in Table 8.2 can be used to estimate the genetic and environmental influences to aggressive behavior. Twice the difference between the MZ and DZ correlations provides an estimate of the relative contribution of additive genetic influences to aggressive behavior [$h^2 = 2(r_{MZ} - r_{DZ})$]. The contribution of the nonadditive genetic effects due to dominance or epistasis (d^2) is obtained by subtracting four times the DZ correlation from twice the MZ correlation ($d^2 = 2r_{MZ} - 4r_{DZ}$). The proportion of the variance that is due to shared environmental influence is given by subtracting the MZ correlation from twice the DZ correlation ($c^2 = 2r_{DZ} - r_{MZ}$). Finally, the contribution of the nonshared environmental influences can be obtained by subtracting the MZ correlation from unit correlation ($e^2 = 1 - r_{MZ}$) (Posthuma *et al.*, 2003). Many twin studies do not specifically examine or test for nonadditive genetic effects and instead report heritability estimates based on additive effects only. However, some twin studies compare models with additive effects and nonadditive effects versus models with additive genetic effects and shared environment.

In sibling adoption studies, the correlation between adoptive siblings is compared with the correlation between biological siblings to estimate the influence of genetic and environmental factors on aggressive behavior (Plomin *et al.*, 2001). Resemblance between adoptive siblings for measures of aggression is indicative of shared (or common) family environment, while the extent to which biological sibling resemblance exceeds that of adoptive siblings is taken as evidence of heritable genetic influences for aggressive behavior.

There have been a few meta-analyses of twin and adoption studies of aggressive behavior and the wider construct of antisocial behavior. In one early meta-analysis of 24 twin and adoption studies, heritable influences explained about half of the total variance in aggressive behavior and the nonshared environment explained the remaining 50% (Miles and Carey, 1997). Rhee and Waldman (2002) also summarized the results from 51 twin and adoption studies on criminal behavior, delinquency, psychopathy, conduct disorder, and antisocial personality disorder, as well as aggressive behavior, in children, adolescents, and adults. Genetic factors explained 41% of the variance in antisocial behavior, 16% was explained by

shared environmental influences, and the remaining 43% of variance was explained by nonshared environmental factors. A more recent review focused on 19 twin and adoption studies using child and adolescent samples; studies including adult subjects were excluded. Heritability was found to explain 65%, shared environment explained 5%, and the nonshared environment explained the remaining 30% of the variance in aggressive behavior (Burt, 2009). Both Burt (2009) and Rhee and Waldman (2002) examined nonadditive genetic effects, but only Rhee and Waldman (2002) found significant nonadditive genetic effects for antisocial behavior. It is noteworthy that genetic influences are consistently found across these reviews, while shared environmental influences are comparatively small or nonexistent. Family similarity in aggressive and antisocial behavior, therefore, is primarily the result of shared genes, not environment.

Tables 8.1 and 8.2, respectively, summarize a large selection of twin and sibling adoption studies which have specifically examined the genetic and environmental influences on aggressive behavior in child, adolescent, and adult samples. Several studies use prospective, longitudinal designs, and large samples, and three of the twin studies were designed, in particular, to study aggressive and antisocial outcomes. All three of these studies are ongoing. One of these is the University of Southern California Twin Study of Risk Factors for Antisocial Behavior (RFAB), which is a prospective study of the interplay of genetic, environmental, social, and biological (psychophysiological) factors on the development of antisocial and aggressive behavior from childhood to emerging adulthood. The project includes more than 750 twin pairs studied on several occasions, at ages 9–10, 11–13, 14–16, and 17–18 years (Baker *et al.*, 2006). A second major twin study is the Environmental Risk Longitudinal Twin Study (E-risk study) in the United Kingdom. The E-risk study involves data on more than 1000 twin pairs at ages 5, 7, and 12 with the special focus on what factors in the home, family, school, and neighborhood (i.e., environmental risks) promote children's aggression (Moffitt, 2002). The Minnesota Study of Twins and Families (MFTS) is a third major longitudinal twin study that specifically investigates antisocial behavior and substance use across development. MTFS was established in 1989 using same-sexed twin pairs aged 11 or 17. Five hundred additional 11-year-old twin pairs were added in 2000. All twins of those ages, who were born in Minnesota, as identified by birth registry data, were invited to participate. Participants are asked about academic ability, personality, and interests; family and social relationships; mental and physical health; and physiological measurements. Of particular interest are prevalence of psychopathology, substance abuse, divorce, leadership, and other traits and behaviors related to mental and physical health, relationships, and religiosity (Iacono *et al.*, 2006; Keyes *et al.*, 2009).

Before reviewing the twin and sibling adoption studies on aggressive behavior presented in Tables 8.1 and 8.2, it is important to consider the ways in which the phenotype of aggressive behavior is defined and measured. The various instruments utilized in the studies reviewed in this chapter are summarized in Tables 8.1 and Tables 8.2, to provide a clear idea of the nature of the aggressive behavior phenotype being investigated. By and large, the Child Behavior Checklist (CBCL; Achenbach, 1991b) has been used more often than any other single instrument in behavioral genetic studies of aggression. Although self-report version of the CBCL is available for older adolescents and young adults (Youth Self Report (YSR); Achenbach, 1991c), studies more commonly rely on parent or teacher (Teacher's Report Form (TRF); Achenbach, 1991a) rating versions. The aggressive behavior subscale of the CBCL includes 20 items on which the child is rated. These include defiance, argumentativeness, physical aggression, and cruelty toward others. Although there are a handful of other instruments that also yield single aggressive behavior scores, two instruments provide multiple scales: the Reactive and Proactive Aggression Questionnaire (RPQ; Raine *et al.*, 2006), which provides separate scales for aggressive reactions to provocation and more planned or proactive forms of aggression; and the Buss-Durkee

Hostility Inventory (BDHI; Buss and Durkee, 1957), which yields several sub-scales of aggression, including assault, verbal, and indirect hostility.

The studies summarized in Tables 8.1 and Tables 8.2 vary on how aggressive behavior was defined (i.e., physical, verbal, relational, reactive, proactive, indirect, bullying) and measured (observation, self-report, parent/caregiver, teacher). A wide range of ages were included, from preschool children to adults; however, the vast majority of studies have used childhood samples (i.e., 12 years of age or younger) which explains why the CBCL is so frequently used to assess aggressive behavior. Correlations for biological and adoptive siblings (Table 8.1), and MZ and DZ twins (Table 8.2) are shown for each study. Most studies reported correlations separately for same-sex pairs of males (M), females (F), and opposite-sex pairs (MF); however, a few studies involve correlations for samples of male and female pairs combined. We review the key questions concerning the genetic influence (heritability) of human aggression based on the effect sizes reported for these studies. We also examine various potential moderators of these effects, including sex, age, method of assessment, form of aggression, study design (twin vs. sibling adoption design), and various social factors and circumstances that may exacerbate or ameliorate the genetic risk for aggression from one person to the next.

A. Does heritability vary depending on sex?

Since it is well documented that males are much more likely than females to engage in most forms of aggressive behavior (Moffitt *et al.*, 2001; Rutter *et al.*, 2003), it is also of interest to examine whether the same genetic and environmental influences are important in both sexes and whether the magnitude of these effects differs between males and females.

In the classical twin design, genetic and environmental variance components for aggressive behavior can be estimated using data from same-sex MZ and DZ twins. Apart from estimating genetic and environmental effects on aggression, it is also possible to investigate whether sex-specific genetic or environmental influences are important. Such effects are referred to as sex-limitation or sex-limited effects. There are two primary questions about sex limitation in genetic research, one being whether there are *qualitative* differences between males and females, such that different genes and/or environmental influences operate in the two sexes, and whether *quantitative* differences exist in the relative magnitude of influences across sexes. To assess whether the magnitude of genetic and environmental effects in aggressive behavior differ between males and females (i.e., quantitative sex differences), only data from same-sex twin pairs are required. However, to determine whether or not it is the same set of genes or shared environmental experiences that influences aggressive behavior in males and females (i.e., qualitative sex differences), data from opposite-sex twin pairs are also needed. If qualitatively different genetic influences are important for aggressive behavior in males and females, then the opposite-sex twins will be less genetically similar for the trait than DZ twins.

Not all twin studies have examined sex-limited effects, either qualitative or quantitative, and several studies combined males and females when computing twin correlations, making it impossible to evaluate these effects based on published results shown in Table 8.2. Nonetheless, quantitative sex differences can be easily evaluated across at least 18 studies in Table 8.2, which present separate twin or sibling correlations by sex. Among these, there are a dozen studies that also include MF, which allow investigation of qualitative sex differences. The average twin correlations across these 18 studies, weighted by their respective sample sizes, shows quite similar twin correlations for both identical ($r_{MZ–Males}$= 0.66; $r_{MZ–Females}$= 0.63) and nonidentical same-sex pairs ($r_{DZ–Males}$= 0.42; $r_{DZ– Females}$= 0.35), indicating that there are no appreciable quantitative sex differences in aggressive behavior. This is consistent with the individual results across studies which formally tested

for quantitative sex differences (e.g., Baker *et al.*, 2008; Czajkowski *et al.*, 2008; Eley *et al.*, 1999; Finkel and McGue, 1997; Tackett *et al.*, 2009; Tuvblad *et al.*, 2009). As indicated in Table 8.2, only a small handful of studies have reported significant differences in heritability of aggression for males and females (and these are primarily for younger samples; e.g., Hudziak *et al.*, 2003; van Beijsterveldt *et al.*, 2003; Vierikko *et al.*, 2004). The lack of quantitative sex differences is also well in line with what was reported in a recent meta-analysis summarizing 19 twin and family/adoption studies, whereby genetic influences were found to explain 54% of the variance in aggressive behavior in boys and 57% of the variance in girls (Burt, 2009).

There is no evidence of qualitative sex differences either, given that the weighted twin correlation for MF ($DZ_{Male–Female}$) is 0.38, which is quite similar to the same-sex DZ twin correlations (0.42 in males and 0.35 in females).

In spite of the consistent sex difference in mean levels of aggression, the underlying etiologies of aggressive behavior appear to be remarkably similar for both sexes. There may still be biological and social differences between the sexes that might account for the greater mean levels of aggression observed in males, yet the same genes and the same environmental factors appear to explain individual differences in aggression within each sex to the same degree. One interesting question that has not been addressed, however, is to what extent there may be sex differences in *moderators* of genetic factors. In other words, there may be different circumstances or experiences in males and females that lead to greater expression of genetic predispositions for aggression. For example, sexual jealousy might trigger genetic propensity for aggression to a greater extent in males than females, while threats to resources might be a more important moderator of genetic influences in females compared to males, as discussed in Chapter 9. Other moderators are discussed later in II.A, although more research is clearly warranted to explore the degree to which they may be sex specific.

B. Does heritability change across age?

Although genetic studies of aggression have spanned from childhood to adulthood, most studies included in Tables 8.1 and Tables 8.2 involved children 12 years of age or younger. This suggests that more studies examining the heritability of aggressive behavior in adolescents and adults are needed. Keeping this in mind, it is useful to examine the magnitude of twin correlations across age groups, which span from early childhood to middle-age adults. These correlations are summarized in Fig. 8.1, according to five age groups (early childhood, age 1.5–6 years; middle childhood, age 7–10; adolescence 11–15; late adolescence/young adulthood, age 16–26; and adulthood, age 27–48; Fig. 8.1). These results show that aggressive behavior is clearly influenced by genetic factors across the lifespan, given the fact that the MZ correlations exceed those for DZ pairs at all ages. (The lack of qualitative sex differences is also evident across the life span, in that the DZ correlation is comparable for same-sex and MF pairs across ages.) However, both MZ and DZ correlations decline steadily across development, suggesting the waning importance of shared environmental effects from childhood to adolescence and then adulthood. The DZ correlation exceeds half the value of the MZ correlation (taken as evidence for shared environment) only in early childhood, but not in later age groups. The pattern shown in Fig. 8.1 is evident in individual studies as well. Aggressive behavior in childhood is influenced by genetic factors in all studies, and most of these studies also report shared environmental influences (Table 8.2; e.g., Baker *et al.*, 2008; Eley *et al.*, 1999; Hudziak *et al.*, 2003; Schmitz *et al.*, 1995; Simonoff *et al.*, 1998; Tuvblad *et al.*, 2009; van den Oord *et al.*, 1996; Vierikko *et al.*, 2004; but for an exception see Dionne *et al.*, 2003; Taylor, 2004). Studies including adolescent twins (~ younger than 19 years of age) do not in most cases report finding any shared environmental influences (Table 8.2; e.g., Button *et al.*, 2004; Cho *et al.*,

2006; Gelhorn *et al.*, 2006; Tackett *et al.*, 2009). Similarly, studies including adult twins do not report finding any shared environmental influences (Table 8.2; e.g., Coccaro *et al.*, 1997; Finkel and McGue, 1997; von der Pahlen *et al.*, 2008; but for an exception see Czajkowski *et al.*, 2008). The overall pattern across the studies presented in Table 8.2 and Fig. 8.1 indicates that genetic influences for aggressive behavior become increasingly more important, while shared environmental effects become less so as children develop from childhood, through adolescence, and into adulthood. Similarly, findings from a recent meta-analysis reported that genetic influences increased from 55.2% at ages 1–5 years to 62.7% at ages 6–10 years and 62.9% at ages 11–18 years. At the same time, shared environmental influences were decreasing from 18.7% at ages 1–5 years to 13.9% at ages 6–10 years and 2.7% at ages 11–18 years (Burt, 2009). This pattern of decrease in shared environment, and a concomitant increase in heritability during development, is relatively common for personality traits and cognitive abilities (Bartels *et al.*, 2002; Loehlin, 1992; Plomin *et al.*, 2001; Scarr and McCartney, 1983), and has also been found for other phenotypes including prosocial behavior (Knafo and Plomin, 2006).

It should also be kept in mind, however, that methods of assessing aggression vary across age, such that studies of children tend to rely on ratings by teachers and parents, while studies of adults (and some older adolescents) rely more heavily on self-report methods. The confound between method of assessment and age of the subjects has made it difficult in prior studies and metaanalyses to disentangle age effects on heritability from differences that arise from different methods of assessment. Increasing heritability estimates from child to adulthood could therefore also be explained by different methods of assessment as well (e.g., parental bias may lead to overestimation of shared environmental effects and thus attenuate heritability estimates in childhood).

C. Do heritabilities vary across methods of assessment?

It is important to examine the magnitude of twin correlations across methods of assessment, as heritability estimates may vary depending on who is rating the subject. This is especially important given the age trends found for heritable influences in Fig. 8.1, since different methods of assessment tend to be employed for different age groups. As previously discussed, studies of younger subjects rely on parent or teacher ratings, while self-report methods are typically used in studies of adults and often adolescents. The twin correlations are summarized in Fig. 8.2, according to laboratory observation, self-reports, teacher ratings, and parent/caregiver ratings. Indeed, twin correlations—and thus the estimates of genetic and environmental influences on aggressive behavior—do appear to vary across method of assessment. According to the twin correlations summarized in Fig. 8.2, the heritability of aggressive behavior based on laboratory observation is approximately 32% [h^2: $2(r_{MZ}-r_{DZ})=2(0.27–0.11)$], dominant genetic effect accounts for approximately 10% [d^2: $2(r_{MZ}-r_{DZ})=2(0.27)–4(0.11)$], and the nonshared environment accounts for the remaining 58% of the variance. The heritability of aggressive behavior based on self-reports is 40% and the nonshared environment accounts for the remaining 60% of the variance. There is no evidence of shared environmental contribution, as the DZ correlation is approximately half the MZ correlation. The heritability of aggressive behavior based on teacher ratings is 54%, shared environmental influences account for 6% [c^2: $2r_{DZ}-r_{MZ}=2(0.33–0.60)$], and the nonshared environment accounts for the remaining 40% of the variance. The heritability of aggressive behavior based on parent/caregiver ratings is 54%, shared environmental influences account for 17%, and the nonshared environment accounts for the remaining 29% of the variance. Thus, parent/caregiver ratings have the largest familial influence, explaining 71% of the variance ($h^2 + c^2 = 0.54\% + 17\%$) in individual differences in aggressive behavior. It should be kept in mind that this is a descriptive approach and that formal modeling is required to determine how well these estimates describe the observed data.

Also, this approach does not allow for actual testing of different hypotheses, for example, to test whether it is possible to set any of these effects to zero. It is difficult to discern whether the parent/caregiver rating patterns reflect true shared environment or are instead an artifact of rater bias whereby raters are less able to discriminate between the two twins' aggressive behavior and thus inflate the similarities between them, regardless of zygosity. In fact, when the two co-twins are rated by different teachers (e.g., they are in different classrooms), twin correlations are lower for both MZ and DZ pairs for the wider construct of antisocial behavior (Baker *et al.*, 2007).

A few specific twin studies in Table 8.2, which utilize multiple raters in their design, also illustrate that genetic and environmental influences on aggressive behavior vary depending on method of assessment. For example, twin similarity for relational aggression was influenced only by genetic factors when using self-reported data, explaining 49% of the total variance. When using parent ratings (only biological mothers were used as raters) of relational aggression both genetic and shared environmental influences were important (boys: h^2 = 21%, c^2 = 46%, e^2 = 33%; girls: h^2 = 42%, c^2 = 22%, e^2 = 36%). However, when using youth self-reports, only genetic and nonshared environmental influences were significant (h^2 = 49%, e^2 = 51%; Tackett *et al.*, 2009). A similar pattern was found for reactive and proactive aggression in 9–10-year-old boys, whereby only genetic influences were important for self-reports, but both genes and shared environment were important for teacher and parent ratings (Baker *et al.*, 2008). Aggressive behavior in another sample of twins aged 7–12 years was found to be largely influenced by genetic (or familial) factors (76%–84%), as reported by parents. Data were collected from one or both parents; however, only mother-reported ratings were included in the analyses, as they accounted for the majority of the ratings collected (85.3%–90.1%). In contrast, when teacher ratings were used, aggressive behavior was found to be slightly less influenced by genetic (or familial) factors (42%–61%; Haberstick *et al.*, 2006a). Significant shared environmental effects were not found for either parent or teacher ratings, and were therefore dropped from the models, suggesting that any shared environmental influences are likely to be included in the genetic component. Apart from rater bias, which may result in inflated twin similarity across the board when using single raters for multiple children, the varying patterns of genetic and environmental influence across methods of assessment could be the result of different raters reporting different aspects of the child's aggressive behavior. This could arise in part because individuals behave differently in different situations (e.g., school vs. home) or because some types of aggressive behaviors are more likely to be noticed (e.g., overt forms such as physical aggression) than other types of aggressive behaviors which may be more subtle or covert (e.g., relational aggression). Different raters provide important and unique pieces of information regarding behaviors. Self-reporters are aware of their own motives and behaviors, which may go undetected by their caregivers, teachers, or peers. On the other hand, caregivers or teachers may be able to understand difficult and complex constructs better than children. A teacher is also more likely to compare a child's behavior to his or her peers, whereas a parent is likely to compare a child's behavior to his or her siblings (Bartels *et al.*, 2003). Regardless of the source of these discrepant results across methods of assessment, it is important to keep in mind that when it comes to studies of aggression, it matters who is doing the rating.

D. Do heritabilities vary across forms of aggression?

Different types of aggressive behavior have been investigated across twin and adoption studies, with notable distinctions between reactive and proactive forms of aggression, as well as direct/physical and indirect/relational aggression (Table 8.3). It is likely that there are different etiologies for different forms of aggression; for example, defensive reactions to threatening stimuli may be more environmentally influenced, while more planned, proactive

forms may be more genetically influenced (Tuvblad *et al.*, 2009). Comparing heritability estimates collectively across the various measures employed is a reasonable way to address this question about whether some kinds of aggression are more heritable than others. When multiple forms of aggressive behavior are measured within the same study, it is also possible to investigate the extent to which the same genes and/or environmental factors are important to different manifestations of aggressive tendencies.

Reactive aggression refers to angry or frustrated responses to a real or perceived threat. This specific type of aggression has been characterized to involve both high emotional arousal, impulsivity, and an inability to regulate or control affect. In contrast, proactive aggression is conceptualized as a more regulated, instrumental form of aggression, with more positive expectancies about the outcomes of aggression (Dodge, 1991; Dodge and Coie, 1987; Schwartz *et al.*, 1998). Although reactive and proactive aggression have each been found to be mainly influenced by genetic and nonshared environmental factors, their genetic correlation is significantly less than 1.0, indicating some genetic specificity for the two forms of aggression. Reactive and proactive aggression each exhibit different developmental patterns in these influences (see Table 8.2; Baker *et al.*, 2008; Tuvblad *et al.*, 2009), that is, the genetic and environmental *stability* in reactive and proactive aggression has been found to differ. In one of the few longitudinal analyses of these constructs, the stability in reactive aggression from childhood to adolescence could be explained by genetic (48%), shared (11%), and nonshared (41%) environmental influences, whereas the continuity in proactive aggression was primarily genetically (85%) mediated (Tuvblad *et al.*, 2009).

Relational forms of aggression, which involve social manipulation such as gossip and peer exclusion, are often more indirect compared to other forms of aggression (Crick and Grotpeter, 1996). Like reactive and proactive forms, relational aggression appears to be influenced by genetic factors, both in self-report (49%) as well as parental reports (boys, 42%; girls, 21%). However, unlike the other more direct forms of aggression, relational aggression is also influenced by shared environmental influences, but only in parental reports (boys, 22%; girls, 46%) and not in self-report measures (Tackett *et al.*, 2009). Together these findings—that is, the less than perfect genetic correlation between reactive and proactive forms, their different developmental etiologies, and the significant shared environmental effects in relational aggression only— provide support for at least some genetic and environmental etiological distinction among different forms of aggression. It should be noted, however, that no study to date has examined the genetic and environmental overlap between relational aggression and other forms such as proactive and reactive aggression.

Other studies based on multifactorial measures of aggression, such as the BDHI, suggest some variability in the heritability estimates across subscales, although the patterns are not entirely consistent across studies. For example, “indirect hostility” showed the lowest heritability (28%) in one study of adult twins (Coccaro *et al.*, 1997), compared to modest heritability for “verbal hostility” (47%) and “assault” (40%). Yet, Cates *et al.* (1993) found no genetic influences for assault, but strong heritability for both verbal hostility (78%) and indirect hostility (70%). In multivariate genetic analyses, both studies found some support for genetic specificity for the various subscales, similar to what has been found for reactive and proactive aggression, in that genetic correlations (r_G) among the BDHI subscales were less than unity: $r_G = 0.39$ between indirect hostility and assault, $r_G = 0.60$ between indirect hostility and verbal hostility, $r_G = 0.17$ between verbal hostility and assault (Coccaro *et al.*, 1997), $r_G = 0.35$ between indirect hostility and assault, $r_G = 0.39$ between indirect hostility and verbal hostility, and $r_G = 0.49$ between verbal hostility and assault (Cates *et al.*, 1993). Overall, genetic influences are generally found for most, if not all forms of aggression, although somewhat different genetic factors may be operating across these different forms.

The mechanisms that underlie more direct, planned, confrontational, and often physical forms of aggression may to some extent be different than those for reactive or indirect aggressive behaviors.

E. Does heritability vary depending on study design (twins vs. adopted siblings)?

There were only a handful of studies identified examining the heritability of aggressive behavior using the sibling adoption design. Visual inspection of the results from these sibling adoption studies (see Table 8.1) compared to the results from studies including twin samples (Table 8.2) indicate that heritability estimates (i.e., h^2) and the shared environmental estimates (i.e.,c^2) for aggressive behavior are very similar. This is also well in line with the results of a meta-analysis on antisocial andaggressive behavior that foundnodifferences between twin and siblingadoption studies (h^2 = 48%; c^2 = 13%, e^2 = 0.39%) (Rhee and Waldman, 2002). Thus, the heritability of aggressive behavior does not seem to vary depending on study design.

F. Criticisms of twin and adoption studies: Assumptions and generalizability

There are several assumptions in both twin and adoption studies that are important to consider when reviewing their findings. In adoption studies, the most important factors are (1) random placement of the adoptees into homes and (2) generalizability. Selective placement or matching (i.e., similarities between adoptive and biological parents) for certain characteristics can lead to inflated correlations between adoptive siblings (and thus overestimated effects of shared environment). Although such matching may occur for physical characteristics (including race), direct selective placement is unlikely to be made for aggressive behavior, per se. (Children with more aggressive or antisocial biological parents would not be placed into homes with more aggressive adoptive parents.) Thus, it is unlikely that the genetic and environmental effects summarized in Table 8.1 are biased in any way as a result of selective placement. In terms of generalizability, it is often the case that adoptive parents tend to be in good health and from higher socioeconomic levels; thus, findings from adoption studies may not always be unquestionably generalized to the entire population (Rutter, 2006). Adopted children may also be at greater risk for aggression compared to nonadoptees, since birth parents giving up their children may have increased rates of disordered behaviors, including substance use, criminal offending, and aggression (Cloninger *et al.*, 1985; Lewis et al., 2001). In the Deater-Deckard and Plomin (1999) study, the adopted children did in fact have higher aggression scores compared to the nonadopted children, consistent with the notion that adoptees may be at higher genetic risk for aggression compared to nonadopted individuals. The elevated levels of aggression in adoptees occurred in spite of the fact that background characteristics of the adoptive families were found to be representative of families with children in the larger Denver area, and that the demographic characteristics of the adoptive grandparents and the adopted children's biological grandparents were similar, with regard to educational and occupational level. Similarly, van der Valk *et al.* (1998) reported mean differences between adoptees and nonadoptees, with adoptees showing higher mean levels in aggressive behavior. About 75% of the adoptees were adopted from Korea, India, Columbia, Indonesia, Bangladesh, or Lebanon and the remaining 25% were adopted from European or other non-European countries in both the van der Valk *et al.* (1998) and the van den Oord *et al.* (1994) studies, and the majority of the adoptive parents had a higher level of occupation. Given the higher aggression scores among adoptees compared to nonadoptees, as well as the somewhat greater affluence and ethnic heterogeneity in at least some of the adoption samples, the generalizability of adoption study results to the wider population could be questioned.

To what extent are the twin study results generalizable to the wider population? Twins and singletons have been found to experience similar rates of psychiatric disorders (e.g.,

attention-deficit hyperactivity disorder (ADHD), oppositional defiant disorder, conduct disorder) and behavioral and emotional problems (Gjone and Novik, 1995; Moilanen *et al.*, 1999; Simonoff *et al.*, 1997; van den Oord *et al.*, 1995). Findings from the RFAB study show no differences in mean levels between MZ and DZ twins in reactive or proactive aggression (Baker *et al.*, 2008; Tuvblad *et al.*, 2009). It can, therefore, be assumed that twins and singletons display equal rates of aggressive behavior.

There are, however, two ways in which twins differ from singletons: (i) lower birth weight, due to shorter length of gestation (Plomin *et al.*, 2001) and (ii) delayed language development (Rutter and Redshaw, 1991). Birth weight has been found to have a minimal effect on academic performance; for twins this effect was judged relative to what is a normal birth weight for twins and not for singletons (Christensen *et al.*, 2006). However, studies have shown that children with birth complications are more likely to later develop antisocial and aggressive behavior (Raine, 2002), but birth complications may not by themselves predispose antisocial and aggressive behaviors, but will require the presence of an environmental risk factor (e.g., poor parenting, maternal rejection). In other words, the relationship between birth complications and antisocial and aggressive behavior is confounded by environmental risk factors (Hodgins *et al.*, 2001; Raine *et al.*, 1997).

In addition to generalizability, there are several key assumptions of the classical twin design that need to be kept in mind when reviewing findings from these studies. These include (1) the equal environments assumption, (2) random mating, and (3) lack of correlation or interaction between genetic and environmental influences. We briefly review each of these assumptions here—both in general and as they pertain to aggressive behavior in particular—and consider their possible effects on the results summarized across studies.

Perhaps the most important and commonly criticized assumption is the “equal environment assumption” (EEA). In the classical twin design, MZ twins, who are assumed to share 100% of their genes, are compared to DZ twins, who are assumed to on average share 50% of their genes. If MZ twins are more similar than DZ twins, it may be inferred that the difference is caused by genetic effects. To make this inference, however, it is necessary to rely on the EEA. It is assumed that environmentally caused similarity is roughly equal for both MZ and DZ twins. If this assumption is violated, higher correlations among MZ twins may be due to environmental factors, rather than genetic factors, and heritability estimate will be overestimated (Plomin *et al.*, 2001).

Several twin studies of various phenotypes have examined the EEA. One way to test the validity of the EEA is to examine whether a trait of interest is influenced by perceived versus assigned zygosity. The effect of perceived zygosity can be added as a “specified” familial environment in a univariate ACE twin model (Kendler *et al.*, 1993) and if this parameter can be omitted without any significant loss in data fit, it can be assumed that the EEA holds for the phenotype under study. These studies generally report that the EEA assumption holds for numerous phenotypes such as physical activity, eating behavior, psychiatric disorders (e.g., major depression, generalized anxiety disorder, phobia, alcohol, and drug abuse; Eriksson *et al.*, 2006; Hettema *et al.*, 1995; Kendler *et al.*, 1993; Klump *et al.*, 2000; Xian *et al.*, 2000), including child and adolescent psychopathology such as anxiety disorder, ADHD, oppositional defiant disorder, conduct disorder, antisocial behavior (Cronk *et al.*, 2002; Jacobson *et al.*, 2002; Tuvblad *et al.*, 2011) as well as aggressive behavior (Derks *et al.*, 2006).

The assumption of random mating for aggression in the parents of the twins is also important to consider, since nonrandom mating can lead to increased resemblance for DZ but not MZ twin pairs. Assortative mating in the parent generation acts to increase the

resemblance between dizygotic twins and thereby bias shared environmental estimates upward and additive genetic effects downward. A significant correlation between spouses for a particular trait is often interpreted as assortative mating (Maes *et al.*, 1998). This assumption is probably violated when it comes to antisocial and aggressive behavior, as significant spouse correlations have been found suggesting that assortative mating exists in this behavioral domain (Krueger *et al.*, 1998; Maes *et al.*, 2007; Taylor *et al.*, 2000). Taylor *et al.*, 2000 found that parents of twins were correlated for retrospectively reported delinquency ($r = 0.23$ in families of boys and $r = 0.35$ in families of girls) and concluded that assortative mating is modest in degree. Another study using data from the Dunedin sample in New Zealand (Silva and Stanton, 1996) when the participants were 21-years-old found a correlation ($r = 0.54$) between couple members' reports of antisocial behavior in their peers (i.e., participants were asked how many of their friends had aggression, personal, alcohol, or drug problems, or did things against the law), which was identical to the correlation for couple members' reports of their own antisocial behavior as measured by a variety of offenses (e.g., theft, force, fraud, vice). They concluded that assortative mating for antisocial behavior is substantial and that antisocial individuals tend to cluster in peer groups with similar antisocial peers. As such, assortative mating should to be taken into account when modeling antisocial behavior (Krueger *et al.*, 1998). It is interesting, however, that the shared environmental effects are fairly negligible in twin studies of aggressive behavior, both in the prior meta-analyses as well as in our summary in Table 8.2. Thus, any assortative mating for aggression does not appear to have resulted in severe overestimates of shared environment when considering these studies *en masse*. It is possible, on the other hand, that genetic influences themselves have been underestimated and could be larger than the 50% or so than these meta-analyses and our summary suggest.

It is also assumed in the classical twin design that genetic and environmental influences combine additively (i.e., do not interact) and are uncorrelated. It is possible, however, that some genetic predispositions may be associated with certain kinds of social environments or experiences, leading to a correlation between genes and environments. (G × E interactions are also discussed at length in a later section of this chapter.) Such G × E correlations (r_{GE}) can arise in three different ways (Scarr and McCartney, 1983): (i) Passive r_{GE} occurs when genes overlap between parents and their offspring. For example, a child with aggressive parents inherits genetic susceptibility for aggression as well as experiences an adverse rearing environment. An example of passive r_{GE} was reported in a study comparing genetic and environmental influences on mothering. Passive r_{GE} correlations were suggested for mother's positivity and monitoring. For mother's negativity and control, primarily nonpassive r_{GE} correlations were suggested (Neiderhiser *et al.*, 2004). (ii) Evocative/reactive r_{GE} can arise when a specific child characteristic elicits a particular response from the environment. For example, aggressive children tend to elicit more negative affect and harsh discipline from their parents (Ge *et al.*, 1996; O'Connor *et al.*, 1998). In a more recent study, using the classical twin design the association between parental criticism and adolescent antisocial behavior was found to be entirely genetically influenced. Approximately half of the genetic contribution to this association was explained by early adolescent aggression. Thus, child aggression seemed to elicit negative parenting followed by adolescent antisocial behavior, indicating an evocative r_{GE} (Narusyte *et al.*, 2006). (iii) Active r_{GE} is defined as the process whereby an individual actively seeks out environmental situations that are more closely matched to the person's genotype. Active r_{GE} has been suggested in adolescent drinking behavior, specifically among girls (Loehlin, 2010). Ifthe assumption of no G × E correlation is violated, heritability estimates for aggressive behavior in twin studies could include both additive genetic effects and the effects of G × E correlation (i.e., heritability estimates are inflated). Apart from these specific examples cited here, few studies have examined the effects of r_{GE} in aggressive behavior, making it difficult to know the extent of their effect on heritability estimates in twin studies.

In conclusion, findings from adoption studies should probably be generalized cautiously to other populations as adoptees tend to show higher scores on aggressive behavior compared to controls. On the other hand, most of the assumptions of the classical twin design seem to hold for aggressive behavior. The EEA has been tested and found to hold for various phenotypes including aggressive behavior, and twins and singletons have been found to display similar scores on aggressive behavior, suggesting that findings from twin studies can be generalized to other populations. Most twin studies report finding little or no shared environmental influences on aggressive behavior, suggesting that random mating is of little importance for aggressive behavior. Only a few studies have examined the influence of G × E correlation on aggressive behavior, suggesting that more research is needed on this topic before we can draw any firm conclusions. Last, in the classical twin design, it is assumed that genetic and environmental influences combine additively and do not interact. This assumption is probably violated to some extent when it comes to aggressive behavior, as several studies have reported finding significant interaction effects. G × E interaction is discussed in detail in the next section of this chapter.

II. G × E Interaction in Aggressive Behavior

There is a general recognition that genes and environment work together—often in complex ways—to produce wide variations in behavior and psychological function. G × E interaction, by definition, is a statistical term indicating that genetic effects on a given phenotype depend upon environmental factors or vice versa. Gene expression, for example, can be moderated by an individual's experiences or exposure to certain environments. Likewise, various individuals may respond differently to the same environmental exposure because they have different genotypes. Such genetic sensitivity to the environment has been demonstrated extensively in plant and animal species for a variety of traits. But even though the importance of G × E interactions in human behavior has long been considered (Eaves, 1984; Mather and Jinks, 1982), G × E interactions have been rarely reported in human traits until relatively recently. The failure to find G × E interactions in studies of human characteristics may be due to a number of factors. One likely explanation is related to statistical power. In general, it is difficult to detect G × E effects due to their low statistical power (Rowe, 2003). For example, behavioral genetic studies rely on genetic relatedness for groups of individuals, rather than on sharing of specific alleles between pairs of relatives. Studies relying on variance partitioning often do not find significant G × E effects, or find that they explain a very small portion of the total variance, and are thus dropped from further analysis. When G × E is not taken into account in behavioral genetic studies, heritability estimates will tend to be biased, although the direction of the bias depends on whether the moderating environmental influences are of the shared or nonshared variety.

G × E interactions can be tested or modeled in behavioral genetic studies using several different study designs (e.g., twin or adoption). The two most frequently used methods testing for G × E interactions in twin and adoption studies include: (1) a *mean levels approach*, testing whether mean values of a phenotype differ across different combinations of genetic risk and environmental settings and (2) *a moderated variance components approach*, examining whether genetic and environmental variance for a trait varies across different measured environmental settings. These two different methods stem from the same conceptual idea, namely, that genetic effects vary across environments or vice versa. Their interpretations and meanings can be rather different, since one is based on means and the other is based on variances. The mean levels G × E is perhaps a more traditional approach, is typically presented as a statistical interaction in an ANOVA, and indicates whether particular experiences, exposures, or other conditions may (on average) ameliorate or exacerbate specific genetic effects in groups of individuals with similar genetic and environmental risks. In comparison, the moderated effects approach does not address mean

levels of risk, but instead evaluates whether variance in genes or environment differs across various measured conditions. Moderation can occur in either raw variances (V_A, V_C, and V_E) or in relative effects (i.e., h^2, c^2, *and* e^2), and may not necessarily coincide with G × E interactions found in mean levels. The latter point is important when evaluating G × E interactions across studies using these different approaches, since different patterns can emerge from them. For example, it is possible that certain adverse environments (e.g., low socioeconomic status (SES)) may lead to some genes exerting stronger effects (mean levels), while overall, the relative variance explained by genes (heritability) may be greater in other environments (e.g., high SES). The approach used for testing G × E interactions can vary across study design, such that adoption designs or studies with measured genes are generally required for the mean levels approach, while the moderated variance components approach may be used in both twin and adoption studies in the absence of measured genes.

In molecular genetic studies, both genes and environment are measured, rather than inferred from correlations among family members. G × E interactions can therefore be tested in the general population, that is, without necessary reliance on a twin or adoption design. There are still advantages, nonetheless, to include measured genes and measured environments in the context of a family-based design, including twins and other siblings as well as parents and offspring.

Evidence of G × E interaction in aggressive behavior has been reported in twin and adoption studies, and more recently in molecular genetic studies. Below is a summary of some of the G × E interaction findings in aggressive behavior from adoption, twin and molecular genetic studies. We also discuss two potential moderators of genetic and environmental influences on aggressive behavior, exposure to media violence, and alcohol use.

A. Potential moderators of genetic influence found in adoption and twin studies

1. Family adversity and social disadvantage—G × E interaction for aggressive behavior has been found in several of the early adoption studies, using a mean levels approach. What these early adoption study findings generally showed was that early adverse environments had a greater negative impact on genetically “higher risk” children. Adopted children with criminal biological parents reared by a family where there was adversity showed higher rates of antisocial and aggressive behavior than adopted children with antisocial biological parents not raised in a home with adversity, and than adopted children raised in adversity who are not at higher genetic risk. For example, the interaction of inherited and postnatal factors was examined in about 800 Swedish men adopted at an early age. When both inherited factors and environmental risk factors were present, 40% were found to be criminal; if only genetic factors were present, 12.1% were criminal; if only environmental factors were present, 6.7% were criminal; and with neither inherited nor environmental factors being present, 2.7% were criminal (Cloninger *et al.*, 1982). The fact that 12.1% plus 6.7% is less than 40% would thus be an indication of G × E interactions. This finding was later replicated in females (Cloninger and Gottesman, 1987). It should be pointed out, however, that in the adoption design, the genetic risk factors themselves are considered in a general way, such that the exact nature of the genes is left unspecified, both in terms of which loci or alleles may be involved and what underlying mechanisms may be involved in the path from genes to phenotype. Similarly, the environmental risk factors as indexed by certain traits in the adoptive parents or characteristics of their home do not necessarily specify the exact nature of the child's experiences or how these lead to various outcomes.

Further, maltreatment places children at risk for psychiatric morbidity, especially conduct problems. However, not all maltreated children will develop conduct problems. A recent twin study tested whether the effect of physical maltreatment on risk for conduct problems

was strongest among those who were at high genetic risk for these problems using data from the E-risk study, a representative cohort of 1116 5-year-old British twin pairs and their families. Maltreatment was found to be associated with a greater increase in the probability of developing conduct problems among children who had a high genetic liability for conduct disorder compared to children who had a low genetic liability (Jaffee *et al.*, 2005). This finding is consistent with the G × E interaction found in adoption studies of antisocial and aggressive behavior, in which genetic effects were more pronounced in adverse environments. This clearly suggests that children in risky environments would benefit from interventions. However, another view of this interaction is that favorable genotypes can play a protective role on children's risk for conduct problems, especially under circumstances of maltreatment.

There are also a few studies based on twin samples that have used the moderated variance components approach to examine whether measured environmental (risk) factors moderate the genetic and environmental variances for aggressive behavior. For example, the heritability of conduct problems was found to be lower in children growing up in dysfunctional families and higher in children growing up in families where dysfunction was absent (Button *et al.*, 2005). Another twin study used DeFries-Fulker regression analysis to examine whether genetic and environmental influences on aggressive behavior varied depending on levels of family warmth (DeFries and Fulker, 1985). Genetic influence on aggressive behavior was found to be higher in schools with higher average levels of family warmth. In contrast, environmental influences (both shared and nonshared) were more important in schools with lower average levels of family warmth (Rowe *et al.*, 1999). These findings suggest that genetic effects are more likely to explain individual differences in aggression in more benign environments, whereas in more disadvantaged environments negative family-related factors and context-dependent risks may play a greater role than genetic predispositions in aggressive and antisocial outcomes.

Many early theories about the causes of delinquency and crime assumed that delinquents come from socially disadvantaged backgrounds. For example, Merton postulated that antisocial behavior resulted from the strain caused by the gap between cultural goals and the means available for their achievement (Merton, 1957). Social disadvantage and poverty constitute a reasonable robust, although not always a strong, indication of an increased risk for antisocial and aggressive behavior, assessed by self-reports and official convictions (Leventhal and Brooks-Gunn, 2000; Rutter *et al.*, 1998). SES has also been found to moderate the relative influence of genetic factors on antisocial and aggressive behavior. In a sample of Swedish 16–17-year-old twins, heritability for antisocial and aggressive behavior was higher in the more affluent neighborhoods (boys, 37%; girls, 69%) compared to the less advantaged neighborhoods (boys, 1%, girls, 61%). Conversely, the shared environment was higher in the less advantaged neighborhoods (boys, 69%; girls, 16%) compared to better-off neighborhoods (boys, 13%; girls, 6%). Following the “social push hypothesis,” Raine (2002) would suggest that the genetic factors on antisocial and aggressive behavior are more expressed in a socioeconomically advantaged environment where the environmental risk factors are absent. On the contrary, genetic factors for antisocial behavior will be weaker and the shared environment more important in a socioeconomically disadvantaged environment because the environmental risk factors will “camouflage” the genetic contribution (Tuvblad *et al.*, 2006).

These studies using the moderated variance components approach (e.g., Button *et al.*, 2005; Rowe *et al.*, 1999; Tuvblad *et al.*, 2006), all examine whether an environmental (risk) factor moderates genetic and environmental *variance* on antisocial and aggressive behavior. Findings from these studies show that heritable influences on aggressive behavior vary

depending on environmental context, indicating the importance of the environmental risk factors in the development of aggressive behavior as well as for gene expression.

2. Violent media exposure—There is an ongoing debate about whether exposure to violent video games increases aggressive behavior, and it is very possible that exposure to media violence could moderate the influences of genetic and environmental influences on aggressive behavior. One line of research argues that mass media exposures contribute to a child's socialization. A primary process in such socialization is observational learning (Bandura, 1973). Children and adolescents mimic what they see and acquire complicated scripts for behaviors, beliefs about the world, and moral precepts about how to behave in the long run from what they observe (Huesmann, 2010). In contrast, another line of research argues that there is little empirical evidence for a link between media exposure and violence. This line of research argues that media violence cannot have any important psychological effect on the risk for aggressive behavior (Ferguson and Kilburn, 2010).

A recent meta-analysis that included 136 studies examined the effects of violent video games on aggressive behaviors. The evidence suggested that exposure to violent video games is a risk factor for increased aggressive behavior, aggressive cognition, and aggressive affect, and for decreased empathy and prosocial behavior. Moderator analyses showed significant research design effects, weak evidence of cultural differences in susceptibility and type of measurement effects, and no evidence of sex differences in susceptibility. Sensitivity analyses were also carried out and they revealed these effects to be robust, with little evidence of selection (publication) bias (Anderson *et al.*, 2010).

Others studies examining the relationship between violent video games and aggressive acts have found little evidence for a relationship. A recent review included a total of 25 studies comprising 27 independent observations. The corrected overall effect size for all included studies was only $r = 0.08$ (Ferguson and Kilburn, 2009). The mixed findings in the literature clearly suggest that more research is needed to resolve whether there is a link between exposure to violent video games and aggressive behavior. Also, some studies have found that exposure to violent video games only explains a small fraction of the variance. An explanation for this paradox could be that exposure to violent video games moderates the influence of genetic and environmental effects on aggressive behavior, rather than exerting direct effects. No genetically informative studies have examined violent video game exposure as a possible moderator of genetic influence on aggression, however, leaving this as an important area in need of study.

3. Alcohol use—It has long been known that some individuals become aggressive after consuming alcohol, and the relationship of violence and aggression with alcohol is well established (Bushman and Cooper, 1990; White *et al.*, 2001). For example, a review including 130 independent studies found that alcohol was correlated with both criminal and domestic violence (Lipsey *et al.*, 1997). Despite this, there is so far no behavioral genetic study that had examined whether alcohol use moderates the influence of genetic and environmental factors on aggressive behavior.

However, the genetic and environmental relationship among alcohol use and aggressive behavior as well as other disruptive and problem behaviors within the disinhibitory spectrum such as antisocial behavior, ADHD, conduct disorder, impulsive and sensation seeking personality traits has been examined in several large population-based twin studies. On a phenotypic level, disruptive and problem behaviors within the disinhibitory spectrum can be united by a common higher order externalizing factor (Krueger *et al.*, 2002, 2005, 2007). This higher order externalizing factor has been found to be largely influenced by genetic factors. For example, the genetic influences on a common externalizing factor describing

conduct disorder, substance use, ADHD, and novelty seeking was found to account for more than 80% of the variation in an adolescent sample (Young *et al.*, 2000). Strong heritable influences on an externalizing factor of antisocial behavior, substance abuse, and conduct disorder has also been found among adults (Kendler *et al.*, 2003). Together these studies provide important insight into our understanding of externalizing behaviors. It seems that behaviors and disorders within the externalizing spectrum, including aggressive behavior, share a common genetic liability.

III. Specific Genes for Aggressive Behavior: Findings from Molecular Genetic Studies

Increasing evidence suggests the importance of heritable factors in the development of aggressive behavior (Burt, 2009; Miles and Carey, 1997; Rhee and Waldman, 2002). The first study that showed a link between a specific genotype and aggressive behavior examined the genetic material of members of a large Dutch family. This specific family had for decades been found to be prone to violent, aggressive, and impulsive behavior, including fighting, arson, attempted rape, and exhibitionism. Some of the male family members were also intellectually disabled. The aggressive males in this large family were shown to share a mutation in the gene that codes for the enzyme MAO (monoamine oxidase A). MAO breaks down brain chemicals (neurotransmitters) such as serotonin, nor-adrenaline, and dopamine, which transmit messages from one nerve cell to the next. In the afflicted males, however, a mistake in the coding sequence governing proper production of MAO was detected. As a result, abnormally large quantities of these neurotransmitters were found in the blood of the affected males (Brunner *et al.*, 1993). Although this genetic defect remains the first such link to aggressive behavior in humans, exactly how the genetic defect causes aggressive, impulsive behavior, or mental retardation is not known.

Apart from MAO, only a few candidate genes have been linked to aggressive behavior to date. The candidate genes that have been found to be associated with aggressive behavior in humans have, in many cases, been replicated in animal studies. The majority of these candidate genes are genes of the dopamine, serotonin, and norepinephrine neurotransmitter systems. The dopa-mine system is involved in mood, motivation and reward, arousal, as well as other behaviors. The serotonin system is involved in impulse control, affect regulation, sleep, and appetite, whereas the epinephrine and norepinephrine system facilitate fight-or-flight reactions and autonomic nervous system activity (Niv and Baker, 2010). For example, dopaminergic candidates, including dopamine receptor *DRD4*, has been found to be involved in ADHD and externalizing behavior, and *DRD2* has been found to be involved in substance abuse and disinhibition (Niv and Baker, 2010). The *DRD3* polymorphism has been found to be associated with impulsivity. This association was significant in violent, but not in nonviolent individuals, and there were no association between *DRD3* and violence per se (Retz *et al.*, 2003). Dopamine transporter gene *DAT1* has also been linked to ADHD (Waldman *et al.*, 1998), as well as with violent behavior and delinquency in adolescents and young adults (Guo *et al.*, 2008). Cateocholamine-O-methyl-transferase (*COMT*) has been examined primarily in children and adults with ADHD, and mixed evidence emerged for its association with conduct disorder and aggression (Caspi *et al.*, 2008). Several studies have provided evidence that the low activity *VNTR* alleles of *5HTTLPR* show associations with aggression, violence, aggressive symptoms of conduct disorder, and other forms of externalizing behavior (Haberstick *et al.*, 2006b; Linnoila *et al.*, 1983). Aggressive behavior has also shown associations with *SNPs* of epinephrine and norepinephrine. A recent study linked two *SNPs* of *PNMT* to cognitive and aggressive impulsivity in children and adolescents (Oades *et al.*, 2008).

A. G × E interaction involving specific genes for aggressive behavior

Advances in the field of molecular genetics have also made it possible for researchers to identify G × E interactions much more specifically. One of the most influential studies examining G × E in antisocial and aggressive behavior is Caspi *et al.*, (2002), a famous study from 2002. The relationship between a functional polymorphism in the *MAO-A* gene encoding the neurotransmitter-metabolizing enzyme and early childhood maltreatment was examined in the development of antisocial behavior in males. A significant G × E interaction was detected, in that maltreated boys with a genotype conferring low levels of *MAO-A* were found to be more likely to later develop antisocial problems, including conduct disorder, adult violent crime, and antisocial personality disorder, than maltreated boys who had a genotype conferring high levels of *MAO-A* (Caspi *et al.*, 2002). So far, there have only been a few replications of this important finding (Foley *et al.*, 2004; Kim-Cohen *et al.*, 2006; Nilsson *et al.*, 2006). For example, Kim-Cohen *et al.* (2006) found that the *MAO-A* polymorphism moderated the development of psychopathology after experiencing physical abuse in a sample of 975 seven-year-old boys. This finding was extended to the maltreatment exposure closer in time as the subjects were 7-years-old compared with previous work by Caspi *et al.*, (2002) in which the subjects were 26-years-old, and therefore the possibility of a spurious finding by accounting for passive and evocative G × E correlation could be ruled out. Passive G × E correlation, as discussed earlier, refers to the association between the genotype a child inherits from his/her parents and the environment in which the child is raised, and evocative G × E correlation occurs when an individual's (heritable) behavior evokes an environmental response. Further, the authors also conducted a meta-analysis including the following five studies: Caspi *et al.*, (2002), Foley *et al.* (2004), Haberstick *et al.* (2005), Kim-Cohen *et al.* (2006), and Nilsson *et al.* (2006). The association between maltreatment and mental health problems was significantly stronger in the group of males with a genotype conferring low versus high *MAO-A* activity. This provides strong evidence that the *MAO-A* gene influences vulnerability to environmental stress and that this biological process can be initiated early in life. However, there is at least one published failure to replicate (Haberstick *et al.*, 2005), and this finding has been replicated neither in females (Sjöberg *et al.*, 2007) nor in African Americans (Widom and Brzustowicz, 2006).

A G × E interaction between the *DRD2* A1 allele and risk-level in family environments has been suggested in a sample of adolescents with criminal offenses, the National Longitudinal Study of Adolescent Health (Ad-Health). Polymorphisms in genes related to the neurotransmitter dopamine were associated with age of first police contact and arrests, but only for youth from low-risk family environments. More specifically, among those adolescents with a history of criminal offending, those at greatest risk for later onset were those with the A1 allelic form of the *DRD2* gene, in combination with favorable home environments as defined by maternal attachment, involvement, and engagement (DeLisi *et al.*, 2008). It is important to emphasize that this finding involves the age of onset of first police contact and not the overall risk for offending versus not offending.

There is also some evidence for a G × E interaction in the *5HTTLPR* genotype with adult violence, whereby home violence, familial economic difficulties, and educational or home-life disruptions during childhood were found to predict violent behavior later in life only in individuals with the short promoter alleles present (Reif *et al.*, 2007). A similar G × E interaction between the short allele of *5HTTLPR* and childhood adversity has also been reported for ADHD (Retz *et al.*, 2008).

The ability to detect G × E interactions in molecular genetic studies is both exciting and controversial. The identification of specific genetic markers and specific experiences provides the opportunity to evaluate genetic and environmental risk factors at the individual level. This significantly increases opportunities for developing effective treatments and

preventions for antisocial and aggressive behavior as well as other forms of psychopathology, which is exciting. At the same time, increased understanding of individual risks has often been considered cautiously because of the potential for bias and discrimination of those individuals who are identified as being at highest risk for being afflicted with disorders.

IV. Conclusions

Studies (and meta-analyses) including both twin and adoption samples show that about half (50%) of the variance in aggressive behavior is explained by genetic influences in both males and females, with the remaining 50% of the variance being explained by nonshared environmental factors. Form of aggression (reactive, proactive, direct/physical, indirect/relational), method of assessment (observation, self, teacher, parent/caregiver), and age of the subjects—all seem to be significant moderators of the magnitude of genetic and environmental influences on aggressive behavior. Neither study design (twin vs. sibling adoption design) nor sex, on the other hand, seems to impact the nature or magnitude of these genetic and environmental influences on aggression.

Although we are unaware of any twin or adoption studies of aggression induced in authoritative situations such as in the Milgram or Stanford Prison studies, the vast evidence for genetic influences in most forms of aggression that have been studied could suggest that individual differences in those early studies might have stemmed in part from different genetic propensities in their subjects. Findings from G × E studies on aggressive behavior suggest that not all individuals will be affected to the same degree by these environmental exposures, and also that not all individuals will be affected to the same degree by the genetic predispositions. Adoption and twin studies rely on relationships between family members when examining G × E interaction effects, whereas molecular genetic studies are using both a measured environmental (risk) factor and a measured genetic factor. To date, there have only been a few twin/adoption and molecular studies that report finding G × E in aggressive behavior, either using the mean levels approach or the moderated effects approach. These studies have shown that various measures of family adversity and social disadvantage interact (or act as moderators) with genetic factors on aggressive behavior.

Today, we have the potential to identify genetic risks at the level of specific genes, and identify aspects of the environment that make some individuals more vulnerable than others. Yet, there will always be groups of individuals with the same combination of genetic risk and environmental vulnerability who will not engage in aggressive behavior. So, it is still only an increased (probabilistic) risk and not a biological determinism. In spite of such strong support for a genetic basis to aggressive behavior, the importance of potential interventions which are environmentally based must not be ignored. Environmental interventions could be developed, for example, through family or school-based programs, to reduce aggressive behavior. In fact, a general view held by behavioral genetics researchers is that the best way to understand environment—and hence develop effect treatment interventions—is through genetically informative designs such as twin and family data. By using twin and family data, it is not only possible to estimate the influence of heritable factors on a trait or a phenotype, but also the influence of environmental factors. Modern methods for identifying and understanding G × E interactions will provide a means for doing exactly this.

References

Achenbach, TM. Manual for Teacher's Report form and 1991 Profile. University of Vermont, Department of Psychiatry; Burlington, VT: 1991a.

Achenbach, TM. Manual for the Child Behavior Checklist/4-18 and 1991 Profile. University of Vermont, Department of Psychiatry; Burlington, VT: 1991b.

Achenbach, TM. Manual for the Youth Self-Report and 1991 Profile. University of Vermont, Department of Psychiatry; Burlington, VT: 1991c.

Anderson CA, Shibuya A, Ihori N, Swing EL, Bushman BJ, Sakamoto A, Rothstein HR, Saleem M. Violent video game effects on aggression, empathy, and prosocial behavior in eastern and western countries: A meta-analytic review. Psychol Bull. 2010; 136(2):151–173. [PubMed: 20192553]

Baker LA, Barton M, Lozano DI, Raine A, Fowler JH. The southern California twin register at the university of southern California: II. Twin Res Hum Genet. 2006; 9:933–940. [PubMed: 17254433]

Baker LA, Jacobson KC, Raine A, Lozano DI, Bezdjian S. Genetic and environmental bases of childhood antisocial behavior: A multi-informant twin study. J Abnorm Psychol. 2007; 116(2):219–235. [PubMed: 17516756]

Baker LA, Raine A, Liu J, Jacobson KC. Differential genetic and environmental influences on reactive and proactive aggression in children. J Abnorm Child Psychol. 2008; 36:1265–1278. [PubMed: 18615267]

Bandura, A. Aggression: A Social Learning Theory Analysis. Prentice-Hall; New York: 1973.

Bartels M, Rietveld MJ, van Baal CM, Boomsma D. Genetic and Environmental Influences on the Development of Intelligence. Behav Genet. 2002; 32(4):237–249. [PubMed: 12211623]

Bartels M, Hudziak JJ, van den Oord EJCG, van Beijsterveldt CEM, Rietveld MJH, Boomsma D. Co-occurrence of aggressive behavior and rule-breaking behavior at age 12: Multi-rater analyses. Behav Genet. 2003; 33:607–621. [PubMed: 14574136]

Brunner H, Nelen M, Breakefield XO, Ropers HH, van Oost BA. Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. Science. 1993; 262(5133):578–580. [PubMed: 8211186]

Burt AS. Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. Clin Psychol Rev. 2009; 29:163–178. [PubMed: 19193479]

Bushman BJ, Cooper HM. Effects of alcohol on human aggression: An integrative research interview. Psychol Bull. 1990; 107(3):341–354. [PubMed: 2140902]

Buss AH, Durkee A. An inventory for assessing different kinds of hostility. J Consult Clin Psychol. 1957; 21:343–349.

Buss AH, Perry M. The aggression questionnaire. J Pers Soc Psychol. 1992; 63:452–459. [PubMed: 1403624]

Button TMM, Scourfield J, Martin N, McGuffin P. Do aggressive and non-aggressive antisocial behaviors in adolescents result from the same genetic and environmental effects? Am J Med Genet B Neuropsychiatr Genet. 2004; 129 B:59–63. [PubMed: 15274042]

Button TMM, Scourfield J, Martin N, Purcell S, McGuffin P. Family dysfunction interacts with genes in the causation of antisocial symptoms. Behav Genet. 2005; 35:115–120. [PubMed: 15685425]

Caspi A, McClay J, Moffitt TE, Mill J, Martin J, Craig IW, Taylor A, Poulton R. Role of genotype in the cycle of violence in maltreated children. Science. 2002; 297(5582):851–854. [PubMed: 12161658]

Caspi A, Langley K, Milne B, Moffitt TE, O'Donovan M, Owen MJ, Polo Tomas M, Poulton R, Rutter M, Taylor A, Williams B, Thapar A. A replicated molecular genetic basis for subtyping antisocial behavior in children with attention-deficit/hyperactivity disorder. Arch Gen Psychiatry. 2008; 65(2):203–210. [PubMed: 18250258]

Cates DS, Houston BK, Vavak CR, Crawford MH, Uttley M. Heritability of hostility-related emotions, attitudes, and behaviors. J Behav Med. 1993; 16(3):237–256. [PubMed: 8350340]

Cho H, Guo G, Iritani BJ, Hallfors DD. Genetic contribution to suicidal behaviors and associated risk factors among adolescents in the US. Prev Sci. 2006; 7(3):303–311. [PubMed: 16775759]

Christensen K, Petersen I, Skytthe A, Herskind AM, McGue M, Bingley P. Comparison of academic performance of twins and singletons in adolescence: Follow-up study. Br Med J. 2006; 6:1–5.

Cloninger, CR.; Gottesman, II. Genetic and environmental factors in antisocial behavior disorders. In: Mednick, SA.; Moffitt, TE.; Stack, SA., editors. The Causes of Crime: New Biological Approaches. Cambridge University Press; Cambridge: 1987. p. 92-109.

Cloninger CR, Sigvardsson S, Bohman M, von Knorring AL. Predisposition to petty criminality in Swedish adoptees. II. Cross-fostering analysis of gene-environment interaction. Arch Gen Psychiatry. 1982; 39:1242–1247. [PubMed: 7138224]

Cloninger CR, Bohman M, Sigvardsson S, von Knorring AL. Psychopathology in adopted-out children of alcoholics. The Stockholm Adoption Study. Recent Dev Alcohol. 1985; 3:37–51. [PubMed: 3975456]

Coccaro EF, Bergeman CS, Kavoussi RJ, Seroczynski AD. Heritability of aggression and irritability: A twin study of the Buss-Durkee aggression scales in adult male subjects. Biol Psychiatry. 1997; 41(3):273–284. [PubMed: 9024950]

Conners CK. Symptom patterns in hyperkinetic, neurotic, and normal children. Child Dev. 1970; 41:667–682.

Crick NR, Grotpeter JK. Children's treatment by peers: Victims of relational and overt aggression. Dev Psychopathol. 1996; 8:267–280.

Cronk N, Slutske WS, Madden PA, Bucholz KK, Reich W, Heath A. Emotional and behavioral problems among female twins: An evaluation of the equal environments assumption. J Am Acad Child Adolesc Psychiatry. 2002; 41:829–837. [PubMed: 12108808]

Czajkowski N, Kendler KS, Jacobson K, Tambs K, Røysamb E, Reichborn-Kjennerud T. Passive-aggressive (negativistic) personality disorder: A population-based twin study. J Pers Disord. 2008; 22(1):109–122. [PubMed: 18312126]

Deater-Deckard K, Plomin R. An adoption study of the etiology of teacher and parent reports of externalizing behavior problems in middle childhood. Child Dev. 1999; 70:144–154. [PubMed: 10191519]

DeFries JC, Fulker DF. Multiple regression analysis of twin data. Behav Genet. 1985; 15:467–473. [PubMed: 4074272]

DeLisi M, Beaver KM, Wright JP, Vaughn MG. The etiology of criminal onset: The enduring salience of nature and nurture. J Crim Justice. 2008; 36:217–223.

Derks EM, Dolan CV, Boomsma DI. A test of the equal environment assumption (EEA) in multivariate twin studies. Twin Res. 2006; 9(3):403–411.

Dionne G, Tremblay R, Boivin M, Laplante D, Perusse D. Physical aggression and expressive vocabulary in 19-month-old twins. Dev Psychol. 2003; 39(2):261–273. [PubMed: 12661885]

Dodge, K. The structure and function of reactive and proactive aggression. In: Pepler, DJ.; Rubin, KH., editors. The Development and Treatment of Childhood Aggression. Erlbaum; Hillsdale, NJ: 1991. p. 201-218.

Dodge KA, Coie JD. Social-information processing factors in reactive and proactive aggression in children's peer groups. J Pers Soc Psychol. 1987; 53:1146–1158. [PubMed: 3694454]

Dodge KA, Lochman JE, Harnish JD, Bates JE, Pettit GS. Reactive and proactive aggression in school children and psychiatrically impaired chronically assaultive youth. J Abnorm Psychol. 1997; 106:37–51. [PubMed: 9103716]

Eaves LJ. The resolution of genotype x environment interaction in segregation analysis of nuclear families. Genet Epidemiol. 1984; 1:215–228. [PubMed: 6544238]

Edelbrock C, Rende R, Plomin R, Thompson LA. A twin study of competence and problem behavior in childhood and early adolescence. J Child Psychol Psychiatry. 1995; 36(5):775–785. [PubMed: 7559844]

Eley TC, Lichtenstein P, Stevenson J. Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: Results from two twin studies. Child Dev. 1999; 70(1):155–168. [PubMed: 10191520]

Eriksson M, Rasmussen F, Tynelius P. Genetic factors in physical activity and the equal environment assumption—The Swedish young male twin study. Behav Genet. 2006; 36(2):238–247. [PubMed: 16502139]

Ferguson CJ, Kilburn J. The public health risks of media violence: A meta-analytic review. J Pediatr. 2009; 154(5):759–763. [PubMed: 19230901]

Ferguson CJ, Kilburn J. Much ado about nothing: The misestimation and overinter-pretation of violent video game effects in eastern and western nations: Comment on Anderson *et al.* (2010). Psychol Bull. 2010; 136(2):174–178. discussion 82-87. [PubMed: 20192554]

Finkel D, McGue M. Sex differences and nonadditivity in heritability of the Multidimensional Personality Questionnaire Scales. J Pers Soc Psychol. 1997; 72(4):929–938. [PubMed: 9108705]

Foley DL, Eaves L, Wormley B, Silberg J, Maes H, Kuhn J, Riley B. Childhood adversity, monoamine oxidase a genotype, and risk for conduct disorder. Arch Gen Psychiatry. 2004; 61:738–744. [PubMed: 15237086]

Ge X, Conger RD, Cadoret RJ, Neiderhiser JM, Yates WR, Troughton E, Stewart MA. The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. Dev Psychol. 1996; 32(4):574–589.

Gelhorn HL, Stallings MC, Young SE, Corely RP, Rhee SH, Hopfer C, Hewitt JK. Common and specific genetic influences on aggressive and nonaggressive conduct disorder domains. J Am Acad Child Adolesc Psychiatry. 2006; 45(5):570–577. [PubMed: 16670651]

Ghodesian-Carpey J, Baker LA. Genetic and environmental influences on aggression in 4- to 7-year-old twins. Aggress Behav. 1987; 13:173–186.

Gjone H, Novik TS. Parental ratings of behaviour problems: A twin study and general population comparison. J Child Psychol Psychiatry. 1995; 36(7):1213–1224. [PubMed: 8847381]

Gough HG. The adjective check list as a personality assessment research technique. Psychol Rep. 1960; 6:107–122.

Guo G, Roettger ME, Shih JC. Contributions of the DAT1 and DRD2 genes to serious and violent delinquency among adolescents and young adults. Hum Genet. 2007; 121:125–136. [PubMed: 17120049]

Haberstick BC, Lessem JM, Hopfer CJ, Smolen A, Ehringer MA, Timberlake D, Hewitt JK. Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. Am J Med Genet B Neuropsychiatr Genet. 2005; 135B(1):59–64. [PubMed: 15806601]

Haberstick BC, Schmitz S, Young SE, Hewitt JK. Genes and developmental stability of aggressive behavior problems at home and school in a community sample of twins aged 7–12. Behav Genet. 2006a; 36:809–819. [PubMed: 16816994]

Haberstick BC, Smolen A, Hewitt JK. Family-based association test of the 5HTTLPR and aggressive behavior in a general population sample of children. Biol Psychiatry. 2006b; 59:836–843. [PubMed: 16412987]

Haney C, Banks WC, Zimbardo PG. A study of prisoners and guards in a simulated prison. Naval Res Rev. 1973; 30:4–17.

Hettema JM, Neale MC, Kendler KS. Physical similarity and the equal-environment assumption in twin studies of psychiatric disorders. Behav Genet. 1995; 25(4):327–335. [PubMed: 7575361]

Hodgins S, Kratzer L, McNeil TF. Obstetric complications, parenting, and risk of criminal behavior. Arch Gen Psychiatry. 2001; 58:746–752. [PubMed: 11483140]

Hudziak JJ, Rudiger LP, Neale MC, Heath AC, Todd RD. A twin study of inattentive, aggressive, and anxious/depressed behaviors. J Am Acad Child Adolesc Psychiatry. 2000; 39(4):469–476. [PubMed: 10761349]

Hudziak JJ, van Beijsterveldt CEM, Bartels M, Rietveld MJH, Rettew DC, Derks EM, Boomsma D. Individual differences in aggression: Genetic analyses by age, gender, and informant in 3-, 7-, and 10-year-old Dutch Twins. Behav Genet. 2003; 33(5):575–589. [PubMed: 14574134]

Huesmann LR. Nailing the coffin shut on doubts that violent video games stimulate aggression: Comment on Anderson *et al.* (2010). Psychol Bull. 2010; 136(2):179–181. [PubMed: 20192555]

Iacono WG, McGue M, Krueger RF. Minnesota center for twin and family research. Twin Res Hum Genet. 2006; 9(6):978–984. [PubMed: 17254440]

Jacobson KC, Prescott CA, Kendler K. Sex differences in the genetic and environmental influences on the development of antisocial behavior. Dev Psychol. 2002; 14:395–416.

Jaffee SR, Caspi A, Moffitt TE, Dodge KA, Rutter M, Taylor A, Tully LA. Nature × nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. Dev Psychopathol. 2005; 17(1):67–84. [PubMed: 15971760]

Kendler K, Neale MC, Kessler RC, Heath AC, Eaves LJ. A test of the equal-environment assumption in twin studies of psychiatric illness. Behav Genet. 1993; 23(1):21–27. [PubMed: 8476388]

Kendler K, Prescott CA, Myers J, Neale MC. The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. Arch Gen Psychiatry. 2003; 60:929–937. [PubMed: 12963675]

Keyes MA, Malone SM, Elkins IJ, Legrand LN, McGue M, Iacono WG. The enrichment study of the Minnesota twin family study: Increasing the yield of twin families at high risk for externalizing psychopathology. Twin Res Hum Genet. 2009; 12(5):489–501. [PubMed: 19803776]

Kim-Cohen J, Caspi A, Taylor A, Williams B, Newcombe R, Craig IW, Moffitt TE. MAOA, maltreatment, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. Mol Psychiatry. 2006; 11(10):903–913. [PubMed: 16801953]

Klump KL, Holly A, Iacono WG, McGue M, Willson LE. Physical similarity and twin resemblance for eating attitudes and behaviors: A test of the equal environments assumption. Behav Genet. 2000; 30(1):51–58. [PubMed: 10934799]

Knafo A, Plomin R. Prosocial behavior from early to middle childhood: Genetic and environmental influences on stability and change. Dev Psychol. 2006; 42(5):771–786. [PubMed: 16953685]

Krueger RF, Moffitt TE, Caspi A, Bleske A, Silva PA. Assortative mating for antisocial behavior: Developmental and methodological implications. Behav Genet. 1998; 28(3):173–186. [PubMed: 9670593]

Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. J Abnorm Psychol. 2002; 111:411–424. [PubMed: 12150417]

Krueger RF, Markon KE, Patrick CJ, Iacono WG. Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM–V. J Abnorm Psychol. 2005; 114(4):537–550. [PubMed: 16351376]

Krueger RF, Markon KE, Patrick CJ, Benning SD, Kramer MD. Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. J Abnorm Psychol. 2007; 116(4):645–666. [PubMed: 18020714]

Lahey BB, Applegate B, Waldman ID, Loft JD, Hankin BL, Rick J. The structure of child and adolescent psychopathology: Generating new hypotheses. J Abnorm Psychol. 2004; 113:358–385. [PubMed: 15311983]

Leventhal T, Brooks-Gunn J. The neighbourhoods they live in: The effects of neighbourhood residence on child and adolescent outcomes. Psychol Bull. 2000; 126:309–337. [PubMed: 10748645]

Lewis DO, Yeager CA, Gidlow B, Lewis M. Six adoptees who murdered: Neuropsychiatric vulnerabilities and characteristics of biological and adoptive parents. J Am Acad Psychiatry Law. 2001; 29(4):390–397. [PubMed: 11785610]

Linnoila M, Virkkunen M, Scheinin M, Nuutila A, Rimon R, Goodwin FK. Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. Life Sci. 1983; 33:2609–2614. [PubMed: 6198573]

Lipsey MW, Wilson DB, Cohen MA, Derzon JH. Is there a causal relationship between alcohol use and violence? A synthesis of evidence. Recent Dev Alcohol. 1997; 13:245–282. [PubMed: 9122498]

Loehlin, JC. Genes and environment in personality development. Newbury Park: SAGE CA; 1992.

Loehlin JC. Is there an active gene-environment correlation in adolescent drinking behavior? Behav Genet. 2010; 40(4):447–451. [PubMed: 20213348]

Maes HHM, Neale MC, Kendler K, Hewitt JK, Silberg JL, Foley DL, Meyer JM, Rutter M, Simonoff E, Pickles A, Eaves LJ. Assortative mating for major psychiatric diagnosis in two population-based samples. Psychol Med. 1998; 28:1389–1401. [PubMed: 9854280]

Maes HH, Silberg JL, Neale MC, Eaves LJ. Genetic and cultural transmission of antisocial behavior: An extended twin parent model. Twin Res Hum Genet. 2007; 10(1):136–150. [PubMed: 17539373]

Mather, K.; Jinks, JL. Biometrical Genetics: The Study of Continuous Variation. Chapman & Hall; London: 1982.

Mathew KA, Angulo J. Measurement of the type A behavior pattern in children: Assessment of children's competitiveness, impatience-anger, and aggression. Child Dev. 1980; 51:466–475. [PubMed: 7398452]

Mauger, PA. Interpersonal Behavior Survey (IBS) Manual. Western Psychological Services; Los Angeles: 1980.

McGue M, Bacon S, Lykken DT. Personality stability and change in early adulthood: A behavioral genetic analyses. Dev Psychol. 1993; 29:96–109.

Meininger JC, Hayman LL, Coates PM, Gallagher P. Genetics or environment? Type A behavior and cardiovascular risk factors in twin children. Nurs Res. 1988; 37(6):341–346. [PubMed: 3186476]

Merton, RK. Social Theory and Social Structure. The Free Press; New York: 1957.

Miles DR, Carey G. Genetic and environmental architecture of human aggression. J Pers Soc Psychol. 1997; 72(1):207–217. [PubMed: 9008382]

Milgram S. Behavioral study of obedience. J Abnorm Soc Psychol. 1963; 67:371–378.

Moffitt T. Teen-aged mothers in contemporary Britain & the E-risk Study Team (39 authors). J Child Psychol Psychiatry. 2002; 43:727–742. [PubMed: 12236608]

Moffitt, TE.; Caspi, A.; Rutter, M.; Silva, P. Sex Differences in Antisocial Behaviour: Conduct Disorder, Delinquency and Violence in the Dunedin Longitudinal Study. Cambridge University Press; Cambridge: 2001.

Moilanen I, Linna SL, Kumpulainen K, Tamminen K, Piha J, Almqvist F. Are twins' behavioural 7emotional problems different from singletons'? Eur Child Adolesc Psychiatry. 1999; 8:62–67. [PubMed: 10654135]

Narusyte J, Andershed AK, Neiderhiser JM, Lichtenstein P. Aggression as a mediator of genetic contributions to the association between negative parent–child relationships and adolescent antisocial behavior. Eur Child Adolesc Psychiatry. 2006; 16(2):128–137. [PubMed: 17136502]

Neale, MC.; Cardon, LR. Methodology for Genetic Studies of Twins and Families. Kluwer Academic, Publications; Dordrecht, The Netherlands: 1992.

Neiderhiser JM, Reiss D, Pedersen NL, Lichtenstein P, Spotts EL, Hansson K, Cederblad M, Elthammar O. Genetic and environmental influences on mothering of adolescents: A comparison of two samples. Dev Psychol. 2004; 40(3):335–351. [PubMed: 15122961]

Nilsson KW, Sjöberg RL, Damberg M, Leppert J, Ohrvik J, Alm PO, Lindström L, Oreland L. Role of monoamine oxidase A genotype and psychosocial factors in male adolescent criminal activity. Mol Psychiatry. 2006; 10(11):903–913.

Niv, S.; Baker, LA. Genetic marker for antisocial behavior. In: Thomas, C.; Pope, K., editors. The Origins of Antisocial Behavior: A Developmental Perspective. Oxford University Press; New York: 2010.

Oades RD, Lasky-Su J, Christiansen H, Faraone SV, Sonuga-Barke EJS, Banaschewski T, Chem W, Anney RJL, Buitelaar JK, Ebstein RP, Franke B, Gill M, et al. The influence of serotonin and other genes and impulsive behavioral aggression and cognitive impulsivity in children with attention-deficit/hyperactivity disorder (ADHD): Finding from a family-based association test (FBAT) analysis. Behav Brain Funct. 2008; 48:4–48.

O'Connor M, Foch T, Sherry T, Plomin R. A twin study of specific behavioral problems of socialization as viewed by parents. J Abnorm Child Psychol. 1980; 8(2):189–199. [PubMed: 7190581]

O'Connor TG, Deater-Deckard K, Fulker D, Rutter M, Plomin R. Genotype-environment correlations in late childhood and early adolescence: Antisocial behavioral problems and coercive parenting. Dev Psychol. 1998; 34:970–981. [PubMed: 9779743]

Olweus, D. Prevalence and incidence in the study of antisocial behavior: Definitions and measurements. In: Klein, MW., editor. Cross-National Research in Self-Reported Crime and Delinquency. Kluwer Academic Publishers; Dordrecht: 1989.

Owen DR, Sines JO. Heritability of personality in children. Behav Genet. 1970; 1(3):235–248. [PubMed: 5535858]

Parker, T. Television Viewing and Aggression in Four and Seven Year Old Children. Boulder; Boulder, CO: 1989.

Pfohl, B.; Blum, N.; Zimmerman, M. Structured Interview for DSM-IV Personality: SIDP-IV. American Psychiatric Press; Washington, DC: 1997.

Plomin, R.; DeFries, JC.; McClearn, GE.; McGuffin, P. Behavioral Genetics. Worth Publisher; United States of America: 2001.

Polderman TJ, Posthuma D, De Sonneville LM, Verhulst FC, Boomsma DI. Genetic analyses of teacher ratings of problem behavior in 5-year-old twins. Twin Res Hum Genet. 2006; 9(1):122–130. [PubMed: 16611477]

Posthuma D, Beem AL, de Geus EJC, van Baal CM, von Hjelmborg JB, Iachine I, Boomsma DI. Theory and practice in quantitative genetics. Twin Res Hum Genet. 2003; 6(5):361–376.

Pulikkined L, Kaprio J, Rose RJ. Peers, teachers, and parents as assessors of the behavioural and emotional problems of twins and their adjustment: The Multidimensional Peer Nomination Inventory. Twin Res Hum Genet. 1999; 2:274–285.

Rahe RH, Hervig L, Rosenman RH. Heritability of type A behavior. Psychosom Med. 1978; 40(6): 478–486. [PubMed: 569878]

Raine A. Biosocial studies of antisocial and violent behavior in children and adults: A review. J Abnorm Child Psychol. 2002; 30(4):311–326. [PubMed: 12108763]

Raine A, Brennan PA, Mednick SA. Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: Specificity to serious, early-onset violence. Am J Psychiatry. 1997; 154(9):1265–1271. [PubMed: 9286187]

Raine A, Dodge K, Loeber R, Gatzke-Kopp L, Lynam DR, Reynolds C, Stouthamer-Loeber M, Liu J. The reactive-proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. Aggress Behav. 2006; 32:159–171. [PubMed: 20798781]

Reif A, Rosler M, Freitag CM, Schneider M, Eujen A, Kissling C, Wensler D, Jacob CP, Retz-Junginger P, Thome J, Lesch KP, Retz W. Nature and nurture predispose to violent behavior: Serotonergic genes and adverse childhood environment. Neuropsychopahrmacology. 2007; 32:2375–2383.

Retz W, Rosler M, Supprian T, Retz-Junginger P, Thome J. Dopamine D3 receptor gene polymorphism and violent behavior: Relation to impulsiveness and ADHD-related psychopathology. J Neural Transm. 2003; 110:561–572. [PubMed: 12721816]

Retz W, Freitag CM, Retz-Junginger P, Wenzler D, Schneider M, Kissling C, et al. A functional serotonin transporter promoter gene polymorphism increases ADHD symptoms in delinquents: Interaction with adverse childhood environment. Psychiatry Res. 2008; 158:123–131. [PubMed: 18155777]

Rhee SH, Waldman ID. Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. Psychol Bull. 2002; 128:490–529. [PubMed: 12002699]

Rowe, DC. Assessing genotype-environment interactions and correlations in the postgenomic era. In: Plomin, R.; DeFries, JC.; Craig, IW.; McGuffin, P., editors. Behavioral Genetics in the Postgenomic Era. American Psychological Association; Washington, DC: 2003. p. 71-99.

Rowe DC, Almeida DM, Jacobson KC. School context and genetic influences on aggression in adolescence. Psychol Sci. 1999; 10(3):277–280.

Rushton JP, Fulker DW, Neale MC, Nias DK, Eysenck HJ. Altruism and aggression: The heritability of individual differences. J Pers Soc Psychol. 1986; 50(6):1192–1198. [PubMed: 3723334]

Rutter, M. Nature-nurture interplay explained. Blackwell Publishing Ltd; Oxford, UK: 2006. Genes and behavior.

Rutter M, Redshaw J. Annotation: Growing up as a twin: Twin-singleton differences in psychological development. J Child Psychol Psychiatry. 1991; 32(6):885–895. [PubMed: 1744192]

Rutter, M.; Giller, H.; Hagell, A. Antisocial Behavior by Young People. Cambridge University Press; Cambridge, UK: 1998.

Rutter M, Caspi A, Moffitt TE. Using sex differences in psychopathology to study causal mechanisms: Unifying issues and research strategies. J Child Psychol Psychiatry. 2003; 44(8):1092–1115. [PubMed: 14626453]

Scarr S. Genetic factors in activity motivation. Child Dev. 1966; 37(3):663–673. [PubMed: 6008135]

Scarr S, McCartney K. How people make their own environments: A theory of genotype-environment effects. Child Dev. 1983; 54:425–435.

Schmitz S, Fulker DW, Mrazek DA. Problem behavior in early and middle childhood: An initial behavior genetic analysis. J Child Psychol Psychiatry. 1995; 36(8):1443–1458. [PubMed: 8988277]

Schwartz D, Dodge K, Coie JD, Hubbard JA, Cillessen AHN, Lemerise EA, Bateman H. Social-cognitive and behavioral correlates of aggression and victimization in boys' play groups. J Abnorm Child Psychol. 1998; 26(6):431–440. [PubMed: 9915650]

Shaffer, D.; Fisher, P.; Lucas, C.; Comer, J. Diagnostic Interview Schedule for Children-DISC-IV Scoring Manual. Columbia University, NY: 2000.

Silva, PA.; Stanton, WR. From child to adult: The Dunedin Multidisciplinary Health and Development Study. Oxford University Press Inc.; New York, NY: 1996.

Simonoff E, Pickles A, Meyer J, Silberg J, Maes H, Loeber M, Rutter M, Hewitt JK, Eaves L. The Virginia twin study of adolescent behavioral development. Arch Gen Psychiatry. 1997; 54:801–808. [PubMed: 9294370]

Simonoff E, Pickles A, Meyer J, Silberg J, Maes H. Genetic and environmental influences on subtypes of conduct disorder behavior in boys. J Abnorm Child Psychol. 1998; 26(6):495–509. [PubMed: 9915655]

Sines, JO.; Pauker, JD.; Sines, LK. The Development of an Objective, Non-verbal Personality Test for Children. Midwestern Psychological Association; Chicago: 1966.

Sjöberg RL, Nilsson KW, Wargelius HL, Leppert J, Lindström L, Oreland L. Adolescent girls and criminal activity: Role of MAOA-LPR genotype and psychosocial factors. Am J Med Genet B Neuropsychiatr Genet. 2007; 144B:159–164. [PubMed: 17034017]

Tackett JL, Waldman ID, Lahey BB. Etiology and measurement of relational aggression: A multi-informant behavior genetic investigation. J Abnorm Psychol. 2009; 118(4):722–733. [PubMed: 19899842]

Taylor A. The consequences of selective participation on behavioral-genetic findings: evidence from simulated and real data. Twin Res. 2004; 7(5):485–504. [PubMed: 15527665]

Taylor J, McGue M, Iacono WG. Sex differences, assortative mating, and cultural transmission effects on adolescent delinquency: A twin family study. J Child Psychol Psychiatry. 2000; 41(4):433–440. [PubMed: 10836673]

Tellegen, A. Manual for the Multidimensional Personality Questionnaire. University of Minnesota Press; Minneapolis: 2011.

Tuvblad C, Grann M, Lichtenstein P. Heritability for adolescent antisocial behavior differs with socioeconomic status: Gene-environment interaction. J Child Psychol Psychiatry. 2006; 47(7):734–743. [PubMed: 16790008]

Tuvblad C, Raine A, Zheng M, Baker LA. Genetic and environmental stability differs in reactive and proactive aggression. Aggress Behav. 2009; 35(6):437–452. [PubMed: 19688841]

Tuvblad C, Narusyte J, Grann M, Sarnecki J, Lichtenstein P. The genetic and environmental etiology of antisocial behavior, from early adolescence to emerging adulthood. Behav Genet. 2011; 41(5):629–640. [PubMed: 21431322]

van Beijsterveldt CEM, Bartels M, Hudziak JJ, Boomsma D. Causes of stability of aggression from early childhood to adolescence: A longitudinal genetic analysis in Dutch twins. Behav Genet. 2003; 33:591–605. [PubMed: 14574135]

van den Oord EJCG, Boomsma D, Verhulst FC. A study of problem behaviors in 10- to 15-year-old biologically related and unrelated international adoptees. Behav Genet. 1994; 24:193–205. [PubMed: 7945150]

van den Oord EJCG, Koot HM, Boomsma DI, Verhulst FC, Orlebeke JF. A twin-singleton comparison of problem behaviour in 2–3-year-olds. J Child Psychol Psychiatry. 1995; 36(3):449–458. [PubMed: 7782408]

van den Oord EJ, Verhulst FC, Boomsma DI. A genetic study of maternal and paternal ratings of problem behaviors in 3-year-old twins. J Abnorm Psychol. 1996; 105(3):349–357. [PubMed: 8772005]

van der Valk FC, Verhulst FC, Stroet TM, Boomsma D. Quantitative genetic analysis of internalizing and externalizing problems in a large sample of 3-year-old twins. Twin Res Hum Genet. 1998; 1:25–33.

Vierikko E, Pulkkinen L, Kaprio J, Rose RJ. Genetic and environmental influences on the relationship between aggression and hyperactivity-impulsivity as rated by teachers and parents. Twin Res Hum Genet. 2004; 7(3):261–274.

von der Pahlen B, Santtila P, Johansson A, Varjonen M, Jern P, Witting K, Kenneth Sandnabba N. Do the same genetic and environmental effects underlie the covariation of alcohol dependence, smoking, and aggressive behaviour? Biol Psychol. 2008; 78(3):269–277. [PubMed: 18456382]

Waldman ID, Rowe DC, Abramowitz A, Kozel ST, Mohr JH, Sherman SL, Cleveland HH, Sanders ML, Gard JMC, Stever C. Association and linkage of the dopamine transporter gene and attention-deficit hyperactivity disorder in children: Heterogeneity owing to diagnostic subtype and severity. Am J Hum Genet. 1998; 63:1767–1776. [PubMed: 9837830]

White HR, Xie M, Thompson W, Loeber R, Stouthamer-Loeber M. Psychopa-thology as a predictor of adolescent drug use trajectories. Psychol Addict Behav. 2001; 15(3):210–218. [PubMed: 11563798]

Widom CS, Brzustowicz LM. MAOA and the “cycle of violence”: Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. Biol Psychiatry. 2006; 1(1):60.

Xian H, Scherrer JF, Eisen SA, True WR, Heath AC, Goldberg J, Lyons MJ, Tsuang MT. Self-Reported zygosity and the equal-environments assumption for psychiatric disorders in the Vietnam Era Twin Registry. Behav Genet. 2000; 30(4):303–310. [PubMed: 11206085]

Young SE, Stallings MC, Corley RP, Krauter KS, Hewitt JK. Genetic and environmental influences on behavioral disinhibition. Am J Med Genet. 2000; 96:684–695. [PubMed: 11054778]

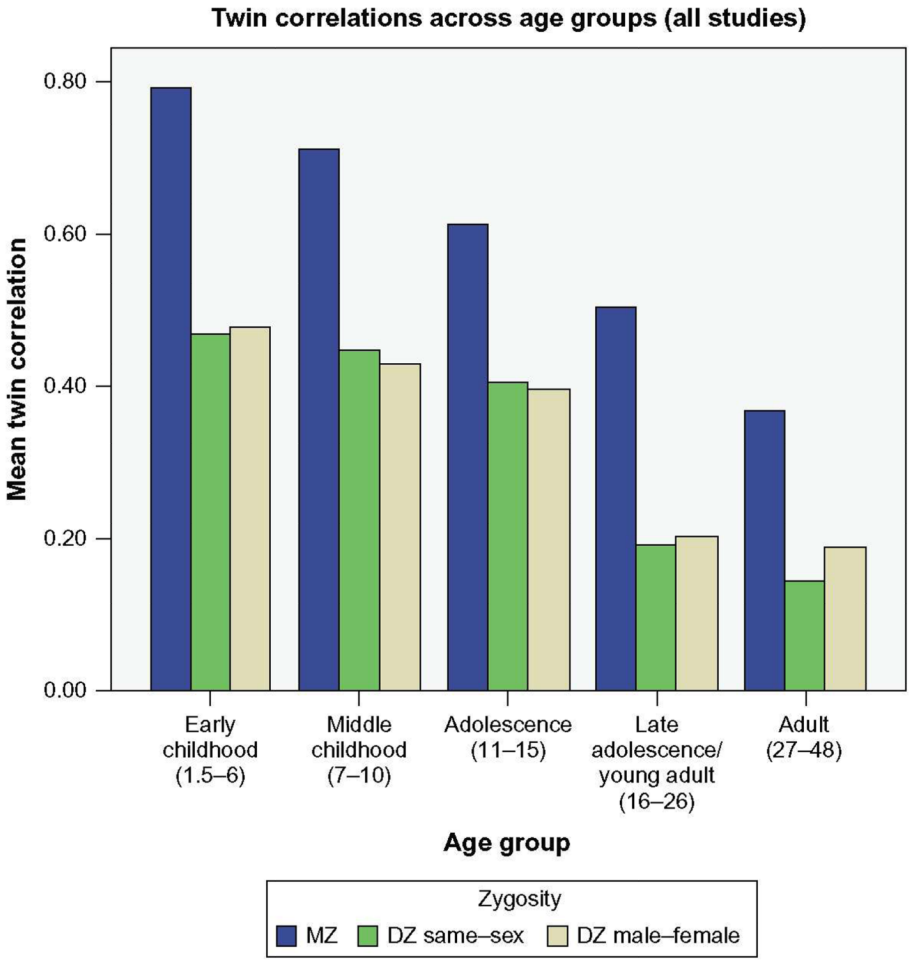

Figure 8.1.
Twin correlations across age groups (all studies).

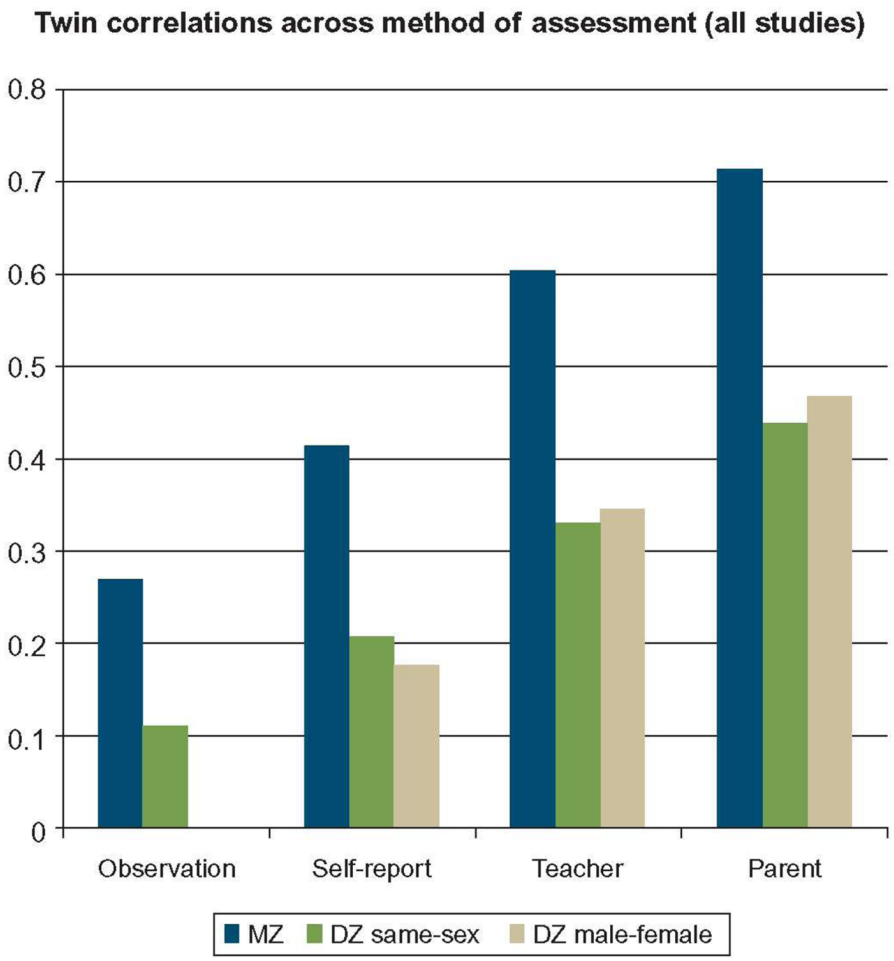

Figure 8.2.
Twin correlations across method of assessment (all studies).

Table 8.1

Effect Sizes for Aggressive Behavior from Adoption Studies

Study (author, year)	Aggression measure	Informant	Age in years	Sex	Biological siblings *r*(*N*)	Adoptive siblings *r*(*N*)	h^2	c^2
Dutch adoptees (van den Oord *et al.*, 1994)	Aggression (CBCL)	Parent ratings	10–15 Mean=12.4	M F MF	0.40 (30) 0.45 (35) 0.38 (46)	0.02 (44) 0.21 (48) 0.05 (129)	0.52 0.32	0.00 0.25
Dutch adoptees (van der Valk *et al.*, 1998)	Aggression (CBCL)	Parent ratings	10–15 13–18	M+F M+F	0.42 (111) 0.36 (152)	0.13 (221) 0.26 (156)	0.61 0.52	0.13 0.12
Colorado adoptees (Deater-Deckard and Plomin, 1999)	Aggression (CBCL)	Parent ratings	7, 9, 10, 11, 12 Mean=9.5	M+F	0.39 (94)	0.26 (78)	0.24	0.27
	Aggression (TRF)	Teacher ratings		M+F	0.25 (188)	−0.06 (156)	0.49	0.00
Unknown (Parker, 1989)[a]	Aggression (as reported in Rhee and Waldman, 2002; Burt, 2009)	Parent ratings	4–7	M+F	0.44 (66)	0.47 (45)		

M, Male; F, Female; h^2, heritability; c^2, shared environment; CBCL, Child Behavior Checklist (Achenbach, 1991b); TRF, Teacher Report Form (Achenbach, 1991a).

[a]Genetic and shared environmental estimates were not reported by the authors.

Table 8.2

Effect Sizes (Correlations) for Aggressive Behavior from Twin Studies

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
Boston twins (Scarr, 1966)	Aggression (ACL)	Parent ratings	6–10	F	0.35 (24)	−0.08 (28)	0.40[a]	–	N/A
Missouri twins (Owen and Sines, 1970)	Aggressive reaction (MCPS)	Lab observation	6–14	M F	0.09 (10) 0.58 (8)	−0.24 (11) 0.22 (13)	0.44[a]	–	Not tested
California twins (Rahe *et al.*, 1978)	Aggression (ACL)	Self-report	42–56 Mean=48	M	0.31 (93)	0.21 (97)	0.56[a]	–	N/A
Colorado twins (O'Connor *et al.*, 1980)	Aggression/bullying (PSR)	Parent ratings	Mean=7.6	M+F	0.72 (52)	0.42 (32)	–	–	Not tested
London twins, UK (Rushton *et al.*, 1986)	Aggression (IBS)	Self-report	19–60 Mean=30	M+F	0.40 (296)	0.04 (179)	0.72[a]		Not tested
California preschool twins Ghodesian-Carpey and Baker, 1987	Aggression (CBCL) Aggression (MOCL)	Parent ratings Mothers' observations	Mean=5.2 Mean=5.2	M+F M+F	0.78 (21) 0.65 (21)	0.31 (17) 0.35 (17)	0.94[a] 0.60[a]	– –	Not tested
Philadelphia twins (Meininger *et al.*, 1988)	Impatience/aggression Competitive achievement striving	Teacher rating	6–11	M+F	0.67 (71) 0.63 (71)	0.11 (34) 0.13 (34)	1.12[a] 1.00[a]	– –	Not tested
Minnesota twins (McGue *et al.*, 1993)[c]	Aggression (MPQ) Aggression (MPQ)	Self-report Self-report	Mean=19.8 Mean=29.6	M+F M+F	0.61 (79) 0.58 (79)	−0.09 (48) −0.14 (48)			Not tested Not tested
Midwest twins (Cates *et al.*, 1993)	BDHI—assault BDHI—indirect hostility	Self-report Self-report	Mean=42.5 Mean=42.5	F F	0.07 (77) 0.40 (77)	0.41 (21) 0.01 (21)	0.00 0.78	– –	N/A N/A
	BDHI—verbal hostility	Self-report	Mean=42.5	F	0.41 (77)	0.06 (21)	0.70	–	N/A
Colorado twins (Schmitz *et al.*, 1995)	Aggression (CBCL)	Parent rating	2–3 4–11	M+F	0.68 (77) 0.79 (66)	0.40 (183) 0.41 (137)	0.52 0.55	0.16 0.19	Not tested
Ohio twins, Western Reserve Twin Project (Edelbrock *et al.*, 1995)	Aggression (CBCL)	Parent rating	7–15 Mean=11.0	M+F	0.75 (99)	0.45 (82)	0.60	0.15	Not tested
Dutch twins (van den Oord *et al.*, 1996)	Aggression (CBCL)	Parent rating	3	M F MF	0.81 (210) 0.83 (265)	0.49 (236) 0.49 (238) 0.45 (409)	0.69	0.12	Not tested
Minnesota twins (Finkel and McGue, 1997)	Aggression (MPQ)	Self-report	27–64 Mean=37.8	M F MF	0.37 (220) 0.39 (406)	0.12 (165) 0.14 (352) 0.12 (114)	0.35 0.39	0.00 0.00	NS quantitative sex differences

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
VET twins (Coccaro *et al.*, 1997)	BDHI—assault BDHI—indirect hostility BDHI—indirect hostility	Self-report Self-report Self-report	Mean=44.1	M M M	0.50 (182) 0.42 (182) 0.28 (182)	0.19 (118) 0.02 (118) 0.07 (118)	0.47 0.40 0.28	0.00 0.00 0.00	N/A N/A N/A
Swedish twins (TCHAD; Eley *et al.*, 1999)	Aggression (CBCL)	Parent rating	8–9	M F MF	0.72 (176) 0.82 (160)	0.41 (182) 0.45 (194) 0.41 (310)	0.70	0.07	NS quantitative sex differences, NS qualitative sex differences
UK twins (sample obtained from Register of Child Twins; Eley *et al.*, 1999)	Aggression (CBCL)	Parent rating	12	M F MF	0.68 (99) 0.77 (124)	0.45 (93) 0.44 (80) 0.27 (95)	0.69	0.04	NS quantitative sex differences, NS qualitative sex differences
Virginia twins (Simonoff *et al.*, 1998)	Aggression (physical)	Parent rating	8–16 8–16	M+F M+F	0.76 (268) 0.31 (268)	0.46 (166) 0.22 (166)	0.58 0.21	0.18 0.11	Not tested Not tested
Missouri twins (Hudziak *et al.*, 2000)	Aggression (CBCL)	Parent rating	8–12	M F	0.77 (129) 0.73 (91)	0.50 (156) 0.40 (115)	0.77 0.70	0.00	Not tested
Dutch twins (Hudziak *et al.*, 2003)	Aggression (TRF)	Teacher rating	7	M F MF	0.72 (181) 0.71 (214)	0.33 (160) 0.33 (151) 0.26 (330)	0.69	0.00	NS quantitative sex differences
			10	M	0.73 (153)	0.41 (140)	0.72	0.00	Significant quantitative sex differences
				F	0.73 (202)	0.25 (125)	0.21	0.49	
				MF		0.17 (283)			
Dutch twins (van Beijsterveldt *et al.*, 2003)[c]	Aggression (CBCL)	Parent rating	3	M	0.81 (1055)	0.55 (1066)			Significant quantitative sex differences
				F	0.82 (1226)	0.53 (997)			
				MF		0.48 (2144)			
			7	M	0.83 (927)	0.48 (1069)			Significant quantitative sex differences
				F	0.84 (898)	0.53 (858)			
				MF		0.51 (1723)			
			10	M	0.84 (526)	0.50 (621)			Significant quantitative sex differences
				F	0.79 (471)	0.55 (458)			
				MF		0.47 (907)			
			12	M	0.86 (289)	0.45 (317)			Significant quantitative sex differences

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
				F	0.83 (237)	0.55 (233)			
				MF		0.57 (433)			
Canadian twins (Dionne *et al.*, 2003)	Aggression (physical)	Parent rating	1.5	M+F	0.59 (107)	0.28 (174)	0.58	0.00	Not tested
UK (E-risk) twins (Taylor, 2004)	Aggression (CBCL)	Parent rating	5	M+F	0.73 (602)	0.24 (514)	0.72	0.00	Not tested
South Wales twins (Button *et al.*, 2004)	Aggression (IAB)	Self-report	11–18 Mean=13.8	M+F	0.64 (115)	0.40 (143)	0.68	0.00	Not tested
Finnish twins (Vierikko *et al.*, 2004)	Aggression (MPNI)	Parent rating	12	M	0.72 (260)	0.59 (292)	0.14	0.75	Significant quantitative sex differences
				F	0.78 (300)	0.53 (278)	0.54	0.25	
				MF		0.58 (517)			
Dutch twins (Polderman *et al.*, 2006)	Aggression (TRF)	Teacher rating (same teacher)	5	M+F	0.84 (67)	0.43 (59)	0.49	0.00	Not tested
		Teacher rating (different teachers)	5	M+F	0.40 (45)	0.21 (44)			
Colorado twins (Haberstick *et al.*, 2006a)	Aggression (CBCL)	Parent rating	7[b]	M	0.74 (69)	0.56 (76)	0.79	0.00	NS quantitative sex differences
				F	0.79 (91)	0.44 (62)			
			9	M	0.57 (73)	0.50 (63)	0.76	0.00	NS quantitative sex differences
				F	0.76 (75)	0.55 (60)			
			10	M	0.77 (58)	0.47 (52)	0.76	0.00	NS quantitative sex differences
				F	0.70 (67)	0.56 (57)			
			11	M	0.64 (58)	0.41 (55)	0.84	0.00	NS quantitative sex differences
				F	0.86 (56)	0.59 (49)			
			12	M	0.68 (69)	0.45 (61)	0.79	0.00	NS quantitative sex differences
				F	0.83 (78)	0.45 (65)			
	Aggression (TRF)	Teacher rating	7	M	0.63 (71)	0.39 (70)	0.58	0.00	NS quantitative sex differences
				F	0.56 (79)	0.34 (62)			

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
			8	M	0.58 (66)	0.46 (62)	0.61	0.00	NS quantitative sex differences
				F	0.70 (70)	0.41 (60)			
			9	M	0.54 (63)	0.29 (59)	0.59	0.00	NS quantitative sex differences
				F	0.67 (74)	0.37 (53)			
			10	M	0.39 (63)	0.44 (56)	0.43	0.00	NS quantitative sex differences
				F	0.49 (64)	0.18 (54)			
			11	M F	0.56 (68) 0.50 (70)	0.12 (54) 0.45 (54)	0.52	0.00	NS quantitative sex differences
			12	M	0.35 (55)	0.32 (39)	0.42	0.00	NS quantitative sex differences
				F	0.48 (60)	0.24 (49)			
Ad-Health (Cho *et al.*, 2006)	Aggression	Self-report	12–19	M	0.47 (141)	0.29 (131)	0.50	0.00	Not tested
				F	0.47 (141)	0.27 (114)	0.30	0.00	
				MF		0.21 (197)			
Colorado twins (Gelhorn *et al.*, 2006)	Aggression (DISC items)	Self-report	11–18 Mean=14.5	M+F	0.47 (531)	0.27 (569)	0.49	0.00	Not tested
				MF		0.28 (212)			
Finnish twins (von der Pahlen *et al.*, 2008)	Aggression (BPAQ)	Self-report	18–33	M	0.45 (190)	0.22 (167	0.70	0.00	Not tested
				F	0.52 (608)	+321 sibs)	0.69	0.00	
				MF		0.18 (387 +1838 sibs) 0.20 (508 +1559 sibs)			
Norwegian twins (Czajkowski *et al.*, 2008)	Passive aggression (DSM-IV)	Self-report	19–36 Mean=28.2	M	0.35 (221)	0.45 (116)	0.14	0.18	NS quantitative sex differences,
				F	0.30 (448)	0.19 (261)			
				MF		0.21 (340)			NS qualitative sex differences
Tennessee twins (Tackett *et al.*, 2009)	Relational aggression (CAPS)	Self- report	9–18	M	0.54 (356)	0.39 (328)	0.49	0.00	NS quantitative sex differences
				F	0.41 (376)	0.36 (332)			

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
				MF		0.16 (589)			
		Parent rating	9–18	M	0.66 (356)	0.61 (328)	0.21	0.46	Significant quantitative sex differences
				F MF	0.65 (376)	0.35 (332) 0.48 (589)	0.42	0.22	
California twins—RFAB cohort (Baker *et al.*, 2008)	Reactive aggression (RPQ)	Parent rating	9–10	M	0.48 (141)	0.35 (87)	0.26	0.27	NS quantitative sex differences
				F	0.60 (142)	0.46 (98)			
				MF		0.50 (151)			
		Self-report	9–10	M	0.38 (138)	0.28 (83)	0.38	0.00	Significant quantitative sex differences
				F	0.37 (139)	0.38 (96)	0.00	0.36	
				MF		0.08 (146)			
		Teacher rating	9–10	M	0.59 (67)	0.49 (45)	0.20	0.43	NS quantitative sex differences
				F	0.70 (68)	0.43 (45)			
				MF		0.60 (62)			
	Proactive aggression (RPQ)	Parent rating	9–10	M	0.61 (141)	0.34 (87)	0.32	0.21	NS quantitative sex differences
				F	0.57 (142)	0.48 (98)			
				MF		0.55 (151)			
		Self-report	9–10	M	0.60 (138)	0.34 (83)	0.50	0.00	Significant quantitative sex differences
				F	0.12 (139)	0.28 (96)	0.00	0.14	
				MF		0.14 (146)			
		Teacher rating	9–10	M	0.56 (67)	0.42 (45)	0.45	0.14	NS quantitative sex differences
				F	0.74 (68)	0.38 (45)			
				MF		0.35 (62)			
California twins—RFAB cohort (follow-up) (Tuvblad *et al.*, 2009)	Reactive aggression (RPQ)	Parent rating	11–14	M	0.49 (102)	0.33 (55)	0.43	0.15	NS quantitative sex differences
				F	0.58 (98)	0.38 (77)			
				MF		0.42 (103)			

Study sample (author, year)	Aggression measure	Assessment method	Age in years	Sex	MZ *r*(N)	DZ *r*(N)	h^2	c^2	Sex limitation effects
	Proactive aggression (RPQ)	Parent rating	11–14	M	0.55 (102)	0.35 (55)	0.48	0.08	NS quantitative sex differences
				F	0.46 (98)	0.27 (77)			
				MF		0.40 (103)			
Weighted average				M	0.66	0.42			
				F	0.63	0.35			
				MF	0.59	0.38			
				M+F		0.28			

MZ, monozygotic; DZ, dizygotic; M, male twin pairs; F, female twin pairs; MF, male–female twin pairs; M + F, male and female pairs combined; h^2, heritability; c^2, shared environment; CBCL, Child Behavior Checklist (Achenbach, 1991b) [20 items scored as 0 (not true), 1 (somewhat true), and 2 (very true), e.g., bragging and boasting, argues a lot, cruelty or meanness to other, disobedience (home and school)]; MOCL, Mothers' Observational Checklist, including the following behaviors: rejection, destructiveness, negativism, noncompliance, teasing, physical negative, insult, verbal threat, yelling; ACL, Adjective Checklist (Gough, 1960) [consists of 300 adjectives that yields 26 scales]; MCPS, Missouri Children's Picture Series (Sines *et al.*, 1966) [consists of 238 line drawings, each portrays the figure of a child engaged in some activity or situation, the subject is required to sort the cards into two groups, those that look like fun and those that do not look like fun]; PSR, Parent Symptom Ratings (Conners, 1970) [includes six aggression items: bullying, hits or kicks other children, mean, sassy to grown-ups, fights constantly, picks on other children]; IBS, Interpersonal Behavior Survey (Mauger, 1980) [includes items such as: “some people think I have a violent temper” or “I try not to give people a hard time”]; The Mathews Youth Test for Health (MYTH; Mathew and Angulo, 1980) [developed to measure Type A behavior in school-aged children. The instrument consists of 17 items characterized by overt type A behavior and yields two subscales: impatience/aggression and competitive achievement striving]; BDHI, Buss-Durkee Hostility Inventory (Buss and Durkee, 1957) [contains of three subscales: the assault scale (10 items on physical aggression), the verbal hostility scale (13 items on verbal aggression), and the indirect hostility scale (nine items on indirect or undirected or displaced aggression)]; MPQ, Minnesota Personality Questionnaire (Tellegen, unpublished) [physically aggressive, vindictive, likes violent scenes, higher order factor, negative emotionality]; Physical Aggression scale, (Simonoff *et al.*, 1998) [items on physical aggression, extortion, public fight, use of weapon in a fight, cruelty to animal, thrown objects at people, carried a weapon, sworn at teacher, based on Olweus, 1989]; Physical Aggression scale (Dionne *et al.*, 2003) is a 37 item check list on which parents reported whether the child engaged, sometimes engaged, often engaged in a behavior. Based on factor analysis, 10 of the 37 behaviors were determined as direct physical aggression, for example, is cruel toward others, bullies other children, bites others, kicks, fights, takes things away from others, pushes, threatens to hit; IAB, instrument of aggressive behavior (Olweus, 1989) [contains two subscales: aggressive and nonaggressive antisocial behavior. The aggression scale contains 11 items of direct verbal and physical aggression, e.g., swearing at a teacher, bullying]; MPNI, Multidimensional Peer Nomination Inventory (Pulikkined *et al.*, 1999) [contains of 38 items and the aggression subscale contains of six items, e.g., calls people names, may hurt other kids, bullying, goes around telling people's secrets to others]; Ad-Health [aggression is based on four items, got into a serious physical fight, hurt someone badly enough they needed medical care, used to threaten to use a weapon to get something from someone, took part in a gang fight]; DISC, Diagnostic Interview Schedule for Children (Shaffer *et al.*, 2000) [Gelhorn *et al.* (2006) only included aggression to people or animals, items 1–7]; BPAQ, Buss and Perry Aggression Questionnaire (Buss and Perry, 1992) [nine items on physical aggression and five items on verbal aggression, e.g., I cannot help getting into arguments when people disagree with me, I have threatened people I know, I get into fights a little bit more often than average people]; DSM-IV, the Norwegian version of the Structured Interview for DSM-IV personality (Pfohl *et al.*, 1997) [the instrument is a semi-structured diagnostic interview for the assessment of all DSM axis II disorders, including passive-aggressive personality disorder]; CAPS, Child and Adolescent Psychopathology Scale (Lahey *et al.*, 2004) [relational aggression was assessed via the CAPS, a structured interview assessing DSM-IV symptoms of common childhood disorders. Seven items measured relational aggression, for example, tried to keep kids he/she does not like outside his/her friend group, spread rumors to make others stop liking someone, stopped talking to people because he/she was mad at them, teased other people in a mean way]; RPQ, Reactive and Proactive Questionnaire (Raine *et al.*, 2006). [The RPQ is a validated 23-item questionnaire designed to measure reactive and proactive aggression in children and adolescents from the age of 8. The RPQ includes 11 reactive items (e.g., “he/she damages things when he/she is mad”; “he/she gets mad or hits others when they tease him/her”) and 12 proactive items (e.g., “he/she threatens and bullies other kids”; “he/she damages or breaks things for fun”). The items in the RPQ have a three-point response format: 0=never, 1=sometimes, 2=often.]; RFAB, USC twin study of Risk Factors for Antisocial Behavior; TCHAD: Twin Study of Child and Adolescent Development.

[a] Heritability estimate is based on either Holzingers' H or Falconer equation and did not report shared environmental influences.

[b] Parent reported CBCL ratings were not collected at age 8.

[c] Genetic and shared environmental estimates were not reported by the authors.

Table 8.3

Forms of Aggression

Form of aggression	Description
Reactive/hostile/affective	Angry or frustrated responses to a real or perceived threat
Proactive/instrumental	Planning, the motive of the act extends beyond harming the victim
Direct/physical	Intentionally causing pain or harm to the victim
Indirect/relational	Relational social manipulation such as gossip and peer exclusion