

Genetic Influence on the Development of Antisocial Behavior

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Antisocial behavior is studied by virtually every field of social science, including psychology, sociology, epidemiology, and criminology, as well as psychiatry. Discussions concerning the complex interplay between genes and environments are therefore of interest to researchers across many domains. Although each field has its own particular ways of assessing antisocial behavior, there is a strong correlation between different measures. For example, in recent versions of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (American Psychiatric Association 1987, 1994, 2000), a diagnosis of adult antisocial personality disorder (ASPD) is given only if the individual also meets criteria for earlier child and adolescent conduct disorder. Psychiatrists and psychologists have recognized that criminal behavior may be preceded by other psychiatric disorders, such as attention-deficit disorder and conduct disorder in childhood (Mannuzza et al. 1989; Satterfield et al. 1982). The majority of adult criminals are also thought to have ASPD, although this is partly due to the fact that committing illegal activities is one of the diagnostic criteria for ASPD.

Many measures of adolescent delinquent and aggressive behavior contain items similar to those found in DSM-III-R and DSM-IV criteria for conduct disorder. Because of the strong correlation across different measures of antisocial behavior, I focus here on genetic and environmental influences on antisocial

behavior, broadly defined. One notable exception is that I omit discussion of attention-deficit/hyperactivity disorder (ADHD). Although there is evidence that ADHD may be related to conduct disorder, the fact that there may be specific genetic influence governing the more cognitive processes of inattention (e.g., Faraone et al. 2000; Silberg et al. 1996b) means that extrapolation from results of behavioral genetic studies of ADHD may be misleading.

In contrast to findings from studies of many other phenotypes, when taken as a whole, results from behavioral genetic research on antisocial behavior are often inconsistent across studies. Although there is strong evidence of at least some degree of genetic influence on individual differences in antisocial behavior, overall estimates of heritability (i.e., the proportion of variation due to genetic factors) can vary substantially across studies, from zero influence to .90, and appear to be influenced by a multitude of methodological and substantive factors, including sample age, the rater (e.g., self-report versus maternal ratings), and developmental heterogeneity. Also in question is the extent to which between-family environmental factors (i.e., shared environments) impact on individual differences in antisocial behavior.

In this chapter, I attempt to bring some sort of rhyme and reason to the underlying inconsistencies across studies. The chapter begins with a simple overview and comparison of results from twin and adoption studies of antisocial behavior divided into three different developmental epochs (early childhood, adolescence, and adulthood). I then discuss results from the handful of longitudinal and quasi-longitudinal behavioral genetic studies regarding genetic influences on stability and change in antisocial behavior. It should be noted that time and space constraints limit the number of studies that can be reviewed, so the focus of this chapter is primarily on general trends derived from population-based samples. However, in a recent review article of the genetics of antisocial behavior, Slutske (2001) reported that there are more than 100 published twin and adoption studies of antisocial behavior. Interested readers are encouraged to consult this brief review, as well as to read results from different meta-analyses of antisocial behavior that try to account for some of the systematic variation in results across studies (e.g., Miles and Carey 1997; Rhee and Waldman 2002).

In the second part of the chapter, I discuss factors that may account for heterogeneity of results across studies. These factors include methodological artifacts, such as different raters, as well as potentially theoretical reasons for actually expecting observed heterogeneity of genetic influence, such as the effects of age on expression of genetic and environmental influence, and evidence for different developmental trajectories of antisocial behavior across the life span.

Finally, I conclude the chapter by discussing one of the most exciting new areas of research—the role of gene \times environment interactions in the development of antisocial behavior—which promises to make our understanding of genetic and environmental influences on antisocial behavior even more complex.

Genetic Influence on Developmental Epochs

Problem Behavior in Early Childhood

There is reasonably compelling and consistent evidence for genetic influences on early childhood aggression and problem behaviors, although the absolute magnitude of this influence varies across studies (and sometimes within a study). For example, in one of the earlier studies of problem behavior in young childhood, Ghodsian-Carpey and Baker (1987) reported wide-ranging heritabilities of .24 to .94 for maternal ratings of different indicators of aggressive behavior among 4- to 7-year-old twins. With the exception of a small study of 229 Colorado preschool twin pairs (Schmitz et al. 1994), which found only limited evidence for genetic influence on antisocial behavior, the majority of other studies using maternal ratings of early childhood problem behavior have reported heritability estimates in the range of .50 to .60 (e.g., Bartels et al. 2004; van den Oord and Rowe 1997; van den Oord et al. 1996). In one of the few studies using child self-report, Arseneault et al. (2003) used an innovative interview, the Berkeley Puppet Interview, to obtain self-reports from 5-year-old children on their own hostile/aggressive, conduct-disordered, and oppositional behavior. In a univariate analysis of a composite measure of these three behaviors, heritability was estimated at .30, with a nonsignificant shared environmental influence of .10. Thus, results from

behavioral genetic studies of problem behavior in early childhood are, with only one major exception (i.e., the Schmitz et al. 1994 study), largely in agreement that genetic factors are important for individual differences in problem behavior, with most studies showing estimates of moderately high heritabilities (.50-.60).

Late Childhood and Adolescent Antisocial Behavior

Delinquency and Conduct Disorder

In contrast to studies of problem behavior in early childhood, there is considerable evidence that genetic influences are weaker, and shared environmental influences are stronger, for late childhood and adolescent delinquent or conduct-disordered behavior. In one of the first twin studies of adolescent self-reports of antisocial behavior (including items assessing minor delinquency, theft, and aggression) from approximately 265 same-sex American twin pairs, Rowe (1983) concluded that both genetic and shared environmental factors may contribute to adolescent delinquency.

More recent studies using large-scale population-based samples have also concluded that genes and environments each account for between 20% and 25% of the variation in adolescent self-reported delinquency and conduct problems (e.g., Eaves et al. 1997; Taylor et al. 2000). In addition, at least three published twin studies have examined genetic and environmental influences on adult retrospective reports of conduct disorder. Two of the studies found nearly identical results among males. In the earlier study of male twins from the Vietnam Era Twin Registry, Lyons et al. (1995) reported only a modest heritability of .07, and a shared environmental influence of .31, for conduct-disordered behavior prior to age 15. Among male twins from the Virginia Twin Registry, Jacobson et al. (2002), reported estimates of .06 and .28, respectively, for genetic and shared environmental influences on conduct-disordered behavior prior to age 15, although heritability was higher, and shared environmental influences were lower, among females. The third study (Slutske et al. 1997), using adult twin data from the Australian Twin Register, may be viewed as an outlier, as the heritability for conduct disorder under the

best-fitting model was much higher than in other studies (.71) for both males and females, and shared environmental influences were zero.

Studies of maternal ratings of adolescent delinquency and conduct-disordered behavior also report strong evidence for both significant genetic and shared environmental influences, with estimates for genetic factors typically ranging from .20 to .50, and estimates of shared environmental influences ranging from .30 to .40 (Edelbrock et al. 1995; Eley et al. 1999; Thapar and McGuffin 1996).

Finally, a handful of adoption and twin-sibling studies have examined genetic and environmental influences on problem behavior in adolescence. Results from these studies are somewhat less consistent. For one of the studies, based on a sample of adolescents in the Iowa Adoption Study, results suggested a complete absence of genetic influence on variation in antisocial behavior (Cadoret 1978). Specifically, this study found virtually identical rates of psychiatric disorders among children with antisocial biological background (25%) and control subjects (27%); this finding indicates that the presence of a biological (genetic) risk for antisocial behavior was not related to greater likelihood of antisocial behavior compared with a lack of a family history of antisocial behavior. In contrast, in the second study, based on a sample of 172 adoptive and biological twin pairs from the Colorado Adoption Project, Deater-Deckard and Plomin (1999) reported significant influences on both genetic (.36) and shared environmental (.22) influences on maternal ratings of delinquent behavior. Different still are results from the third study, based on data from twin, full siblings, half-siblings, and unrelated siblings in the Non-shared Environment and Adolescent Development Project, which reported fairly high estimates of heritability (.63-.66) and little evidence for shared environmental factors (Neiderhiser et al. 1996). In conclusion, although many studies of adolescent delinquency find stronger shared environmental influences and weaker genetic influences than studies of problem behaviors in young children, there is inconsistency across studies.

Aggressive Behavior

One of the factors that might account for differences in estimates of heritability and shared environmental influences on antisocial behavior in late childhood and adolescence is whether studies focused on aggressive antisocial behavior, nonaggressive antisocial behavior, or a measure combining both aggressive and nonaggressive behaviors. Specifically, there is accumulating evidence that aggressive behavior in late childhood and adolescence has a stronger genetic component than nonaggressive delinquent or conduct-disordered behavior. For example, at least three published studies using parental reports on the Child Behavior Checklist (CBCL; Achenbach and Edelbrock 1979) examined differences in heritability for the Aggressive versus Delinquent Behavior subscales. All three of these studies reported substantially higher estimates of heritability (range = .60–.80) for aggressive behaviors than for nonaggressive delinquent behaviors (range = .30–.60; Edelbrock et al. 1995; Eley et al. 1999; Gjone and Stevenson 1997). In addition, two of these studies reported that shared environmental factors did not significantly influence twin similarity for aggressive behavior, although they explained between 27% and 64% of the variation in delinquent behavior (see Edelbrock et al. 1995; Eley et al. 1999). What is remarkable about these results is the overall similarity of pattern across studies, especially since the results were based on data from eight different birth cohorts across four different countries (Sweden, Britain, Norway, and United States).

To test the hypothesis that more severe aggressive or psychopathological behavior is more heritable than nonaggressive antisocial behavior, we recently fitted multivariate behavioral genetic models to antisocial data from 9- to 10-year-old twins in the University of Southern California (USC) Risk Factors for Antisocial Behavior Study (K. C. Jacobson, L. Baker, A. Raine, manuscript in preparation, 2005). Figure 6-1 presents this model, which was fitted to maternal ratings on five different indices of antisocial behavior: 1) Aggressive (AGG) and 2) Delinquent (DEL) subscales of the CBCL (Achenbach and Edelbrock 1979); 3) ratings of conduct disorder symptoms from the Diagnostic Interview Schedule for Children (DISC; Costello et al. 1985); 4) a newly developed

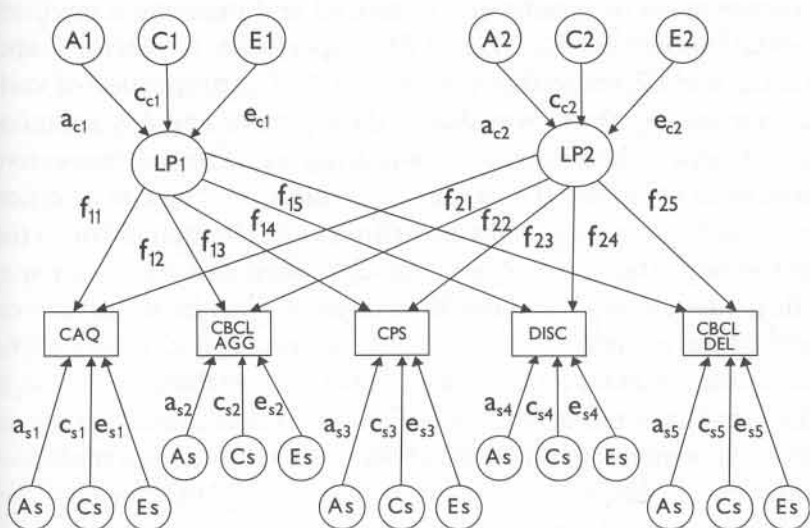


Figure 6-1. Multivariate behavioral genetic model for covariation across five measures of antisocial behavior.

CAQ=Child Aggression Questionnaire; CBCL=Child Behavior Checklist (AGG=Aggression subscale; DEL=Delinquent Behavior subscale); CPS=Child Psychopathy Scale; DISC=Diagnostic Interview Schedule for Children.

A=additive genetic influence; C=shared environmental influence; E = non-shared environmental influence; LP1=first latent factor; LP2=second latent factor. Subscript *c* represents genetic and environmental influences that are common across the five measures and operate through the latent phenotypes. In contrast, subscript *s* denotes genetic and environmental influences that are specific to each variable. Parameters f_{11} – f_{15} through f_{21} – f_{25} represent the factor loadings for each of the two latent factors.

questionnaire, the Childhood Aggression Questionnaire (CAQ; A. Raine, K. Dodge, R. Loeber, et al., manuscript under review, 2005); and 5) the Child Psychopathy Scale (CPS; Lynam 1997). These measured variables are represented by the rectangles in Figure 6-1.

The model shown assumes that there are two underlying latent phenotypes (i.e., factors)—latent factors LP1 and LP2—that account for the correlations across the five different measures. Arrows from the latent phenotypes to the measured variables represent factor loadings (f_{11} – f_{15} for Factor 1; f_{21} – f_{25} for Factor 2).

Variation in these latent phenotypes, in turn, is accounted for by a common set of genetic and of shared and nonshared environmental influences (A1, C1, and E1, respectively, for Factor 1, and A2, C2, and E2, respectively, for Factor 2). The proportion of variation in each of the factors due to these genetic and environmental influences is obtained by squaring each of the respective parameter estimates (i.e., a_{c1} , c_{c1} , e_{c1} , and a_{c2} , c_{c2} , e_{c2}). In addition to modeling variation in the measured variables that is due to the underlying latent phenotypes, this approach also allows for specific genetic, shared, and nonshared environmental influences on each of the measured variables that are not shared with genetic and environmental influences on the other variables (i.e., a_{s1} – e_{s5}). The two-factor model shown in Figure 6–1 was tested against an alternative model in which all of the covariation across measures could be accounted for by a single underlying latent phenotype. This latter model tested the hypothesis that all five measures of antisocial behavior represented a single, unitary construct.

Preliminary analyses of both the same-sex male and same-sex female twins strongly indicated the presence of two distinct, underlying latent factors that account for the correlation across measures. Figure 6–2 presents the results from the analysis of the female twins. For simplicity, the specific genetic and environmental influences are not shown, as they relate to genetic and environmental variation in each of the five measures that is not shared with the other measures. Dashed lines represent parameter estimates that are not significantly different from zero. As can be seen in this figure, the first factor (LP1) was defined by significant factor loadings (range = .50 to .99) on all five measures of antisocial behavior. This suggests the presence of a “general deviance” factor, which accounts for 25% to over 90% of the variance in each measure. In contrast, the second factor (LP2) loaded significantly on only the two measures of aggression and the CPS, indicating that it may be tapping more severe aggressive and psychopathic behavior that is not shared with a general deviance factor. This factor accounted for 20%–36% of the variance in these three measures.

Figure 6–2 also shows the genetic and environmental contributions to variation in these underlying latent phenotypes. As can be

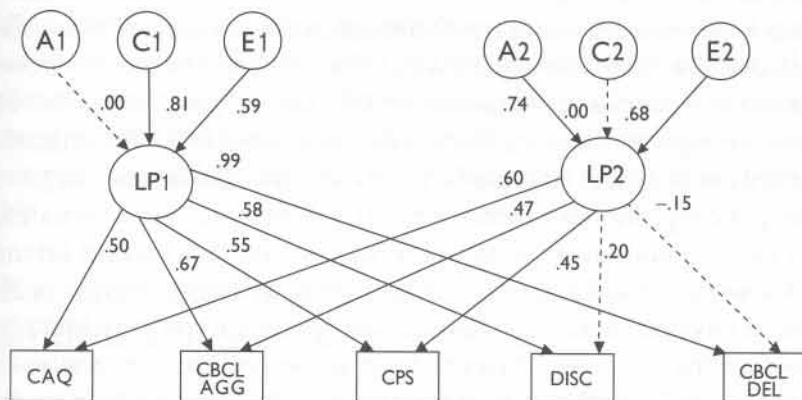


Figure 6-2. Standardized parameter estimates for genetic and environmental factor loadings on five measures of antisocial behavior for female-female twins.

CAQ=Child Aggression Questionnaire; CBCL=Child Behavior Checklist (AGG=Aggression subscale; DEL=Delinquent Behavior subscale); CPS=Child Psychopathy Scale; DISC=Diagnostic Interview Schedule for Children.

A=additive genetic influence; C=shared environmental influence; E = non-shared environmental influence; LP1=first latent factor; LP2=second latent factor. Dashed lines represent parameter estimates that are not significantly different from zero.

seen very clearly in these results for female-female twins, the pattern of genetic and environmental influence on variation differed dramatically across the two different underlying latent phenotypes. Specifically, for Factor 1, the general deviance factor, all of the variation could be accounted for by environmental factors. Shared environmental factors accounted for approximately 65% of the variance ($.81 \times .81$), and nonshared environmental factors accounted for the remaining 35% ($.59 \times .59$). In contrast, the heritability of the aggressive-psychopathology factor (LP2) was estimated at approximately 54% ($.74 \times .74$), and nonshared environmental influences accounted for the remaining 46% of the variance in this factor ($.68 \times .68$).

Moreover, although the phenotypic structure of the first general deviance factor was similar for males and females, the absolute magnitude of genetic and environmental influences on this underlying factor varied across sex. In particular, among males,

genetic factors accounted for 49% of the variance in this underlying factor (results not shown), although the heritability was estimated to be zero among females. Conversely, shared environmental influences on variation in this underlying latent factor were stronger for females (64%) than for males (40%). In contrast, not only was the phenotypic factor structure of the second, aggressive-psychopathology factor similar across males and females, but genetic and environmental influences on this second latent factor were virtually identical across sexes, with genetic and non-shared environmental factors each accounting for approximately one-half the variation. Results from these preliminary analyses support the hypothesis that general problem behavior is more strongly influenced by environmental factors, whereas aggressive, pathological antisocial behavior is more strongly influenced by genetic factors.

Adult Antisocial Behavior and Criminality

In contrast to the somewhat inconsistent results for genetic influence on adolescent antisocial behavior, results from twin and adoption studies of adults are nearly unanimous in support of the hypothesis that aggressive and criminal behavior have a fairly substantial genetic component (see Brennan and Mednick 1993 and DiLalla and Gottesman 1989 for reviews of the earlier literature, and Rhee and Waldman 2002 for a more recent review and meta-analysis). For example, on the basis of data from the Danish Adoption Registry, it was found that the average rate of criminal convictions among male adoptees with neither biological nor adoptive parent convictions was 13.5%. When adoptive parents, but not biological parents, had criminal convictions, the rate of criminality among adoptees increased to only 14.7%. In contrast, the rate of criminal convictions when biological parents, but not adoptive parents, had criminal convictions increased to 20.0% (Brennan and Mednick 1993; Mednick et al. 1984).

Thus, biological (genetic) risk, but not environmental risk, was associated with a higher incidence of criminal convictions, supporting the notion of genetic influence on adult antisocial behavior. Similar findings for high heritability of adult antisocial behavior have been reported in other adoption (e.g., Cadoret 1978) and twin

(e.g., Cloninger and Gottesman 1997; Jacobson et al. 2002; Lyons et al. 1995) samples. Thus, for behavioral genetic investigations of adult antisocial behavior, most studies find evidence for heritabilities between .40 and .60. Moreover, the results from twin studies suggest that the importance of shared environmental influences on individual differences in adult antisocial behavior is negligible.

Differences in Heritability of Antisocial Behavior Over Time

Thus far, I have presented results from a variety of twin and adoption studies of antisocial behavior at different developmental periods. Although there is considerable variation across studies in terms of the actual magnitude of genetic and environmental factors, at least two general patterns emerge: 1) genetic influence increases from adolescence to adulthood, and 2) environmental influences appear to be the most important factor in accounting for variation in antisocial behavior during the adolescent years. However, drawing conclusions about developmental changes in the relative influence of genetic and shared environmental factors on antisocial behavior from cross-study comparisons is problematic, because studies use different samples and different measures of antisocial behavior. These differences in methodologies may introduce systematic biases.

To circumvent these possible biases, at least two meta-analyses have been conducted to statistically test for differences in genetic and environmental influences across age while controlling for potential differences across study in sample and methodology. In the first study, which focused on studies of aggressive antisocial behaviors, Miles and Carey (1997) concluded that although juvenile aggressive behavior was influenced equally by both genetic and shared environmental factors (each accounting for approximately 20% of the variance), genetic influences on adult aggressive behavior were markedly higher (40%) and shared environmental influences were negligible. The second study, which reviewed 51 twin and adoption studies of both aggressive and nonaggressive behavior, also found evidence of sig-

nificant age differences (Rhee and Waldman 2002). However, in these authors' analysis, the primary difference was in the effect of the shared environment. Among studies of children and adolescents, shared environmental effects accounted for 16%–20% of the variance, compared with estimates of only 9% among studies of adults. Genetic factors, in contrast, accounted for 41%–46% of the variance at all ages.

Although meta-analyses can account for some of the factors that may bias cross-sectional comparisons, the best way of estimating changes in the relative importance of genetic and environmental influences on antisocial behavior over time is to rely on data that assesses antisocial behavior from the same sample of twins at two or more different ages. At least two, large-scale, quasi-longitudinal twin studies have examined the question of age-related changes in heritability estimates, and both studies reported remarkably consistent evidence for significant changes in the heritability of antisocial behavior across age. In the first study, using retrospective data from adult male-male twins in the Vietnam Twin Registry, Lyons et al. (1995) found that heritability increased from .07 for antisocial behavior assessed prior to age 15 (juvenile antisocial behavior) to .43 for antisocial behavior assessed at ages 15 and older (adult antisocial behavior). Conversely, shared environmental influences explained 31% of the variation in juvenile antisocial traits but only 5% of the variation in adult antisocial traits (Lyons et al. 1995). The second study, based on an analysis of retrospective reports of child, adolescent, and adult antisocial behavior that drew on data from both male and female twin pairs from the Virginia Twin Registry, also found evidence for increasing genetic effects and decreasing shared environmental effects, especially among males (Jacobson et al. 2002). Among males, heritability increased from .06 to .40, and shared environmental influences decreased from .28 to .11. Among females, the difference in heritability was less marked (.29 to .42), but still showed a pattern of increasing genetic effects across age.

Thus, taken together, studies suggest that there is systematic heterogeneity across age in estimates of the overall magnitude of genetic and shared environmental factors. These findings are consistent with the patterns of increasing heritability and decreas-

ing effects of shared environment with age that have been observed in studies of other phenotypes, such as personality and cognition (see Plomin 1986 for review). These patterns suggest that effects of shared family, community, and neighborhood characteristics that influence antisocial behavior during adolescence may have little continuing influence on adult antisocial behavior.

Genetic and Environmental Influences on Continuity and Change in Antisocial Behavior

Longitudinal Behavioral Genetic Studies

In addition to estimating more reliably the potential changes in the magnitude of genetic and environmental influences on antisocial behavior over time, longitudinal behavioral genetic studies can tell us the extent to which genetic and environmental factors contribute to *continuity and change* in antisocial behavior over time. When different manifestations of antisocial behavior across developmental periods are considered, psychiatrists, psychologists, and criminologists are mainly in agreement that there is remarkable phenotypic stability to antisocial behavior.

In a seminal study using data from four longitudinal studies of males, Robins (1978) concluded that although most children with antisocial behavior problems do not become antisocial adults, virtually all adults with antisocial behavior problems were antisocial children. What is particularly remarkable about this result is that it was replicated in four independent samples, even though the samples represented males from different racial and social backgrounds and from different historical eras (Robins 1978). Childhood behaviors were more predictive of adult antisocial behavior than family variables, including parental antisocial behavior, family structure, and social class.

Following this landmark report, there have been dozens of studies demonstrating a strong continuity between child, adolescent, and adult antisocial behaviors. For example, rates of adult criminal convictions are higher among samples of individuals diagnosed with ADHD compared with control groups (Mannuzza

et al. 1989; Satterfield et al. 1982), and attention problems, impulsivity, and activity have been found to be precursors to later antisocial behavior (Loeber and Hay 1991). Likewise, aggressive behavior in childhood has also been found to predict delinquent and criminal behavior during adolescence and young adulthood. For instance, in a follow-up study of more than 1,000 subjects from a cohort of 10-year-olds in Sweden, Stattin and Magnusson (1989) reported that teacher ratings of aggression at age 13 predicted conviction by age 26, particularly multiple convictions. Finally, childhood ADHD and conduct disorder predict adult ASPD and criminality (e.g., Simonoff et al. 2004).

Developmental Behavioral Genetic Studies

Although the studies summarized above provide evidence for a strong phenotypic continuity of antisocial behavior, they cannot tell us *why* there is stability of behavior over time. Understanding the mechanisms behind both stability and change in antisocial behavior is of particular importance to clinicians, since any plans for prevention need to incorporate the processes through which antisocial children turn into antisocial adolescent and adults. Developmental behavioral genetic studies are a means of investigating the *sources* of developmental stability and change in behavior—that is, the extent to which genes and environments contribute to continuity and change. In this type of study, genetically informative kin are interviewed or observed on more than one occasion, and genetic influence on continuity and change is estimated from the pattern of cross-twin, cross-time correlations. Although longitudinal samples of this type are rare, inspection of the results from the existing published studies reveals some interesting patterns.

Sources of Stability in Antisocial Behavior Across Childhood and Adolescence

In one of the first developmental behavioral genetic studies of antisocial behavior, van den Oord and Rowe (1997) examined the developmental stability of problem behavior, including antisocial behavior, in a study of full siblings, half siblings, and cousins from ages 4–6 to 6–8 to 8–10 years. The authors concluded that

there was substantial evidence of a genetic effect on the stability of problem behavior, because the cross-time sibling correlation was higher among full sibling pairs (who share, on average, 50% of their segregating genes) than among half siblings (who share only 25% of their segregating genes). Structural equation models revealed a moderate correlation of antisocial behavior across waves ($r = .57$). Of this stability, 46% of the correlation could be attributable to genetic influences, and the remaining 54% was attributed to shared environmental influences.

Other evidence for genetic influence on the continuity of antisocial behavior across childhood and early adolescence comes from a large body of research using longitudinal data from a sample drawn from The Netherlands Twin Registry. In this study, assessments of problem behavior using the maternal reports of the CBCL were obtained at ages 3, 7, 10, and 12. In one of the most recent and comprehensive analyses from this sample, Bartels et al. (2004) reported cross-time correlations for maternal reports of CBCL Externalizing symptoms ranging from .46 to .76, demonstrating remarkable stability of antisocial behavior across age. Moreover, these phenotypic correlations from age 3 to age 12 were explained predominantly by both genetic and shared environmental factors.

Evidence for a genetic basis for stability of antisocial behavior across adolescence has also been found (Eley et al. 2003; O'Connor et al. 1998). In the first longitudinal study of 405 twin and sibling pairs from the Nonshared Environment and Adolescent Development Project, O'Connor et al. (1998) found that genetic factors accounted for over 50% of the phenotypic continuity of antisocial behavior symptoms from ages 10 to 18. Interestingly, one of the few studies to examine genetic and environmental influences on aggressive versus nonaggressive delinquent behavior found evidence for a slightly different pattern of genetic and environmental influences on continuity of antisocial behavior from childhood (ages 8–9) to adolescence (ages 13–14) (Eley et al. 2003). Specifically, continuity in aggressive behavior was attributed almost entirely to genetic factors (accounting for 84% of the phenotypic correlation). In contrast, both genetic (44%) and shared environmental (54%) influences accounted for the stability in nonaggressive delinquent behaviors.

Sources of Stability in Antisocial Behavior From Adolescence to Adulthood

As of this writing, there are no published studies of change in antisocial behavior between childhood and adulthood using prospective, genetically informative samples. This is unfortunate, given the dramatic differences in heritability estimates that appear in a comparison of cross-sectional research. Nevertheless, two published studies using population-based twin samples have been able to examine the genetic and environmental influences on stability and change across antisocial behavior from childhood to adolescence by using retrospective reports of juvenile antisocial behavior and retrospective/current reports of adult antisocial behavior. In both studies, genetic factors accounted for 20%–50% of the correlations among juvenile and adult antisocial behaviors, and shared environmental factors accounted for 6%–40% of these correlations. It is especially interesting that even though shared environmental factors had only a negligible influence on adult antisocial behavior in these studies (less than 10% of the variance), these same factors appeared to explain the continuity of antisocial behavior from adolescence to adulthood.

Factors Accounting for Change in Antisocial Behavior

Results from the foregoing longitudinal studies point to significant genetic effects on continuity in problem behavior, both within and across developmental epoch. Shared environmental factors are also implicated, especially with respect to continuities of childhood antisocial behavior. In addition, some, but not all, of these studies provide evidence for the appearance of age-specific genetic effects on antisocial behavior, indicating that genetic factors may also account for *change* in antisocial behavior. Although an individual's DNA is present at birth and does not change throughout the life span, it is possible for studies to reveal the presence of "new" genetic effects on behavior. The appearance of the new genetic effects can stem from a variety of sources, including significant biological changes (e.g., puberty) that may "turn on or off" genes or from changes in the social environment that may either enhance or suppress genetic influences on behavior.

Interestingly, in contrast to the remarkable consistency in results for *stability* of genetic influence over time, studies vary greatly in their evidence for *new* genetic effects. For example, in the van den Oord and Rowe (1997) study, the influence of new genetic factors did not appear at any point in their assessments from ages 4–6 to 8–10. In contrast, Bartels et al. (2004) found evidence for significant age-specific genetic effects on behavior in their longitudinal sample of twins assessed from ages 3 to 12. Results from longitudinal studies across childhood and adulthood are likewise inconsistent. In the study of male twin pairs from the Vietnam Era Twin Registry, Lyons et al. (1995) did not find any evidence for new genetic effects on adult antisocial behavior that were not shared with genetic influences on adolescent antisocial behavior. In contrast, Jacobson et al. (2002) reported significant effects of new genetic factors on both adolescent and adult antisocial behavior in their sample of male and female twins.

Although these results may at first appear contradictory, it is possible that the demonstration of age-specific effects is related to significant biological and social changes that may moderate the expression of genetic differences that occur during a relatively short window of time. For example, Bartels et al. (2004) found that the strongest evidence for age-specific genetic influence on antisocial behavior occurred sometime between the age 3 and age 7 assessments. It is possible that the presence of new genetic influence at age 7 was related to the marked developmental, social, and biological changes that occur between the ages of 3 and 6. Because the van den Oord and Rowe (1997) study combined the ages of 4–7 into a single assessment, they may have missed the critical period in which new genetic influences are expressed. Likewise, Jacobson et al. (2002) speculated that the presence of new genetic influence on adolescent antisocial behavior may be related to pubertal processes that “trigger” the expression of genetic influence. They supported this hypothesis by noting 1) that the presence of new genetic effects appeared later among males than females and 2) that although the heritability of child antisocial behavior was greater among females than among males, by middle adolescence and adulthood, the males had “caught up” to the females. Lyons et al. (1995), in their study, assessed antisocial

behavior with only two distinct time periods, using a cutoff point of age 15. Thus, the measure of "adult" antisocial traits in this study included behaviors during middle and late adolescence, as well as adult behaviors. This choice of cutoff may have obscured any potential age-related changes.

Sources of Heterogeneity of Results Across Studies

Rater Effects

One of the most important factors to consider when comparing estimates of heritability at different ages *across studies* is that of potential *rater effects*. As can be seen in the review presented in the first part of this chapter, studies of antisocial behavior in early childhood typically find high estimates of heritability compared with studies of adolescent antisocial behavior, in which genetic influence is less certain. However, virtually all published studies of early childhood rely on ratings from others (typically parental ratings), whereas many of the studies of adolescent antisocial behavior rely on self-report. If the heritability of antisocial behavior varies systematically across reporter, biases due to rater effects could explain this particular pattern of results.

It has long been known that there are only modest correlations of reports of antisocial behavior across raters. Because of these relatively low levels of agreement across raters, it is difficult to know which person's view represents the most "accurate" perception of antisocial behavior. Agreement among parents and children regarding children's own behaviors is particularly poor. Possible explanations for this lack of agreement include the following:

1. *Differences among parents and children in interpreting and understanding the conceptualization of behaviors that are being assessed.* This is especially true for studies of young children, who may lack the cognitive capacity to understand the questions.
2. *Differences in response bias, in which parents or children may be more or less likely to report certain behaviors.* For example, parents are more likely than their children to report symptoms of

child oppositional behavior and inattention (Edelbrock et al. 1986; Loeber et al. 1989).

3. *Differences in parental supervision.* Parents are not around their children 24 hours a day, especially during adolescence, when children spend considerable amounts of time in unsupervised settings. Thus, parents have the potential to underreport certain behaviors, especially behaviors such as problem behavior, which children may be reluctant to disclose to their parents.

Potential rater effects can be particularly problematic in studies of multiple siblings in the same family (e.g., twin studies) when the same rater (usually the mother) is reporting on the behavior of both twins. If the mother has her own particular reporting bias, this could artificially influence twin correlations for both MZ and DZ twins. Such influence would result in an estimate of shared environmental influence that is upwardly biased. On the other hand, it has been suggested that mothers of MZ twins are more likely than mothers of DZ twins to view their twins as more similar (a violation of the equal environments assumption), or conversely, that mothers of DZ twins are more likely than mothers of MZ twins to rate the behavior of their twins more differently (a sibling contrast effect). These types of biases would result in artificially lower correlations for DZ twins compared with those for MZ twins, which would in turn result in heritability estimates that are upwardly biased.

Cross-sectional comparisons of results using different raters of antisocial behavior do sometimes yield higher estimates of both heritability and shared environmental influences for parental reports compared with other ratings, such as self-report (L. Baker, A. Raine, D. Lozano, manuscript in preparation, 2005; Simonoff et al. 1998). However, the only way to assess whether these higher heritabilities are due to parental bias or to different views of antisocial behavior is to conduct multivariate behavioral genetic models using data from multiple raters simultaneously. For example, Simonoff et al. (1995) obtained mother, father, and child reports of disruptive behaviors from families with adolescent male-male twin pairs. Results for questionnaire data revealed significant effects of a shared parental view. That is, mothers and fa-

thers had higher levels of agreement with each other than either parent had with the children. This lends support to the hypothesis that part of the disagreement between parents and their children is due to situational specificity (i.e., parents do not see all of their children's behaviors, and children may behave quite differently at home than they do with their friends).

When these data were analyzed separately across rater, there was evidence for greater influence of shared environmental factors for mother and father reports (.42-.58) compared with child self-report (.34), although, interestingly, there was little difference in estimates of heritability across rater (.23-.34). When shared parental view was taken into account, estimates of shared environmental influence on the underlying latent phenotype were similar to those obtained through individual child self-report. Thus, although this study cannot determine whose viewpoint on behavior is "correct," it does provide evidence that studies of adolescents based on parental report may overestimate shared environmental influences.

Heterogeneity of Antisocial Behavior

So far, this chapter has focused on estimates of genetic and environmental influences on the development of antisocial behavior while assuming that antisocial behavior is a homogeneous phenomenon. However, during the past few decades, there has been increased recognition that antisocial behavior may, in fact, represent a heterogeneous phenomenon, with a variety of distinct causes and consequences.

Although use of behavioral genetic strategies to assess potential heterogeneity in antisocial behavior is a relatively new approach, two aspects of heterogeneity have received attention in behavioral genetic designs: 1) heterogeneity as defined by different *developmental trajectories* and 2) heterogeneity based on *comorbidity with other behaviors*.

Developmental Typologies of Antisocial Behavior

In a seminal article, Robins (1978) compared life-course patterns of antisocial behavior from four large, longitudinal, population-based studies. Consistent across all studies were two seemingly

paradoxical findings: 1) most adults with ASPD had a history of childhood or adolescent conduct disorder, indicating substantial continuity of antisocial behavior across the life course; and 2) the majority of individuals with conduct disorder did not go on to become antisocial adults, suggesting some discontinuity to antisocial behavior (Robins 1978).

Results from the Cambridge Study of Delinquency among a single-birth cohort in the United Kingdom revealed similar patterns: delinquent behavior during adolescence was normative in this sample, with upward of 70% of male adolescents admitting to participating in at least one delinquent activity (West and Farrington 1973). However, the majority of adult criminal offenses were committed by the same 5%–6% of individuals (Farrington 1995). Finally, more recent examinations of sex differences in rates of antisocial or delinquent behavior have found that males outnumber females in various aspects of adult antisocial behavior (including criminal offenses and rates of adult ASPD) by a magnitude of 5–10 males to every 1 female, whereas the sex ratio for adolescent-onset antisocial behavior is more equal (approximately 1.5:1) (Moffitt and Caspi 2001). Taken together, the results from these studies suggest that the risk factors that influence transitory, adolescent-based antisocial behavior may differ from those that influence long-term, life-course persistent antisocial behavior.

Moffitt's (1993) theory of *life-course persistent* (LCP) versus *adolescent-limited* (AL) delinquency has been especially influential in current research on the development and heterogeneity of antisocial behavior. Moffitt emphasizes differences in timing, continuity, frequency, and severity of antisocial behavior across these two primary groups, and also points to different biological, psychological, and social mechanisms. In brief, *AL delinquency* is defined by transient antisocial behavior, with an onset generally in adolescence, that is less frequent and consists of more benign antisocial behaviors. In contrast, *LCP delinquency* is characterized by earlier onset of antisocial behavior, a wider variety of antisocial behaviors, higher rates of severe antisocial behavior (such as aggressive behavior), and more likelihood that the antisocial behavior will continue into adulthood (Moffitt 1993).

Recent research using longitudinal studies has also confirmed that LCP and AL delinquents can be differentiated on the basis of a number of social factors, including psychosocial risk composites comprising factors such as maternal depression, maternal life stress, low socioeconomic status (SES), single parenthood, home environment, and parental treatment (Aguilar et al. 2000); frequency of family transitions and disrupted family processes (e.g., Patterson et al. 1998); and inadequate parenting practices (Moffitt and Caspi 2001). In addition, accumulating evidence suggests that personality and temperamental factors—such as early childhood hyperactivity, fighting, and “difficulty” (Moffitt and Caspi 2001); lack of control (Henry et al. 1996); high impulsivity combined with low reward dependence (Tremblay et al. 1994); and lower cognitive ability (Donnellan et al. 2000)—may differentiate early-onset and later-onset delinquency. Overall, this body of research suggests that LCP and AL delinquents represent two distinct groups of individuals. Because behavioral genetic research concentrates on the underlying genetic and environmental influences on variation in human behavior and characteristics, genetically informative studies can be a useful tool for testing whether different groups of individuals have different genetic and environmental etiologies for the “same” phenotype.

In a 1989 review article on the genetics of aggressive behavior, DiLalla and Gottesman, like Moffitt, proposed that typologies based on different life-course patterns of antisocial behavior should yield different estimates of genetic and environmental influences. They predicted that AL antisocial behavior (individuals with such behavior were defined in their article as “transitory antisocials”) should show the strongest influence of environment, particularly environments that are shared across children in the same family, such as community or neighborhood characteristics, family characteristics such as social class or education level, shared parental treatments, and shared peer influences. In contrast, they predicted that LCP antisocial behavior (individuals with such behavior were defined as “continuous antisocials”) should show the strongest genetic influence (i.e., the highest heritabilities).

In this chapter, I have already reviewed a number of findings that may support the idea of genetic heterogeneity of antisocial

behavior. The fact that longitudinal and quasi-longitudinal studies strongly indicate that genetic factors influence the continuity of antisocial behavior across childhood, adolescence, and adulthood may suggest that life-course patterns of antisocial behavior are genetically influenced. Likewise, the repeated findings of higher heritability for adult versus adolescent antisocial behavior might indicate that persistent patterns of antisocial behavior are more strongly influenced by genetic factors than are transient patterns of behavior. Finally, the increasing evidence for a higher heritability of aggressive versus nonaggressive antisocial behavior may also be taken as suggestive of a higher heritability among individuals with LCP patterns of antisocial behavior, who typically show more aggressive patterns of behavior than do individuals with AL patterns of antisocial behavior.

Nevertheless, although these studies offer indirect support for the hypothesis of differential genetic and environmental influence on different life-course patterns of antisocial behavior, they do not test this hypothesis directly. Predictions pertaining to different genetic and environmental etiologies of LCP and AL individuals require some type of *person-centered analyses* that focus on group differences in the relative influence of genetic and environmental factors on individual differences in antisocial behavior. To date, there are two studies that have revealed higher heritability of antisocial behavior among individuals with LCP patterns of antisocial behavior. Using a sample of 147 twin boys, ages 10 to 12 years, Taylor et al. (2000) tested the hypothesis that genetic influence on antisocial behavior would be greatest among "early starters" than among "late starters" or nondelinquents. They found higher rates of antisocial behavior among identical (i.e., monozygotic, or MZ) co-twins of early starters compared with fraternal (i.e., dizygotic, or DZ) co-twins of early starters, indicating genetic influence on antisocial behavior among early starters. In contrast, rates of antisocial behavior among co-twins of late starters were similar for MZ and DZ co-twins, indicating little or no genetic influence. Results were also replicated in an analysis of probandwise concordance rates, supporting the hypothesis of greater genetic influence on early-onset delinquency than on late-onset delinquency, and greater shared environmental influence on late-onset delinquency.

This first study focused on earlier age at onset as a potential indicator of the LCP pattern of antisocial behavior. One limitation of this study, however, is that the average age of the sample was quite young (mean age=11 years), so that subjects were not past the "risk-period" for normative adolescent delinquency, which typically peaks between ages 14 and 16. The second study, therefore, used data from adult twins from the Virginia Twin Registry to investigate whether the heritability of adolescent conduct-disordered behavior would be higher among individuals who *persisted* in their antisocial behavior during adulthood than among individuals who were not antisocial during adulthood (K.C. Jacobson, M. Neale, C.A. Prescott, et al., manuscript under review, 2005). Results of this study (Figure 6-3) confirmed the hypothesis that the heritability of adolescent conduct-disordered behavior was higher among individuals who were antisocial during adulthood as well, compared with individuals who were not antisocial during adulthood; conversely, shared environmental influences were significant only in the non-antisocial adult group. To the extent that persistence of antisocial behavior into adulthood represents a LCP pattern of behavior, these results are consistent with the hypothesis that heritabilities of LCP patterns of behavior are higher than those for AL antisocial behavior.

One important implication of the results from these studies is that the aforementioned conclusions concerning age-related changes in genetic influence on antisocial behavior may be overstated. In previous reviews and meta-analyses (e.g., Miles and Carey 1997), the aggregate heritability of antisocial behavior was estimated at approximately .20 among adolescents and .40-.50 among adults, suggesting modest genetic influence on antisocial behavior in adolescence and moderate genetic influence on antisocial behavior among adults. Results from the Jacobson et al. study (K.C. Jacobson et al., manuscript under review, 2005) indicate that moderately high levels of heritability for adolescent antisocial behavior do exist (.40), but only among specific subgroups of the population (i.e., individuals with LCP patterns of behavior). Interestingly, the estimate of heritability among the non-antisocial adult group (.20) was identical to the estimate of heritability of adolescent antisocial behavior in the Miles and Carey (1997) meta-analysis.

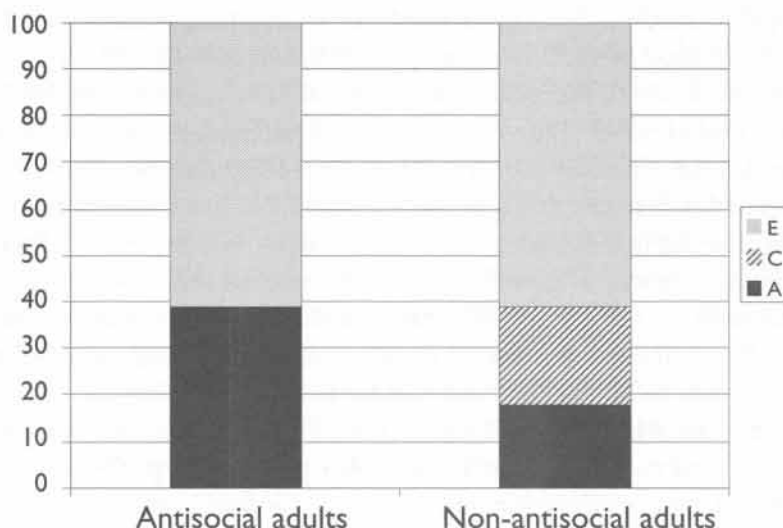


Figure 6-3. Genetic and environmental influences on adolescent conduct disorder.

A=additive genetic influence; C=shared environmental influence; E = non-shared environmental influence.

Moreover, results from these two studies of developmental heterogeneity may help to explain the somewhat counter-intuitive pattern of results from the studies of children, adolescents, and adults—namely, that the heritabilities of antisocial behavior in both child and adult samples are higher than the heritabilities obtained with adolescent samples. To the extent that early childhood problem behavior and continuation of antisocial behavior into adulthood are both manifestations of LCP patterns of behavior, this inverted U-shaped pattern of heritability is to be expected.

Patterns of Comorbidity With Antisocial Behavior

Psychiatrists and psychologists have recognized that comorbidity among psychiatric disorders is the norm, not the exception. In particular, conduct disorder is often comorbid with oppositional defiant disorder, ADHD, and substance use during adolescence, and ASPD is often comorbid with alcohol and drug disorders.

Moreover, there is considerable evidence from family, twin, and adoption studies that comorbidity in externalizing disorders is due to, at least in part, an overlap of common genetic factors (e.g., Eaves et al. 2000; Pickens et al. 1989; Schmitz and Mrazek 2001; Slutske et al. 1998; True et al. 1999). Of particular interest is the question of whether patterns of comorbidity can be used as a method for defining subtypes of antisocial behavior. Using latent class analysis in a sample of adolescent twins, Silberg et al. (1996a) found differences in genetic and environmental influences on different subtypes of antisocial behavior. Of most relevance are the results that variance in "pure" conduct-disordered behavior was largely due to shared environmental influences, whereas variation in comorbid conduct-disordered behavior was largely genetic in origin. These results again support the hypothesis that more extreme, pathological antisocial behavior is more strongly influenced by genetic factors than is "normative" adolescent antisocial behavior.

Gene \times Environment Interactions (G \times E) in the Study of Antisocial Behavior

In this chapter, I have largely focused on the additive roles of genetic and environmental factors in the etiology of delinquent, antisocial, aggressive, and criminal behavior. In other words, many traditional twin and adoption models assume that genetic and environmental factors have unique and independent effects on variation in behavior. However, there is a growing consensus that genetic and environmental influences on variation in antisocial behavior may not operate in such a straightforward, linear fashion and that studies that attempt to identify the *independent* effects of genetic and environmental factors may mask the true complexity of organism-environment *interaction*.

Promising directions for research on aggressive and criminal behavior can be found in models that emphasize the *joint* influences of both environment and biology. Examples of such models are the biosocial perspective (e.g., Raine et al. 1997a) and the diathesis stress model currently used in behavioral genetic research on psychopathology (including aggressive behaviors) (e.g., Cado-

ret et al. 1995; Kendler and Eaves 1986). What is particularly novel about these approaches is that in addition to acknowledging that biology, genes, and environments may contribute independently to development of criminality, both models emphasize the point that these factors may also interact with one another to influence behavior. In particular, it is thought that a genetic "liability" toward aggressive and criminal behavior may be expressed only in a given environment—most often a harsh rearing environment.

For example, Raine et al. (1997b) reported that birth complications interacted with early maternal rejection to predict adult violence. Specifically, adult violent offenders were more likely to have experienced both birth complications and early maternal rejection than just one of these risk factors. The interactive effect of birth complications and early maternal rejection was especially strong in predicting the onset of violence before age 18 (Raine et al. 1997b). Interestingly, the authors found that this interaction effect did not generalize to nonviolent criminal offenses. Similar results were reported in a large birth cohort sample from Sweden (Hodgins et al. 2001).

Recently, studies have examined community- and family-level risk factors as potential moderators of biological or genetic indices of vulnerability to antisocial behavior. Results from the Pittsburgh Youth Study found that the relationship between impulsivity and juvenile offending was stronger for boys in poorer neighborhood (Lynam et al. 2000). In addition, poorer environments were not associated with higher levels of juvenile offending among nonimpulsive boys, suggesting that environmental risk has a significant effect on offending only among individuals with potentially predisposing personality characteristics. What is particularly impressive about this study is that the initial, cross-sectional results from 13-year-old males were replicated at age 17 with a longitudinal design.

Using data from male American veterans, Dabbs and Morris (1990) reported that higher levels of testosterone predicted adult deviance and retrospective reports of childhood delinquency only among males from a low SES background. Among adolescents from higher SES backgrounds, testosterone was not related to childhood or adult deviance. Likewise, a different study using

the same sample found an interaction effect between testosterone levels and a measure of social integration (defined by factors such as educational attainment, organizational ties, job stability, and marriage) for adult deviance (Booth and Osgood 1993). Specifically, the difference in adult deviance associated with low, medium, and high levels of testosterone was greatest when participants reported low levels of social integration. When social integration was high, there was virtually no relationship between level of testosterone and adult deviance.

There is also evidence from behavioral genetic studies for genetic \times environmental interactions ($G \times E$) in the development of antisocial behavior. For example, Cadoret and colleagues found evidence for an "environmental trigger" in their Iowa adoption study of aggressivity and conduct disorders (Cadoret et al. 1995, 1997). In this study, biological risk, defined as having a biological parent with ASPD, interacted with adverse adoptive home environments (defined by factors such as psychopathology, drug abuse, and divorce or separation in the adoptive family) to predict diagnoses of child and adolescent aggressivity and conduct disorder. For example, Figure 6-4 shows the relationship between number of adverse home environmental factors and number of adolescent aggressivity symptoms for adoptees with and without a biological parent with ASPD. Among adoptees without a biological parent with ASPD, there was no relationship between number of adverse adoptive home environment factors and number of aggressivity symptoms. In contrast, among adoptees with a biological parent diagnosed with ASP, the number of aggressivity symptoms increased with the number of adverse adoptive home environmental factors. Cadoret et al. (1997) also reported that specific environmental conditions, such as conflict with the adoptive mother and father, interacted with biological predisposition to predict adolescent aggressive behavior.

Finally, one of the first published studies of *measured* $G \times E$ for a complex behavioral phenotype focused on the interaction between a genotype that codes for monoamine oxidase-A (MAO-A) activity and the environmental risk of child maltreatment as a possible explanation for why some individuals from abusive homes do not grow up to be violent themselves (Caspi et al. 2002).

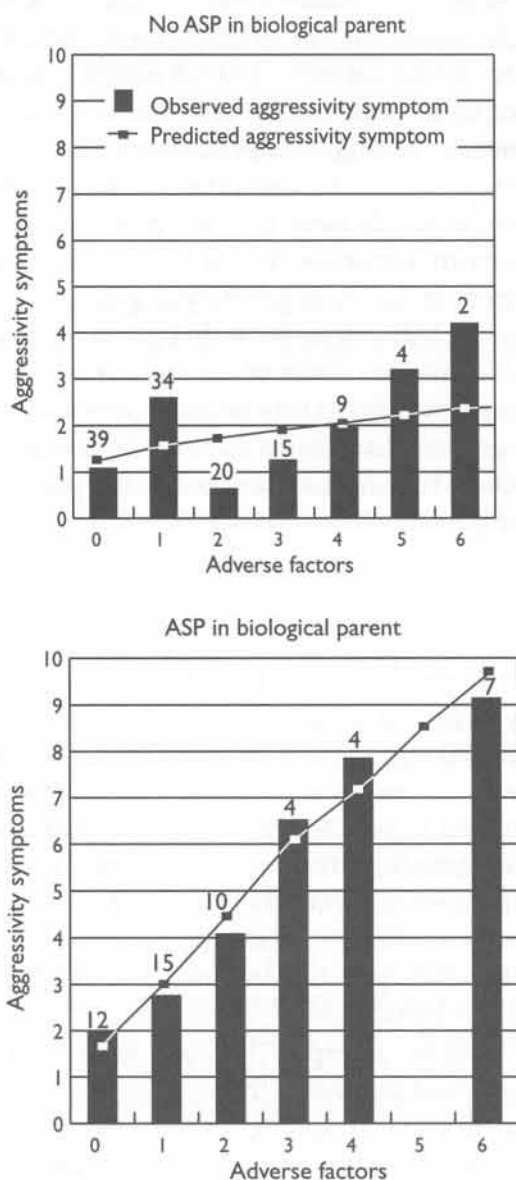


Figure 6-4. Interaction between biological risk and adverse environmental experiences.

Numbers over bars indicate observations. ASP=antisocial personality.

Source. Reprinted from Cadoret RJ, Leve LD, Devor E: "Genetics of Aggressive and Violent Behavior." *Psychiatric Clinics of North America* 20:301-322, 1997. Used with permission.

adverse environmental experiences may trigger latent genetic liability to antisocial behavior. This hypothesis has important implications for treating individuals who exhibit antisocial behavior, as it suggests that modifications to the environment may be an effective means of suppressing these latent liabilities.

References

- Achenbach TM, Edelbrock CS: The Child Behavior Profile, II: boys aged 12–16 and girls aged 6–11 and 12–16. *J Consult Clin Psychol* 47:223–233, 1979
- Aguilar B, Sroufe LA, Egeland B, et al: Distinguishing the early-onset/persistent and adolescence-onset antisocial behavior types: from birth to 16 years. *Dev Psychopathol* 12:109–132, 2000
- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, Revised. Washington, DC, American Psychiatric Association, 1987
- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition. Washington, DC, American Psychiatric Association, 1994
- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000
- Arsenault L, Moffitt TE, Caspi A, et al: Strong genetic effects on cross-situational antisocial behaviour among 5-year-old children according to mothers, teachers, examiner-observers, and twins' self-reports. *J Child Psychol Psychiatry* 44:832–848, 2003
- Bartels M, van den Oord EJCG, Hudziak JJ, et al: Genetic and environmental mechanisms underlying stability and change in problem behaviors at ages 3, 7, 10, and 12. *Dev Psychol* 40:852–867, 2004
- Booth A, Osgood DW: The influence of testosterone on deviance in adulthood: assessing and explaining the relationship. *Criminology* 31:93–117, 1993
- Brennan PA, Mednick SA: Genetic perspectives on crime. *Acta Psychiatrica Scand Suppl* 370:19–26, 1993
- Cadoret RJ: Psychopathology in adopted-away offspring of biologic parents with antisocial behavior. *Arch Gen Psychiatry* 35:176–184, 1978
- Cadoret RJ, Yates WR, Troughton E, et al: Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. *Arch Gen Psychiatry* 52:916–924, 1995

- Cadoret RJ, Leve LD, Devor E: Genetics of aggressive and violent behavior. *Psychiatr Clin North Am* 20:301-322, 1997
- Caspi A, McClay J, Moffitt TE, et al: Role of genotype in the cycle of violence in maltreated children. *Science* 297:851-854, 2002
- Cloninger CR, Gottesman II: Genetic and environmental factors in antisocial behavior disorder, in *The Causes of Crime: New Biological Approaches*. Edited by Mednick SA, Moffitt TE, Stack SA. Cambridge, England, Cambridge University Press, 1997, pp 92-109
- Costello EJ, Edelbrock CS, Costello AJ: Validity of the NIMH Diagnostic Interview Schedule for Children: a comparison between psychiatric and pediatric referrals. *J Abnorm Child Psychol* 13:579-595, 1985
- Dabbs JM Jr, Morris R: Testosterone, social class, and antisocial behavior in a sample of 4,462 men. *Psychol Sci* 1:209-211, 1990
- Deater-Deckard K, Plomin R: An adoption study of teacher and parent reports of externalizing behavior problems in middle childhood. *Child Dev* 70:144-154, 1999
- DiLalla LF, Gottesman II: Heterogeneity of causes for delinquency and criminality: lifespan perspectives. *Dev Psychopathol* 1:339-349, 1989
- Donnellan MB, Ge X, Wenk E: Cognitive abilities in adolescent-limited and life-course-persistent criminal offenders. *J Abnorm Psychol* 109:396-402, 2000
- Eaves LJ, Silberg JL, Meyer JM, et al: Genetics and developmental psychopathology, 2: the main effects of genes and environment on behavioral problems in the Virginia Twin Study of Adolescent Behavioral Development. *J Child Psychol Psychiatry* 38:965-980, 1997
- Eaves LJ, Rutter M, Silberg JL, et al: Genetic and environmental causes of covariation in interview assessments of disruptive behavior in child and adolescent twins. *Behav Genet* 30:321-334, 2000
- Edelbrock CS, Costello AM, Dulcan MK, et al: Parent-child agreement on child psychiatric symptoms assessed via structured interview. *J Child Psychol Psychiatry* 27:181-190, 1986
- Edelbrock CS, Rende R, Plomin R, et al: A twin study of competence and problem behavior in childhood and early adolescence. *J Child Psychol Psychiatry* 56:775-785, 1995
- Eley TC, Lichtenstein P, Stevenson J: Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: results from two twin studies. *Child Dev* 70:155-168, 1999
- Eley TC, Lichtenstein P, Moffitt TE: A longitudinal behavioral genetic analysis of the etiology of aggressive and nonaggressive antisocial behavior. *Dev Psychopathol* 15:383-402, 2003

- Faraone SV, Biederman J, Monuteaux MC: Attention-deficit disorder and conduct disorder in girls: evidence for a familial subtype. *Biol Psychiatry* 48:21-29, 2000
- Farrington DP: The Twelfth Jack Tizard Memorial Lecture: The development of offending and antisocial behaviour from childhood: key findings from the Cambridge Study in Delinquent Development. *J Child Psychol Psychiatry* 36:929-964, 1995
- Foley DL, Eaves LJ, Wormley B, et al: Childhood adversity, monoamine oxidase A, genotype, and risk for conduct disorder. *Arch Gen Psychiatry* 61:738-741, 2004
- Ghodsian-Carpey J, Baker LA: Genetic and environmental influences on aggression in 4- to 7-year-old twins. *Aggress Behav* 13:173-186, 1987
- Gjone H, Stevenson J: A longitudinal twin study of temperament and behavior problems: common genetic or environmental influences? *J Am Acad Child Adolesc Psychiatry* 36:1448-1456, 1997
- Henry B, Caspi A, Moffitt T, et al: Temperamental and familial predictors of violent and nonviolent criminal convictions: age 3 to age 18. *Dev Psychol* 32:614-623, 1996
- Hodgins S, Kratzer L, McNeil TF: Obstetric complications, parenting, and risk of criminal behavior. *Arch Gen Psychiatry* 58:746-752, 2001
- Jacobson KC, Prescott CA, Kendler KS: Sex differences in genetic and environmental influences on antisocial behavior from childhood to adulthood. *Dev Psychopathol* 14:395-416, 2002
- Kendler KS, Eaves LJ: Models for the joint effect of genotype and environment on liability to psychiatric illness. *Am J Psychiatry* 143:279-289, 1986
- Loeber R, Hay D: Key issues in development of aggression and violence from childhood to early adulthood. *Ann Rev Psychol* 48:371-410, 1991
- Loeber R, Green SM, Lahey BB, et al: Optimal informants on childhood disruptive behaviors. *Dev Psychopathol* 1:317-337, 1989
- Lynam DR: Pursuing the psychopath: capturing the fledgling psychopath in a nomological net. *J Abnorm Psychol* 106:425-438, 1997
- Lynam DR, Caspi A, Moffitt TE, et al: The interaction between impulsivity and neighborhood context on offending: the effects of impulsivity are stronger in poorer neighborhoods. *J Abnorm Psychol* 109:563-574, 2000
- Lyons MJ, True WR, Eisen SA, et al: Differential heritability of adult and juvenile antisocial traits. *Arch Gen Psychiatry* 52:906-915, 1995
- Mannuzza S, Klein RG, Konig PH, et al: Hyperactive boys almost grown up, IV: criminality and its relationship to psychiatric status. *Arch Gen Psychiatry* 46:1073-1079, 1989

- Mednick SA, Gabrielli WF Jr, Hutchings B: Genetic influences in criminal convictions: evidence from an adoption cohort. *Science* 224:891-894, 1984
- Miles DR, Carey G: Genetic and environmental architecture of human aggression. *J Pers Soc Psychol* 72:207-217, 1997
- Moffitt T: Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychol Rev* 100:674-701, 1993
- Moffitt T, Caspi A: Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Dev Psychopathol* 13:355-375, 2001
- Neiderhiser JM, Reiss D, Hetherington EM: Genetically informative designs for distinguishing pathways during adolescence: responsible and antisocial behavior. *Dev Psychopathol* 8:779-791, 1996
- O'Connor TG, Neiderhiser JM, Reiss D, et al: Genetic contributions to continuity, change, and co-occurrence of antisocial and depressive symptoms in adolescence. *J Child Psychol Psychiatry* 39:323-336, 1998
- Patterson GR, Forgatch MS, Yoerger KL, et al: Variables that initiate and maintain an early-onset trajectory for juvenile offending. *Dev Psychopathol* 10:531-547, 1998
- Pickens RW, Svikis DS, McGue M, et al: Common genetic mechanisms in alcohol drug and mental disorder comorbidity. *Drug Alcohol Depend* 39:129-138, 1989
- Plomin R: *Development, Genetics, and Psychology*. Hillsdale, NJ, Erlbaum, 1986
- Raine A, Brennan P, Farrington DP: Biosocial bases of violence: conceptual and theoretical issues, in *Biosocial Bases of Violence*. Edited by Raine A, Brennan P, Farrington DP, et al. New York, Plenum, 1997a, pp 1-21
- Raine A, Brennan P, 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* 154:1265-1271, 1997b
- Rhee SH, Waldman ID: Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychol Bull* 128:490-529, 2002
- Robins LN: Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. *Psychol Med* 8:611-622, 1978
- Rowe DC: A biometrical analysis of perceptions of family environment: a study of twin and singleton sibling kinships. *Child Dev* 54:416-423, 1983

- Satterfield JH, Hoppe CM, Schell AM: A prospective study of delinquency in 110 adolescent boys with attention deficit disorder and 88 normal adolescent boys. *Am J Psychiatry* 139:795-798, 1982
- Schmitz S, Mrazek DA: Genetic and environmental influences on the associations between attention problems and other problem behaviors. *Twin Research* 4:453-458, 2001
- Schmitz S, Cherny SS, Fulker DW, et al: Genetic and environmental influences on early childhood behavior. *Behav Genet* 24:25-34, 1994
- Silberg JL, Meyer J, Pickles A, et al: Heterogeneity among juvenile antisocial behaviours: findings from the Virginia Twin Study of Adolescent Behavioral Development, in *Genetics of Criminal and Antisocial Behaviour* (Ciba Foundation Symposium 194). Chichester, England, Wiley, 1996a, pp 76-92
- Silberg J, Rutter M, Meyer J, et al: Genetic and environmental influences on the covariation between hyperactivity and conduct disturbance in juvenile twins. *J Child Psychol Psychiatry* 37:803-816, 1996b
- Simonoff E, Pickles A, Hewitt J, et al: Multiple raters of disruptive child behavior: using a genetic strategy to examine shared views and bias. *Behav Genet* 25:311-326, 1995
- Simonoff E, Pickles A, Meyer J, et al: Genetic and environmental influences on subtypes of conduct disorder behavior in boys. *J Abnorm Child Psychol* 26:495-509, 1998
- Simonoff E, Eleander J, Holmshaw J, et al: Predictors of antisocial personality. *Br J Psychol* 184:118-127, 2004
- Slutske WS: The genetics of antisocial behavior. *Current Psychiatry Reports* 3:158-162, 2001
- Slutske WS, Heath AC, Dinwiddie SH, et al: Modeling genetic and environmental influences in the etiology of conduct disorder: a study of 2,682 adult twin pairs. *J Abnorm Psychol* 106:266-279, 1997
- Slutske WS, Heath AC, Dinwiddie SH, et al: Common genetic risk factors for conduct disorder and alcohol dependence. *J Abnorm Psychol* 107:363-374, 1998
- Stattin H, Magnusson D: The role of early aggressive behavior in the frequency, seriousness, and types of later crimes. *J Consult Clin Psychol* 57:710-718, 1989
- Taylor J, Iacono WG, McGue M: Evidence for a genetic etiology of early-onset delinquency. *J Abnorm Psychol* 109:634-643, 2000
- Thapar A, McGuffin P: A twin study of antisocial and neurotic symptoms in childhood. *Psychol Med* 26:1111-1118, 1996

- Tremblay RE, Pihl RO, Vitaro F, et al: Predicting early onset of male antisocial behavior from preschool behavior. *Arch Gen Psychiatry* 51: 732-739, 1994
- True WR, Heath AC, Scherrer JF, et al: Interrelationship of genetic and environmental influences on conduct disorder and alcohol and marijuana dependence symptoms. *Am J Med Genet* 88:391-397, 1999
- van den Oord EJC, Rowe DC: Continuity and change in children's social maladjustment: a developmental behavior genetic study. *Dev Psychol* 33:319-322, 1997
- van den Oord EJ, Velhurst FC, Boomsma DI: A genetic study of maternal and paternal ratings of problem behaviors in 3-year-old twins. *J Abnorm Psychol* 105:349-357, 1996
- West DJ, Farrington DP: *Who Becomes Delinquent? Second Report of the Cambridge Study in Delinquent Development*. Oxford, England, Crane, Russak, 1973