CHAPTER 4

Evidence from Behavioral Genetics for Environmental Contributions to Antisocial Conduct

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Despite assiduous efforts to eliminate it, antisocial behavior is still a problem. Approximately 20% of people in the developed world experience victimization by perpetrators of violent and nonviolent illegal behavior each year (U.S. Bureau of Justice Statistics, 2002; World Health Organization, 2002). Behavioral science needs to achieve a more complete understanding of the causes of antisocial behavior to provide an evidence base for effectively controlling and preventing antisocial behavior. A new wave of intervention research in the last decade has demonstrated clear success for a number of programs designed to prevent antisocial behavior (www.preventingcrime.org; Heinrich, Brown & Aber, 1999; Sherman et al., 1999; Weissberg, Kumpfer, & Seligman, 2003). Nevertheless, the reduction in antisocial behavior brought about by even the best prevention programs is, on average, modest (Dodge, 2003; Wasserman & Miller, 1998; Olds et al., 1998; Heinrich et al., 1999; Wandersman & Florin, 2003; Wilson, Gottfredson, & Najaka, 2001). The best-designed intervention programs reduce serious juvenile offenders' recidivism only by about 12% (Lipsey & Wilson, 1998). This modest success of interventions that were theory-driven, well designed, and amply funded sends a clear message that we do not yet understand the causes of antisocial behavior well enough to prevent it.

Simultaneous with the new wave of research evaluating interventions is a wave of research pointing to the concentration of antisocial behavior in families. In the 1970s, the astounding discovery that fewer than 10% of individuals perpetrate more than 50% of crimes (Wolfgang, Figlio, & Sellin, 1972) prompted researchers to investigate individual

career criminals (Blumstein & Cohen, 1987) and examine the childhood origins of such persistent reoffenders (Moffitt, 1993). This research constructed the evidence base supporting the new wave of preventive intervention trials (Yoshikawa, 1994). Recently journalists have drawn public attention to certain families that across several generations seem to contain far more than their share of criminal family members (Butterfield, 1996, 2002). This familial concentration of crime has been confirmed as a characteristic of the general population (Farrington, Barnes, & Lambert, 1996; Farrington, Jolliffe, Loeber, Stouthamer-Loeber, & Kalb, 2001; Rowe, & Farrington, 1997). In general, fewer than 10% of the families in any community account for more than 50% of that community's criminal offenses. The family concentration of antisocial behavior could be explained by a genetic influence on antisocial behavior, but it could just as easily be explained by nongenetic social transmission of antisocial behavior within families. Again, causation is not well understood. Studies that cannot disentangle genetic and environmental influences cannot help.

ANTISOCIAL BEHAVIOR RESEARCH IS STUCK IN THE RISK-FACTOR STAGE

Influential reviewers have concluded that the study of antisocial behavior has been stuck in the "risk-factor" stage (Farrington, 1988, 2003; Hinshaw, 2002; Rutter, 2003a, 2003b) because so few studies have used designs that are able to document causality (Rutter, Pickles, Murray, & Eaves, 2001). A variable is called a risk factor if it has a documented predictive relation with antisocial outcomes, whether or not the association is causal. The causal status of most risk factors is unknown; we know what statistically predicts psychopathology outcomes but not how or why (Kraemer, 2003; Kraemer et al., 1997). There are consequences to the field's failure to push beyond the risk factor stage to achieve an understanding of causal processes. Valuable resources have been wasted because intervention programs have proceeded on the basis of risk factors, without sufficient research to understand causal processes.

A central barrier to interpreting an association between an alleged environmental risk factor and antisocial outcome as a cause–effect association is, of course, the old bugbear that correlation is not causation. Some unknown third variable may account for the association, and that third variable may well be heritable. During the 1990s, the assumption that "nurture" influences behavior came under fire. Traditional socialization studies of antisocial behavior, which could not separate environmental influences from their correlated genes, were challenged by four important empirical discoveries: (1) ostensible environmental measures are influenced by genetic factors (Plomin & Bergeman, 1991); (2) parents' heritable traits influence the environments they provide for their children (Kendler, 1996; Plomin, 1994); (3) people's genes influence the environments they encounter (Kendler, 1996; Plomin, DeFries, & Loehlin, 1977); and (4) environmental influences did not seem to account for the similarity among persons growing up in the same family (Rowe, 1994). It was said that although non-behavioral-genetic studies might show that certain rearing experiences predict young people's antisocial outcomes, theories of causation based on findings from such designs were guilty of a fundamental logical error: mistaking correlation for causation (Scarr, 1992). These challenges culminated in admonishments that so far the evidence for genetic influences outweighed the evidence for environmental influences within the family (Harris, 1998; Rowe, 1994). Many social

scientists responded to this claim, reasserting evidence for environmental influences (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000; Reid, Patterson, & Snyder, 2002; Vandell, 2000). However, the reason there is all this controversy about the importance of the family environment in the first place is that the evidence base was not decisive enough to compel both camps. The best way forward to resolve the debate is to use research designs that can provide leverage to test environmental causation.

Ordinary studies cannot test whether a risk factor is causal, and it would be unethical to assign children to experimental conditions expected to induce aggression. Fortunately, researchers can use three other methods for testing causation: natural-experiment studies of within-individual change (Cicchetti, 2003; Costello, Compton, Keeler, & Angold, 2003), treatment experiments (Howe, Reiss, & Yuh, 2002), and the focus of this review: behavioral-genetic designs (Moffitt, 2005). None of the three alone can provide decisive proof of causation, but if all supply corroborative evidence by ruling out alternative noncausal explanations about a risk factor, then a strong case for causation can be made.

TESTING HYPOTHESES ABOUT ENVIRONMENTAL CAUSATION

Inference from Different Types of Behavioral-Genetic Designs

Antisocial behavior has been studied in twins reared together, adoptees, and twins reared apart. Behavioral-genetics research is not limited to exotic samples; researchers also examine ordinary families whose members vary in genetic relatedness (e.g., full siblings, half-siblings, step-siblings, cousins, and unrelated children reared in the same family) (Rowe, Almeida, & Jacobson, 1999). This variety of research designs offers a special advantage for inference, because comparing their estimates tells us that the environmental effect sizes for antisocial behavior are robust across different designs; they are not biased by the limitations and flaws peculiar to one design.

A number of potential flaws are unique to adoption studies. First, adoption agencies could attempt to maximize similarity between the adoptee's biological and adoptive families to increase the child's chance of fitting in with the new family ("selective placement"). Relatedly, biological mothers who intend to give their baby away may neglect prenatal care and continue to abuse substances during pregnancy, and many unwanted babies experience institutionalization before they are adopted. If adoptive homes, prenatal care, and institutional care were selectively worse for the babies given up by antisocial biological mothers, this could bias estimates of heritability upward and estimates of environment effects downward, by misattributing the criminogenic influences of these three unmeasured nongenetic factors to a criminogenic influence of genes (Mednick, Moffitt, Gabrielli, & Hutchings, 1986). Second, both adoptees and twins reared apart are likely to be reared in home environments that are unusually good for children because adoptive parents are carefully screened. Adoption breaks up the association between genetic risk and environmental risk naturally occurring in ordinary families by removing genetically at-risk children from damaging homes and placing them in salutary homes. As a result, interactions between environmental adversity and genetic vulnerability that exacerbate behavioral problems in ordinary children (and twins) are uncommon among adoptees (Stoolmiller, 1999). The restricted range of rearing environments resulting from screening of adoptive parents could suppress estimates of environmental effects and thus bias heritability estimates upward (Fergusson, Lynskey, & Horwood, 1995; Stoolmiller,

1999). However, this flaw of adoption studies is offset by studies of national twin registers (e.g., Cloninger & Gottesman, 1987) or stratified high-risk twin samples (e.g., Moffitt & E-risk Study Team, 2002), because such sampling frames represent the complete population range of environmental and genetic backgrounds.

Studies of twins avoid the potential flaws of adoption studies, but they suffer several potential flaws of their own. First, the logic of the twin design assumes that all the greater similarity between monozygotic (MZ) compared to dizygotic (DZ) twins can safely be ascribed to MZ twins' greater genetic similarity. This "equal environments assumption" requires that MZ twins are not treated more alike than DZ twins on the causes of antisocial behavior (Kendler, Neale, Kessler, Heath, & Eaves, 1994). Because MZ twins look identical, they might be treated more similarly than DZ twins in some way that promotes antisocial behavior, and as a result, estimates of heritability from studies of twins reared together could be biased upward, and estimates of environmental effects could be biased downward, relative to the correct population value (DiLalla, 2002). However, studies of adoptees do not suffer this flaw, and neither do studies of twins reared apart, because MZ twins reared apart do not share environments (unless their genetically influenced behaviors evoke similar reactions from caregivers in their separate rearing environments, which is a genetic effect). Second, in studies of twins, MZ twins differ more than DZ twins in prenatal factors affecting intrauterine growth; for example, MZ twins sharing the same chorion appear to suffer more fetal competition for nutrients. These intrauterine factors also violate the assumption that environments are equal for MZ and DZ twins, but intrauterine differences tend to make MZ twins less alike than their genotypes and thus would bias heritability estimates downward and environmental effects upward (Rutter, 2002). Third, genomic factors that make some MZ twin pairs' genotypes less than perfectly identical (such as random inactivation of genes on one of each girl's two X chromosomes; Jorgensen et al., 1992) could in theory affect twin-study estimates, but so far no evidence shows that these processes influence behavior. Fourth, parental assortative mating can bias heritability estimates. Coupled partners are known to share similarly high or low levels of antisocial behaviors (Galbaud du Fort, Boothroyd, Bland, Newman, & Kakuma, 2002; Krueger, Moffitt, Caspi, Bleske, & Silva, 1998). When parents of twins mate for similarity, it should increase the genetic similarity of DZ twins, but MZ twins' genetic similarity cannot increase beyond its original 100%, and as a result heritability estimates will be biased downward and environmental estimates upward, relative to the correct population value. The implication of biological-parent assortative mating for adoption studies is the opposite; biological-parent similarity for antisocial behaviors would bias adoptees' heritability upward relative to the correct population value (because adoptee/biological-parent correlations would represent a double dose of parental genes). Fifth, twin studies using adult reports to measure behavior sometimes suffer from rater artifacts; for example, adults may mix up or conflate the behavior of MZ twins and they may exaggerate differences between DZ twins. Such a rater artifact does not afflict adoption studies (nor twin studies using the twins' self-reports, as twins do not confuse themselves).

In any case, comparisons between designs have revealed that studies of twins reared together yield estimates that are more similar than different to the estimates from studies of twins reared apart or of adoptees (Rhee & Waldman, 2002). On the one hand, this is because any bias arising from factors such as selective adoptee placement, violations of the equal-environment assumption, intrauterine twin differences, or assortative mating, is only very small (Miles & Carey, 1997; Rutter, 2002). On the other hand, these factors

bias estimates upward as often as they bias them downward, canceling each other out. The bottom line is that it is important for tests of environmental risk to exploit a variety of behavioral-genetics designs, as well as experimental designs and studies of within-individual change.

Behavioral-Genetic Studies of Parenting Effects on Children's Aggression

To illustrate how behavioral-genetic designs are helping to move the study of antisocial behaviors from the risk factor stage to causal understanding, we next review research investigating one risk factor, parents' "bad parenting" of their children, and one antisocial outcome, "children's aggression." Of course, behavioral-genetics studies address other socializing agents (e.g., siblings, peers, teachers, communities, and historical periods) and other behavioral outcomes (e.g. depression, anxiety, prosocial behaviors, cognitive abilities, and personality) but we focus on studies of parenting and aggression as our example, because that is the most developed body of literature.

We have construed bad parenting broadly; this review includes risk factors from mothers' smoking heavily during pregnancy to inconsistent or unskilled discipline to frank child neglect and abuse. The outcome, "children's physical aggression," includes hitting, fighting, bullying, cruelty, and so forth. It is already known that "bad parenting" statistically predicts children's aggression, and bad parenting plays a central causal role in leading theories of antisocial behavior (Lahey, Moffitt, & Caspi, 2003; Thornberry, 1996). The aim of the research reviewed here is to determine whether the relation between bad parenting and children's aggression is a true cause–effect relation.

Our research review systematically tackles six questions:

1. Is there evidence that children's aggression cannot be wholly explained by genetic factors, and must have non-genetic environmental causes as well?
2. Do parents' genes influence bad parenting?
3. Does a genetic effect on parents' bad parenting confound a cause-effect interpretation of the association between bad parenting and children's aggression?
4. Does a genetic "child effect" evoke bad parenting to further confound a cause–effect interpretation of the association between bad parenting and children's aggression?
5. After genetic confounds are controlled, does bad parenting have an environmentally mediated causal effect on children's aggression?
6. Does bad parenting interact with genetic risk, such that the effects of bad parenting are even stronger among genetically vulnerable children?

We address each question in a separate section, first describing research designs that can answer each question and then reviewing findings so far. The research designs covered here are not intended to be exhaustive but to illustrate what kinds of studies could be done, using the logic of behavioral-genetic methods.

1. Is Children's Aggression Wholly Accounted for by Genetic Factors, or Does It Have Nongenetic Causes as Well?

More than 100 twin, adoption, and sibling studies have been carried out to answer this question. This work has revealed that genetic causal processes account for only about

half of the population variation in antisocial behavior, thereby unequivocally proving that environmental influences account for the other half. This fact constitutes a remarkable contribution to the understanding of causation (Plomin, 1994). In addition, it is now recognized that the heritability coefficient indexes not only the direct effects of genes but also the effects of interactions between genes and family-wide environments (Purcell 2002; Rutter & Silberg, 2002). In such interactions the effect of an environmental risk may be even larger than previously reported, among the subgroup of individuals having a vulnerable genotype. This is likely to be the case for antisocial behaviors.

One useful feature of behavioral-genetics research designs is that they offer two powerful methods for documenting the importance of environmental effects (Plomin, DeFries, McClearn, & McGuffin, 2001). One of these methods of detecting environmental influence tests whether any of the family members in a study sample are more similar than can be explained by the proportion of genes they share. For instance, MZ twins' genetic similarity is twice that of DZ twins, and therefore, if nothing but genes influenced antisocial behavior, MZ twins' behavior ought to be at least twice as similar as DZ twins'. If not, then something environmental has influenced the twins and enhanced their similarity. For almost all human behavioral traits studied so far, environmental factors shared by family members (variously labeled the "family-wide," "common," or "shared" environment) have not been found to make family members similar. In other words, the estimated influence of shared environment has been found to be almost nil for most human behavioral traits (Rowe, 1994). Antisocial behavior is a marked exception. A comparison of shared-environment effects across 10 psychiatric disorders revealed that such effects were stronger for antisocial personality and conduct disorder than for affective, anxiety, or substance disorders (Kendler, Prescott, Myers, & Neale, 2003). Estimates of shared-environment effects on population variation in antisocial behavior are about 15–20% as reported by meta-analyses and reviews (Miles & Carey, 1997; Rhee & Waldman, 2002). The small size of this shared-environment estimate should not be too surprising, because the twin-study coefficient indexing the shared environment does not include environmental effects involved in gene–environment interactions. We can think of the shared-environment coefficient as the residual effects of shared environments that remain, after controlling for gene–environment interactions. As most human behavior involves nature–nurture interplay, it is remarkable that as much as 20% of the population variation in antisocial behavior can be attributed to direct environmental effects not conditional on genetic vulnerability.

The second method of detecting the presence of environmental influence is to test whether any family members are less similar than expected from the proportion of genes they share (Plomin & Daniels, 1987). For instance, if a pair of MZ twins, despite sharing all their genes, are not perfectly identical in antisocial behavior, this indicates that experience has reduced their behavioral similarity. After estimates of the influences of heritability (50%) and shared family environment (20%) on antisocial behavior are calculated, the remainder of population variation, 30%, is assumed to reflect environmental influences not shared by family members (variously labeled "unique," "person-specific," or "nonshared" experiences). These experiences might include criminogenic experiences unique to the individual and not shared with his or her sibling, such as a head injury, being the unique target of sexual abuse, living with an antisocial spouse, or serving a prison sentence. There are two caveats about estimates of the effect of nonshared environments. First, measurement error inflates these estimates because random mistakes in measuring behavior will result in scores that look different for twins in an MZ pair, and it is not easy

to differentiate such faux MZ differences from true MZ differences caused by the twins' nonshared experiences. The second caveat is that the coefficient for nonshared environmental effects indexes not only the direct effects of nonshared experiences but also the effects of interactions between nonshared environments and genes (Purcell, 2002; Rutter & Silberg, 2002). Thus, some portion of the nonshared environment effect may be attributable to error or genes, and the size of this portion is unknown.

In sum, behavioral-genetics studies have shown that the answer to question 1, "Does children's aggression have any nongenetic causes?," is a definite yes; there is strong evidence that environmental causes must exist.

2. Do Parents' Genes Influence Bad Parenting?

It is important to know the size of the contribution of parents' genotypes to their bad parenting, because if parenting is substantially influenced by parents' genotype, then its correlation with children's aggression cannot be confidently interpreted as a cause–effect relation. But how much do people's genes influence their parenting? Answering this question requires researchers to treat parenting as a phenotype in behavioral-genetics research.

What Research Designs Can Be Used to Answer This Question?

We can study *adoptions* to test if biological parents' bad parenting (of the children they did not give up for adoption) predicts that their adopted-away child will also engage in bad parenting when she becomes a parent. This study would show that bad parenting is genetically transmitted, in the absence of social transmission. However, this study has not been conducted, because of the difficulty of obtaining parenting data from two generations of adults separated by adoption.

We can study *adult MZ twins reared apart* to test whether they are similar in using bad parenting on their children. The Swedish Adoption Twin Study of Aging carried out this design, by asking 50 pairs of adult MZ twins reared apart to report their own parenting styles using the Moos Family Environment Scale (Plomin, McClearn, Pederson, Nesselroade, & Bergeman, 1989). Results indicated that 25% of the variation in parenting was genetically influenced.

We can study *adult twin parents* to ascertain how much variation in their bad parenting is attributable to genetic versus environmental sources. The aforementioned Swedish twin study carried out this design, studying 386 adult twin pairs, and again results indicated that 25% of the variation in the Family Environment Scale was genetically influenced (Plomin et al., 1989). In another study, 1,117 pairs of midlife twin volunteers who had on average reared three children reported their own parenting styles. The heritability estimate for an overall measure of parenting, called care, was 34% (Perusse, Neale, Heath, & Eaves, 1994). A Virginia sample of 262 pairs of adult twin mothers reported their own parenting styles, and the heritability estimates were 21% for "physical discipline," 27% for "limit-setting," and 38% for "warmth" (Kendler, 1996; Wade & Kendler, 2000). An Oregon sample of 186 pairs of adult twin mothers and adoptee mothers reported their own parenting styles, and the heritability estimates ranged from 60% for "positive support" to 24% for "control" (Losoya, Callor, Rowe, & Goldsmith, 1997). These findings were echoed by a study of 236 pairs of adult twin mothers report-

ing their own parenting, in which genetic effects were found for "positivity" and "monitoring" (Towers, Spotts, & Neiderhiser, 2001; Neiderhiser et al., 2004). Finally, a study of 1,034 adult twin mothers found a heritability estimate of more than 50% for self-reported smoking during pregnancy, which is a known prenatal parenting risk factor for children's aggression (D'Onofrio et al., 2003).

What Research Is Needed?

This very small literature is a good beginning, but a number of limitations need to be overcome. First, the studies have relied on the twin design, and twin-design weaknesses ought to be complemented by the strengths of the adoption design (see Deater-Deckard, Fulker, & Plomin, 1999). Second, measurement has relied on parents' self reports, and thus the findings are a mix between genetic influences on actual parenting behavior and genetic influences on self-perception and self-presentation (Kendler, 1996; Plomin, 1994). As a third limitation, studies have tended to focus on mothers and excluded fathers, for the obvious reason that fathers' nonparticipation in research disproportionately characterizes families of aggressive children. However, fathers' antisocial behavior in the home is a central aspect of bad parenting that predicts children's aggression (Jaffee, Moffitt, Caspi, & Taylor, 2003). Fourth, and most serious for our purposes of investigating antisocial behavior, the samples underrepresent families at serious risk, and the parenting measures do not address the most powerful bad-parenting risk factors for children's aggression, such as exposure to domestic violence, child neglect, maternal rejection, and child abuse. These serious forms of bad parenting themselves constitute antisocial acts, and as a result we should anticipate that the influence of parents' genes on them is much stronger than the genetic influences found for parenting styles within the normative range, such as spanking, monitoring, or limit-setting. Because serious bad parenting is antisocial, it is not unreasonable to expect genetic influence on serious bad parenting to resemble genetic influence on other antisocial behaviors (50%).

The answer to question 2, "Do parent's genes influence bad parenting?," seems to be "probably." It may be surprising that so little research has been done on the question of a genetic contribution to bad parenting. The question has been neglected because parenting has not often been viewed by behavioral-genetics researchers as a phenotypical outcome variable. Moreover, developmental researchers who are interested in parenting as an outcome almost never adopt behavioral-genetics research methods. It is quite likely that bad parenting is under some amount of genetic influence because parenting styles are known to be associated with parents' personality traits (Belsky & Barends, 2002; Spinath & O'Connor, 2003) and personality traits are known to be under genetic influence (Plomin & Caspi, 1999). Bad parenting should be treated as a phenotype in future behavioral-genetics research (McGuire, 2003).

3. Does an Effect of Parents' Genes on Bad Parenting Confound a Cause–Effect Interpretation of the Association between Bad Parenting and Children's Aggression?

The technical term for this question is "passive" correlation between genotype and an environmental measure, often abbreviated as "rGE" (Plomin et al., 1977). A passive rGE confound occurs when a child's behavior and the environment his or her parents provide

are correlated because they have the same origins in his parents' genotype (i.e., not because bad parenting itself causes children's aggression).

It is important to note that the mere evidence that bad parenting is under influence of parents' genes (question 2) is not sufficient to conclude that this genetic influence goes on to mediate the connection between bad parenting and children's aggression. Rutter and Silberg (2002) make this point, explaining that genes influence which mothers have low-birthweight babies but babies' birthweights are wholly determined by environmental conditions, not by any genes inherited from their mothers. For this reason it is important to disentangle (1) the genetic origins of bad parenting from (2) the genetic and environmental mechanisms by which bad parenting produces children's aggression.

What Research Designs Can Be Used to Answer This Question?

There are at least four appropriate research designs, but to our knowledge none of them has been carried out. We can study *adoptions* to test if the biological parents' bad parenting predicts the adopted-away children's aggression, even if parent and child never have contact. This study has not been conducted, because of the difficulty of obtaining parenting data from adopted children's biological parents. We can compare correlations between bad parenting and children's aggression in *natural families versus adoptive families*. If the correlation is stronger in natural families (which have both genetic and environmental processes of transmission) than in adoptive families (which have only environmental transmission), then genetic transmission is taking place (Plomin, 1994). However, this design is biased toward finding evidence of an rGE confound, because there is more variation in bad parenting among natural than adoptive families, which could produce larger correlations with children's aggression in natural families (Stoolmiller, 1999). To avoid such bias, we can conduct a study *within adoptive families* to test if rearing parents' bad parenting is more strongly correlated with their natural children's aggression than with their adoptive child's aggression. The within-family design holds constant the variation in bad parenting across natural versus adoptive parent–child pairs but requires a sample of families having both an adopted and a natural child, not too far apart in age. We are not aware of a study that has compared the correlations between bad parenting and natural children's aggression versus adoptive children's aggression. However, a study was conducted of 667 adoptive families, which found adoptive parents' reports of "family functioning" were more strongly correlated with self-reported antisocial behavior in their natural child than their adopted child (McGue, Sharma, & Benson, 1996).

A promising method studies *the families of adult MZ twins who are mothers* to test if MZ aunts' bad parenting predicts their nephews' aggression. In this twin-mothers design, both MZ sisters are genetic mothers to each others' birth children. However, the MZ aunt does not provide the rearing environment for her nieces and nephews; only the children's birth mother is an environmental mother to them. If the MZ aunts' and the MZ mothers' parenting predicts the children's aggression to the same extent, this would be strong evidence of a complete rGE confound. But, if the MZ mother's parenting predicts the children's aggression better than does the MZ aunt's parenting, this would show that bad parenting has an environmental effect. This design offers the capacity to disentangle sources of bad parenting from mechanisms of risk for the children of bad parents, particularly when DZ twin mothers as well as MZ twin mothers are sampled (D'Onofrio et al., 2003; Silberg & Eaves, 2004). This children-of-twins design is newly being applied

to the question of causes of children's aggression by Silberg (2002), but findings were not available at the time of this writing.

The aforementioned methods test the hypothesis that genetic transmission explains the observed association between bad parenting and child aggression by looking for an effect of parenting on behavior over and above genetic influence on behavior. Another method is to *compare the effect size of the association between bad parenting and children's aggression before versus after genetic influences are controlled.* Any shrinkage estimates the extent to which the association is mediated by genetic transmission. In their meta-analysis of studies of differential treatment of siblings, Turkheimer and Waldron (2000, Table 3) showed that the effect sizes for associations between risk factors and behavior outcomes tended to shrink by at least half when genetic confounds were controlled. However, this meta-analysis compared effect sizes across two groups of studies, those with versus without genetic designs, and the groups of studies differed on design features such as sample composition or sample size. Comparisons of the effect sizes for bad parenting predicting children's aggression before and after genetic controls within the same sample would be more informative.

What Research Is Needed?

A close reading of the literature reveals that researchers have neglected two questions: whether genes contribute to bad parenting, and whether genetic transmission confounds environmental interpretations of the link between bad parenting and children's aggression. The field seems to have presupposed affirmative answers to these questions but not to have built a conclusive evidence base. As such, research applying any of the designs described here to parenting is needed. However, a comparison of effect sizes in studies with versus without genetic controls suggests genetic transmission might explain as much as half the connection. The answer to question 3, "Are cause–effect interpretations of the connection between bad parenting and children's aggression confounded by genetic transmission?," seems to be "probably."

4. Does a Genetic "Child Effect" Evoke Bad Parenting to Confound a Cause–Effect Interpretation of the Association between Bad Parenting and Children's Aggression?

The technical term for this question is "evocative" correlation between genotype and an environmental measure, and it is also abbreviated as "rGE" (Plomin et al., 1977). Evocative rGE occurs when a child's behavior and the parenting he receives are correlated because they have the same origins in his own genotype (i.e., not because bad parenting itself causes children's aggression).

What Research Designs Can Be Used to Answer This Question?

A large number of studies has ascertained *twins' recollections of how they were treated by their parents* during childhood, and found that MZ twins' ratings of their parents' childrearing are more similar than DZ twins' ratings, suggesting an influence of childrens' genotype on parents' parenting (Hur & Bouchard, 1995; Rowe, 1983; Kendler, 1996). There is a basic difficulty with this literature, however. Although it seems reasonable to

interpret the findings as evidence for a child effect on bad parenting, studies of twins' self-reports about their parents' treatment of them do not rule out the alternate interpretation of a genetic effect on perceptual bias, according to which MZ twins are more alike than DZ twins in how they interpret their parents' treatment or how they revise their childhood memories (Krueger, Markon, & Bouchard, 2003). Nonetheless, the body of studies is generally interpreted as evidence for genetic child effects on parenting because several other studies have shown genetic child effects using adoption and sibling family designs instead of twins, and by using observational or multi-informant measures of parenting instead of twins' self-reports (Braungart, Plomin, & Fulker, 1992; Deater-Deckard et al., 1999; Neiderhiser et al., 2004; O'Connor, Hetherington, Reiss, & Plomin, 1995, Reiss, Neiderhiser, Hetherington, & Plomin, 2000; Rende, Slomkowski, Stocker, Fulker, & Plomin, 1992). These numerous studies decidedly demonstrated that a genetic child effect on parenting exists, but they did not demonstrate what it is that children do to provoke bad parenting. In other words, these studies did not include children's aggression as a measured variable.

Another research design is to study *adoptions*, to test whether adoptees' aggression predicts their adoptive parents' bad parenting while establishing that the adoptees' aggression has a genetic basis (i.e., that it is predicted by their biological parents' antisocial behavior). Three studies have used this compelling design (Ge et al., 1996; O'Connor, Deater-Deckard, Fulker, Rutter, & Plomin, 1998; Riggins-Caspers, Cadoret, Knutson, & Langbehn, 2003). All three studies reported that adoptees who are at high genetic risk for psychopathology receive more discipline and control from their adoptive parents than adoptees who are at low genetic risk. Furthermore, unlike prior research, the three studies demonstrated that the link from a child's genetic risk to adoptive parent's parenting is mediated by the child's genetically influenced aggressive behavior problems. Individual studies in this threesome were limited by a small sample, or by single-source retrospective data, but as a set the three studies provide robust evidence for a genetically mediated child effect in which the causal arrow runs from children's aggression to parenting.

A third design for testing genetic child effects is to study *twin children*, asking whether twin A's aggression predicts the bad parenting received by twin B, and vice versa. This is an application of bivariate twin modeling. Its basic logic is that if the correlation between twin A's aggression and twin B's experience of bad parenting is higher among MZ pairs than DZ pairs, it would indicate that the same set of genetic influences causes children's aggression and provokes bad parenting. Bad parenting must be measured separately for each twin, so that it can be used as a phenotype, like each twin's aggression. Two studies of several hundred sibling pairs taking part in the study of Nonshared Environment in Adolescent Development (NEAD) have applied variations of this bivariate approach, using multisource measures of adolescents' and parents' behavior. A genetic-child effect accounted for most of the correlation between adolescents' antisocial behavior and parents' negativity assessed cross-sectionally (Pike, McGuire, Hetherington, Reiss, & Plomin, 1996) and longitudinally after accounting for the continuity of adolescent antisocial behavior (Neiderhiser, Reiss, Hetherington, & Plomin, 1999).

It is important to know whether the genetic-child effect for ordinary parenting (as indicated by previous adoption studies and the NEAD study) also applies to extreme forms of bad parenting associated with serious, persistent antisocial behavior. We applied the bivariate modeling approach to this question in our Environmental Risk ("E-risk") longitudinal study of 1,116 British families with young twins (Jaffee, Caspi, Moffitt, Polo-

Tomas, Price, & Taylor, 2004). To do this, the E-risk study incorporated two innovations (Moffitt & Erisk Study Team, 2002). First, it assessed a birth cohort in which one-third of families were selected to oversample families that were at high risk (findings are weighted back to represent the population of British families having babies in the 1990s). Second, the study interviewed mothers about parenting that was beyond normal limits (physical maltreatment: neglectful or abusive care resulting in injury, sexual abuse, registry with child protection services) as well as about parenting in the normative range (frequency of corporal punishment: grabbing, shaking, spanking). Children's genes influenced which children received corporal punishment, explaining 24% of the variation in the cohort, but children's genes were unrelated to becoming a victim of maltreatment. Bivariate twin modeling of the cross-twin, cross-phenotype correlations revealed that children's genes accounted for almost all the correlation between corporal punishment and children's aggression, indicating that most of the observed association between this form of parenting and children's aggression is a genetic child effect. However, children's genes did not account for the correlation between physical maltreatment and children's aggression, indicating that extreme, serious bad parenting causes children's aggression for reasons that are not genetic. Although difficult children can and do provoke their parents to use frequent corporal punishment in the normal range, factors leading to injurious maltreatment lie not within the child but within the family environment or the adult abuser. There are limits to child effects.

What Research Is Needed?

Taken together, the adoption and twin studies reviewed in this section provide evidence to answer question 4: Yes, the observed association between normative parenting and child aggression is in large part a spurious artifact of a third variable that causes both: the child's genotype. A provocative deduction from the research to date is that Scarr (1991) might have been correct when she argued that improving parenting in the normal range of environments will not produce significant changes in children's antisocial psychopathology because the associations between ordinary parenting and child outcome are not causal: "There is no evidence that family environments, except the worst, have any significant effect on the development of conduct disorders, psychopathy, or other common behavior disorders" (Scarr, 1991, p. 403). Scarr (1992) further argued that damaging environmental conditions outside the expected range will have causal influences on children quite apart from genetic influences, and in keeping with this notion, one study showed maltreatment makes children aggressive apart from any influence of their genotypes. This distinction between normative versus extreme forms of parenting has implications for future research. Most of the genetically informative studies to date have assessed parenting using omnibus measures (e.g., "family functioning," "negativism," and "control") because the goal was to ascertain whether or not genetic child effects existed at all. However, parenting intervention programs try to change specific well-defined forms of parental behavior. To inform these interventions, research is needed to query genetic versus environmental mediation of specific features of parenting. Furthermore, the aspects of parenting that correlate with children's aggression are probably quite different in early childhood, later childhood, and adolescence. Genetically informative studies of samples at different ages are needed to inform parenting interventions tailored to developmental stages.

We have looked here at the specific question of whether children's genotype evokes bad parenting, but it is useful to note that the evocative type of rGE is a subset of a larger class referred to as active rGE. Active rGE encompasses at least three different processes, when people's genetically influenced behavior leads them to "(1) create, (2) seek, or (3) otherwise end up in environments that match their genotypes" (Rutter & Silberg, 2002, p. 473). Antisocial behavior can bring about each of these three processes at any point in the life course (Scarr & McCartney, 1983). These active rGE processes are of enormous importance in understanding the continuity of antisocial behavior across the entire life course (Caspi & Moffitt, 1995; Laub & Sampson, 2003). Once genetically influenced behavior has brought a person into contact with an environment, the environment may have unique causal effects of its own, cutting off opportunities to develop alternative prosocial behaviors, promoting the persistence of antisocial behavior, and exacerbating its seriousness (Moffitt, 1993). Research is needed to test for active rGE processes involved in antisocial behavior at developmental stages across the life course.

5. After Both Genetic Confounds Are Controlled, Does Bad Parenting Have Any Environmentally Mediated Effect on Children's Aggression?

The new generation of research designs that can evaluate whether a risk factor has an environmentally mediated effect on children's aggression has three key features. First, the studies must employ a genetically sensitive design to control for the confounding effects of parents' genes or children's genes on putative environmental measures. Second, the genetically informative samples must accurately represent the full range of families' environmental circumstances. Many behavioral-genetics samples suffer substantial biases in recruitment and attrition, inadvertently restricting their range of participating families to primarily the middle class. The third key feature is that designs must employ an actual measure of the construct alleged to have environmental effects on children; in the case here, bad parenting. Traditional behavioral-genetics studies have reported latent environmental variance components (i.e., these studies report statistical inferences derived from the relative similarity of twins) but not direct measures. This has been problematic because even very large twin studies are underpowered to detect environmental influence on twin similarity as a latent variance component, whereas statistical power to detect such influence is increased if a putative environmental variable is measured so its effects can be estimated empirically (Kendler, 1993). In keeping with this, significant effects for a measured variable have been found even despite the presence of a nonsignificant shared-environment variance component (Kendler, Neale, Kessler, Heath, & Eaves, 1992). In this section we abandon the distinction between "shared" and "nonshared" environmental variance components because shared and nonshared effects are not features of a measured environmental risk; one form of bad parenting, such as maltreatment, can exert either shared or nonshared effects, or both (Rutter & Silberg, 2002; Turkheimer & Waldron, 2000).

What Research Designs Can Be Used to Answer This Question?

Four basic behavioral-genetics methods can be used to rule out gene–environment correlation confounds while testing causation by putative environmental risk factors. As

mentioned before, natural experiments and intervention experiments can also assess environmental causation, but here we focus on genetically sensitive designs.

We can study *adoptions* to test if the adoptive parents' bad parenting increases adoptees' aggression, over and above the genetic influence from the biological parents' aggression. The large adoption studies of antisocial behavior that emerged from Scandinavia and the United States in the 1970s and 1980s were primarily cited for their innovation of demonstrating genetic influences; they showed that adoptees' criminal offending was significantly associated with the antisocial behavior of their biological parents, although these parents did not rear the adoptees. However, some of these same studies asked whether adoptees' criminal offending was also associated with the antisocial behavior of the adoptive parents who did rear them (Bohman, Cloninger, Sigverdsson, & von Knorring, 1982; Cadoret, Cain, & Crowe, 1983; Mednick & Christiansen, 1977; vanDusen, Mednick, Gabrielli, & Hutchings, 1983). Rates of antisocial behavior in adoptive parents were extremely low (because of adoption agency screening), and the adoptive-parent effects were very small and often nonsignificant, but these studies constituted the first real empirical attempts to test if bad parental behavior exerts a nongenetic effect on children's aggression.

We can study the *children of adult MZ twin mothers*. As described earlier, in this children-of-twin mothers design the MZ aunt constitutes a genetic mother to the child but not an environmental mother (Silberg & Eaves, 2004). Thus, if an MZ mother–son correlation is larger than its companion MZ aunt–nephew correlation, this provides evidence that environmental mothering influences children, over and above genes. Such research is under way (D'Onofrio et al., 2003; Silberg, 2002).

We can study *twin children* to test if the shared experience of bad parenting makes children more similar on aggression than could be predicted based on their degree of genetic relationship. A basic approach is to conduct ordinary behavior-genetics modeling that apportions genetic versus environmental effects on child behavior (denoted ACE), and then add a measured putative environmental risk factor (denoted M-ACE) to test if the children's shared experience of that risk factor can account for any of the shared environmental variation in their behavioral phenotype. The first twin study to apply this approach to problem behavior reported that living in a deprived neighborhood explained a significant 5% of the shared environmental variation in 2-year-olds' behavior problems (Caspi, Taylor, Moffitt, & Plomin, 2000). Another study applied this approach to examine 5-year-olds' exposure to their mothers' experience of domestic violence (Jaffee, Moffitt, Caspi, Taylor, & Arseneault, 2002). Exposure to domestic violence over the first 5 years of their lives was particularly relevant for children who developed both externalizing and internalizing problems simultaneously; such co-occurring problems are associated with poor prognosis. Domestic violence exposure explained a significant 13.5% of the shared-environment variance in children's comorbid outcome. A third, unpublished study reports that measured parental monitoring accounted for 15% of the shared-environment variance in behavior problems in a large sample of 11- to 12-year-old Finnish twins (described in Dick & Rose, 2002). A caveat about this approach is in order. Inference of environmental causation is compromised if parent and child share genes that simultaneously influence both the measure of parenting and the measure of child aggression.

The basic twin design can be improved on by adding indicators of mothers' and fathers' behavioral phenotype to the usual indicators of twin behavior. This approach,

the "extended twin-family design" (Kendler, 1993), estimates the effect of the putative environmental risk factor on child behavior while controlling for genetic effects on both parents and children. An assumption of the design is that the parental phenotype measures carry genetic information parallel to that in the child phenotype measures. (Although this assumption is seldom fulfilled perfectly it seems not unreasonable for antisocial behavior, which has strong childhood-to-adulthood continuity.) The first twin study to apply this approach assessed antisocial conduct problems among adolescent twins and their parents (Meyer et al., 2000). The measured parenting variables were called marital discord and family adaptability. No effect was found for marital discord, but measured family adaptability accounted for 4% of the variance in adolescents' conduct problems.

A complementary approach to testing whether a risk factor has a causal (vs. noncausal) role in the origins of antisocial behavior has been used by studies that rule out passive rGE through statistical controls for parental antisocial behavior. This approach does not differentiate whether the risk factor is influenced at the genotype versus phenotype level of parental antisocial behavior. However, it does offer the advantage that it can be employed in nontwin samples, if phenotypical data are collected for all family members. In the aforementioned E-risk longitudinal twin study of 1,116 families, we examined the effects of fathers' bad parenting on young children's aggression (Jaffee et al., 2003). Mothers' antisocial behavior was statistically controlled, to make clear that the findings applied specifically to fathers' behavior. As expected from the literature on single mothers, a prosocial father's absence statistically predicted more aggression by his children. But the study revealed a new finding: An antisocial father's *presence* predicted more aggression by his children, and this harmful effect was exacerbated the more years a father lived with the family and the more time each week he spent taking care of the children. Inference of environmental causation was supported because the finding for conventional fathers (less involvement predicts more child aggression) was opposite that for antisocial fathers (more involvement predicts more child aggression), and the latter association held after ruling out passive rGE by statistically controlling for both parents' antisocial histories. Obtaining data from fathers is challenging (Caspi et al., 2001), but because fathers are often a target of social policies, a better evidence base about their parenting is needed.

In another report, the E-risk study evaluated the hypothesis that maternal depression promotes children's aggression (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005). Research has shown that the children of depressed mothers are likely to develop conduct problems. However, it has not been clear that this correlation represents environmental transmission, because women's depression is under genetic influence (Kendler et al., 1992), it often co-occurs with a girlhood history of antisocial conduct, which is also under genetic influence (Moffitt, Caspi, Rutter, & Silva, 2001), and depressed women often mate assortatively with antisocial men (Moffitt, Caspi, Rutter, & Silva, 2001). We controlled for antisocial behavior in the twins' biological father, and for the mothers' own antisocial history. Although the connection between mothers' depression and children's conduct problems decreased after this stringent control for familial liability, it remained statistically significant. It concerned us that depressed women might exaggerate ratings of their children's problem behaviors, but the pattern of findings remained the same when teachers' ratings of child behavior were substituted as the outcome measure. A temporal analysis showed that the effect of maternal depression on children's aggression depended

on the timing of the depression episodes (a type of natural experiment design). If E-risk mothers experienced depression, but only before their children's birth and not after, the children were not unusually aggressive. In contrast, only if mothers suffered depression while rearing their children were the children likely to develop aggression. Finally, the possibility that a child effect (in which children's aggression provoked mothers' depression) explained the association was ruled out by documenting within-individual change. After controlling for each child's aggression up to age 5, the children exposed to an episode of maternal depression between ages 5 and 7 became more aggressive by the age 7 assessment. Taken together, these four results are not consistent with a genetic account of the association between maternal depression and children's aggression.

The E-risk study also examined the effects of physical maltreatment on young children's aggression (Jaffee, Caspi, Moffitt, & Taylor, 2004), using twin-specific reports of maltreatment. This study satisfied six conditions that together supported the hypothesis that physical maltreatment has an environmentally mediated causal influence on children's aggression: (1) children's maltreatment history prospectively predicted aggression; (2) the severity of maltreatment bore a dose–response relation to aggression; (3) the experience of maltreatment was followed by increases in aggression from prior levels, within individual children; (4) there was no child effect provoking maltreatment; (5) maltreatment predicted aggression while mothers' and fathers' antisocial behavior were statistically controlled; and (6) modest but significant effects of maltreatment on aggression remained present after controlling for genetic transmission of liability to aggression in the family. A similar analytic approach using twin-specific measures of risk was taken by the Minnesota Twin Family Study (Burt, Krueger, McGue, & Iacono, 2003), which studied 808 11-year-old twin pairs. Models revealed that measured parent–child conflict accounted for 12% of the variance in the externalizing syndrome of oppositional, conduct, and attention-deficit/hyperactivity disorders (23% of the common environment variation in this syndrome).

As a final design, we can study *MZ twin children* to test if differences between siblings in their exposure to bad parenting makes them different on aggression. The fact that MZ twins are not perfectly concordant for aggression opens a window of opportunity to uncover if a nongenetic cause specific to one twin has produced the behavioral difference. A number of studies have tested if differential parental treatment can account for antisocial behavior differences between siblings and cousins within a family (e.g., Conger & Conger, 1994; Reiss et al., 2000; Rodgers, Rowe, & Li, 1994). Most of these studies have already been reviewed by Turkheimer and Waldron (2000). However, comparing the parenting experiences of discordant MZ twins allows the least ambiguous interpretation of results. Three studies have reported that MZ twin differences in bad parenting are correlated with MZ twin differences in antisocial behavior (Asbury, Dunn, Pike, & Plomin, 2003; Caspi et al., 2004; Pike, Reiss, Hetherington, & Plomin, 1996).

The E-risk study reported that within 600 MZ twin pairs, the twin who received relatively more maternal negativity and less maternal warmth developed more antisocial behavior problems (Caspi et al., 2004). Negativity and warmth were measured by coding voice tone and speech content in mothers' audiotaped speech about each of their twins separately, according to the well-known "expressed emotion" paradigm. This study provided the strongest evidence to date that the effect of mothers' emotional treatment of children causes aggression, by ruling out five alternative explanations of the finding.

1. Using MZ twin pairs ruled out the possibility that a genetically transmitted liability explained both the mother's emotion and her child's antisocial behavior.
2. Using MZ twins also ruled out the possibility that a genetic child effect provoking maternal emotion accounted for the finding.
3. The study used the longitudinal natural experiment approach to rule out that any non-genetic child effect provoking maternal emotion accounted for the finding, by controlling for prior behavior that could have provoked maternal negative emotion and showing that individual children whose mothers were negative toward them at age 5 evidenced a subsequent increase of antisocial behavior between age 5 and age 7.
4. The study controlled for twin differences in birthweight in an effort to rule out the possibility that twins with neurodevelopmental difficulties had more behavior problems and elicited more negative emotion from mothers.
5. The study measured the children's behavior using teacher reports to rule out the possibility that a mother's negativity toward a child led her to exaggerate her report of the child's behavior problems.

Effect sizes for the influence of maternal emotion on children's aggression ranged from large (r = .53) to small (r = .10), depending on how many controls were applied.

Not All Tests of Putative Environmental Risk Factors Confirm Environmental Effects

Lest readers assume that application of behavioral-genetics methods to a putative environmental risk factor will necessarily affirm that its effects are environmentally mediated, it is useful to mention that some known risk factors do not appear to be causal. First, as noted previously, we found that children's genes accounted for virtually all the association between their corporal punishment (i.e., spanking) and their conduct problems. This indicated a "child effect," in which children's bad conduct provokes their parents to use more corporal punishment, rather than the reverse (Jaffee, Caspi, Moffit, Polo-Tomas, et al., 2004).

Second, studies have reported that mothers' smoking during pregnancy is correlated with children's conduct problems, but pregnancy smoking is known to be concentrated among mothers who are antisocial, have mental health problems, mate with antisocial men, and rear children in conditions of social deprivation. When the family liability for transmission of psychopathology from parents to children was controlled through statistical controls for the parents' antisocial behavior, mental health, and social deprivation, the effect of even heavy smoking during pregnancy disappeared. This study suggests that although pregnancy smoking undoubtedly has undesirable effects on outcomes such as infant birthweight, it is probably not a cause of conduct problems (Maughan, Taylor, Caspi, & Moffitt, 2004).

A third finding of nil environmental influence concerned father absence. In families having absent fathers, the children are known to have more conduct problems. However, absent fathers are more antisocial on average than fathers who stay with their children, and antisocial behavior can be genetically transmitted. When we controlled for mother's and father's antisocial history, we found that the association between father absence and children's conduct problems disappeared. This suggests that father absence is not a direct cause of conduct problems but, rather, is a proxy indicator for familial liability to antisocial behavior (Jaffee et al., 2003).

What Research Is Needed?

To date, question 5, "Does bad parenting have an environmentally mediated causal effect on children's aggression?," has been answered in the affirmative by behavioral-genetics reports from several twin samples, finding such effects for family adaptability, parent–child conflict, parental monitoring, bad fathering, maternal depression, physical maltreatment, and mothers' negative expressed emotions. These studies share an Achilles' heel; because different forms of parenting risk are concentrated in the same families, the particular parenting measure targeted in a study may be a proxy for some other, correlated risk factor. Research is needed that isolates the effects of one risk factor from its correlates. Nevertheless, whatever the most influential parenting behaviors are, the studies attest that parents can have environmentally mediated effects.

It may surprise some developmentalists to learn that when familial liability and child effects are controlled, parenting influences on children drop to small effect sizes. However, small effects ought to be expected, for three reasons. First, it must be remembered that these small effects reflect true environmental associations after they have been purged of the confounding influences that inflate effect sizes in nongenetic studies. Associations between risk factors and behavior outcomes tend to shrink by at least half when genetic confounds are controlled (Turkheimer & Waldron, 2000). This shrinkage suggests that the risk–outcome correlations that social scientists are accustomed to seeing are inflated to about double their true size. Second, small effects for any particular risk factor make sense, in view of evidence that clear risk for antisocial behavior accrues only when a person accumulates a large number of risks (Rutter, Giller, & Hagell, 1998), each of which may individually have only a small effect (Daniels & Plomin, 1985).

A third reason why small effects should not be too surprising is that they represent the main effects of measured environments, apart from any environmental effects involved in gene–environment (G × E) interactions. Recall that adoption studies found no effects of bad adoptive parenting in the absence of genetic liability, but bad adoptive parenting was associated with elevated antisocial outcomes for adoptees at genetic risk (Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Mednick, Gabrielli, & Hutchings, 1984). In twin designs, when testing whether the shared experience of bad parenting enhances twin similarity in aggression over and above genetic influences on similarity, G × E interactions are controlled along with other genetic influences. In twin designs testing whether differential experiences of bad parenting are associated with MZ twin differences in aggression, differential outcomes arising from G × E interactions are ruled out by the twins' identical genotypes. In contrast, genetic risk and bad parenting are not usually disentangled in real life as they are in behavioral-genetics studies. In ordinary lives, genetic and environmental risks often coincide. It is possible in theory that environmental effects conditional on genetic vulnerability could be quite large. We next turn to the question of G × E interactions influencing antisocial behavior.

6. Testing the Hypothesis of Interaction between Genes and Environments

The study of G × E interaction entails substantial methodological challenges. It requires measured environments that are truly environmental, measured genetic influence, some means of separating them from each other, and enough statistical power for a sensitive test of interaction (Rutter & Silberg, 2002). Despite the challenges, theory-driven hypotheses of G × E interaction are well worth testing, because where measured G × E is found

to influence behavior disorders, both specific genes and specific environmental risks can conceivably have moderate-to-large effects, as opposed to the very small effects expected from prior quantitative genetic research. Specific genes revealed to be stronger in the presence of environmental risk would guide strategic research into those genes' expression, possibly leading to genetic diagnostics and improved pharmacological interventions (Evans & Relling, 1999). Specific environmental effects revealed to be stronger in the presence of genetic risk would prompt a new impetus for specific environmental prevention efforts, and would help to identify who needs the prevention programs most. The study of G × E is especially exciting in antisocial behavior research, where investigations have pioneered the way for all behavioral disorders. Studies of antisocial behavior were first to report evidence of interaction between latent genetic and latent environmental risks ascertained in adoption studies, and also first to report evidence of an interaction between a measured genetic polymorphism and a measured environmental risk. Four research designs have been used.

Adoption Studies of Latent G × E

The first evidence that genetic and environmental risks influence antisocial behavior in a synergistic way came from adoption studies. Among the 6,000 families of male adoptees in the Danish Adoption Study, 14% of adoptees were convicted of crime though neither their biological nor their adoptive parents had been convicted, whereas 15% were convicted if their adoptive parent alone was convicted, 20% were convicted if their biological parent alone was convicted, and 25% were convicted if both biological and adoptive parents were convicted, although there were only 143 such cases (Mednick & Christiansen, 1977). This pattern of percentages did not represent a statistically significant cross-over interaction term, but it did illustrate clearly that the effects of genetic and environmental risk acting together were greater than the effects of either factor acting alone. The finding was buttressed by two studies from American and Swedish adoption registers completed about the same time (Cadoret et al., 1983; Cloninger, Sigvardsson, Bohman, & von Knorring, 1982).

Adoption Studies of Latent G × Measured E

In a pool of 500 adoptees from the Iowa and Missouri adoption studies, adoptees had the most elevated antisocial behaviors when they experienced "adverse circumstances" in their adoptive homes as well as having birth mothers with antisocial personality problems or alcoholism (Cadoret et al., 1983). This landmark study documented that the interaction was statistically significant and replicated across two independent samples. This finding was replicated and extended in another Iowa adoption cohort of 200 families (Cadoret, et al., 1995). Adoptive parents' adversity was defined according to the presence of marital problems, legal problems, substance abuse, or mental disorder, and it interacted significantly with biological parents' antisocial personality disorder to predict elevated rates of childhood aggression, adolescent aggression, and diagnosed conduct disorder in the adoptees. This same Iowa adoption study was creatively analyzed to demonstrate that adversity in the adoptive home can moderate the genetic child effect in which children's aggression provokes bad parenting (Riggins-Caspers et al., 2003). Adoptees' genetic liability for antisocial behavior (defined as biological parents' psycho-

pathology) provoked more harsh discipline from the adoptive parents in homes in which the adoptive parents suffered adversity (marital, legal, substance, or psychopathology problems). There is one problem with studying G × E in adoption designs, and it is that adoption itself breaks up the naturally occurring processes of rGE that characterize the nonadopted majority population, thereby precluding the possibility of G × E. This separation allows the empirical study of G × E, but paradoxically, it probably results in an underestimate of the influence of G × E on antisocial outcomes in the general population. For this reason, adoption G × E studies should be complemented with twin studies.

A Twin Study of Latent G × Measured E

Our E-risk twin study also yielded evidence that genetic and environmental risks interact (Jaffee et al., 2005). Because we already knew that conduct problems were highly heritable in the E-risk twin sample at age 5 years (Arseneault et al., 2003), we were able to estimate each child's personal genetic risk for conduct problems by considering whether his or her co-twin had already been diagnosed with conduct disorder, and whether he or she shared 100% versus 50% of genes with that diagnosed co-twin. This method's usefulness had been demonstrated previously in a landmark G × E study showing that the risk of depression following life-event stress depends on genetic vulnerability (Kendler et al., 1995). For example, an individual's genetic risk is highest if his or her co-twin sibling already has a diagnosis of disorder and the pair is monozygotic. Likewise, an individual's genetic risk is lowest if his or her co-twin has been free from disorder and the pair is monozygotic. Individuals in DZ twin pairs fall between the high and low genetic risk groups. In our study an interaction was obtained such that the effect of maltreatment on conduct problem symptoms was significantly stronger among children at high genetic risk than among children at low genetic risk. (Because there was no genetic child effect provoking maltreatment, the genetic risk groups did not differ on concordance for maltreatment or the severity of maltreatment.) In addition, the experience of maltreatment was associated with an increase of 24% in the probability of diagnosable conduct disorder among children at high genetic risk, but an increase of only 2% among children at low risk.

Studies of Measured G × Measured E: Testing a Measured Gene

The aforementioned adoption and twin studies established that genotype does interact with bad parenting in the etiological processes leading to antisocial behavior. However, the studies did not implicate any particular genes. We conducted one study to test the hypothesis of G × E interaction using a measured environmental risk, child maltreatment, and an identified gene, the monoamine oxidase A(MAOA) polymorphism (Caspi et al., 2002). We selected the MAOA gene as the candidate gene for our study for four reasons (supporting research is cited in Caspi et al., 2002). First, the gene encodes the MAOA enzyme, which metabolizes the neurotransmitters linked to maltreatment victimization and aggressive behavior by previous research. Second, drugs inhibiting the action of the MAO enzyme have been shown to prevent animals from habituating to chronic stressors analogous to maltreatment and to dispose animals toward hyperreactivity to threat. Third, in studies of mice having the MAOA gene deleted, increased levels of neurotransmitters and aggressive behavior were observed, and aggression was normalized by restoring MAOA

gene expression. Fourth, an extremely rare mutation causing a null allele at the MAOA locus was associated with aggressive psychopathology among some men in a Dutch family pedigree, although no relation between MAOA genotype and aggression had been detected for people in the general population.

We selected maltreatment for this study for four reasons (supporting research is cited in Caspi et al., 2002). First, childhood maltreatment is a known predictor of antisocial outcomes. Second, not all maltreated children become antisocial, suggesting that vulnerability to maltreatment is influenced by heretofore unstudied individual characteristics. Third, our abovementioned twin research had established that maltreatment's effect on children's aggression is environmentally mediated (i.e., the association is not an artifact of a genetic child effect provoking maltreatment or of transmission of aggression-prone genes from parents). As such, maltreatment can serve as the environmental variable in a test of G × E interaction. Fourth, animal and human studies suggest that maltreatment in early life alters neurotransmitter systems in ways that can persist into adulthood and can influence aggressive behavior.

Based on this logic to support our hypothesis of G × E, we measured childhood maltreatment history (8% severe, 28% probable, 64% not maltreated) and MAOA genotype (37% low-activity risk allele, 63% high-activity allele) in the 442 caucasian males of the longitudinal Dunedin Multidisciplinary Health and Development Study. We found that maltreatment history and genotype interacted to predict four different measures of antisocial outcome: an adolescent diagnosis of conduct disorder, an age-26 personality assessment of aggression, symptoms of adult antisocial personality disorder reported by informants who knew the study members well, and court conviction for violent crime up to age 26, the latest age of follow-up. Among boys having the combination of the low-MAOA-activity allele and severe maltreatment, 85% developed some form of antisocial outcome. Males having the combination of the low-activity allele and severe-to-probable maltreatment were only 12% of the male birth cohort, but they accounted for 44% of the cohort's violent convictions, because they offended at a higher rate on average than other violent offenders in the cohort.

Replication of this study was of utmost importance, because the study reported the first instance of interaction between a measured gene and a measured environment in the behavioral sciences, and because reports of connections between measured genes and disorders are notorious for their poor replication record (Hamer, 2002). One initial positive replication and extension has emerged from the Virginia Twin Study for Adolescent Behavioral Development (Foley et al., 2004). This team studied 514 caucasian male twins and measured environmental risk using an adversity index comprised of parental neglect, interparental violence, and inconsistent discipline. MAOA genotype and adversity interacted significantly such that 15% of boys having adversity but the high-MAOA-activity allele developed conduct disorder, in comparison to 35% of boys having adversity plus the low-activity allele. This study went a step further, controlling for maternal antisocial personality disorder to rule out the possibility that passive rGE might have resulted in the co-occurrence of environmental and genetic risk. This study thus replicated the original G × E between the MAOA polymorphism and maltreatment, extended it to other forms of parental treatment, and showed that it is not an artifact of passive rGE. Another study has tested the MAOA G × E effect, and although the pattern of findings was consistent with the interaction, it did not attain statistical significance (Haberstick et al., 2005).

Genes as Protective Factors Promoting Resilience

An intriguing finding from the two MAOA G × E studies was that, in contrast to the G × E interaction's marked effects on antisocial outcomes, the unique effects of maltreatment apart from its role in the G × E interaction were very modest. Maltreatment initially predicted antisocial outcomes in the full cohorts, but within the high-MAOA-activity genotype group its effects were reduced by more than half (Caspi et al., 2002; Foley et al., 2004). This pattern is in keeping with the findings from adoption and twin studies cited earlier in this section, all of which found that measured bad parenting had relatively little effect on children who were at low genetic risk (Cadoret et al., 1983; Cadoret et al., 1995; Cloninger et al., 1982; Jaffee et al., 2005; Mednick et al., 1984). Taken together, these findings suggest the novel notion that genotype can be a protective factor against adversity. Some people respond poorly to adversity while others are resilient to it, and the reason for this variation has been a holy grail in developmental research. The search for sources of resilience has tended to focus on social experiences thought to protect children, overlooking a potential protective role of genes (but see Kim-Cohen, Moffitt, Caspi, & Taylor, 2004). The potential protective effect of genes deserves more attention (Insel & Collins, 2003).

CONCLUSION

In this chapter we reviewed the first studies in a new generation of research that exploits behavioral-genetics designs to address the interplay between measured environmental risks and genetic risks in the origins of antisocial behavior. This work has only recently accelerated, and more of it is needed before drawing conclusions (Dick & Rose, 2002; Kendler, 2001). However, even the few studies so far counteract prior claims that associations between family risk factors and child antisocial outcome might be nothing more than a spurious artifact of familial genetic transmission. This argument can be subjected to empirical test, and such tests need to address both child effects on environments (involving children's genes) and gene–environment correlations (involving parents' genes). Further, although the "residual main effects" of environmental risk factors may appear small after controlling for genetic transmission, that is not the whole story. Emerging evidence about G × E interactions suggests that environmental risks can affect people more strongly than previously appreciated, in genetically vulnerable segments of the population. Although this chapter has argued that twin and adoption studies together can provide a good evidence base, the most compelling information about gene–environment interplay will come from converging findings from behavioral-genetics designs, treatment experiments, and longitudinal natural experiments showing within-individual change.

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