

# On the Nature and Nurture of Antisocial Behavior and Violence

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**ABSTRACT:** This article focuses on the contribution that behavioral genetic research can make to further the understanding of how antisocial and violent behavior develops. Genetically informative study designs are particularly useful for investigating etiological heterogeneity and can refine the search for developmental pathways to persistent antisocial conduct. While the current data are not yet directly translatable for prevention programs, behavioral genetic research will have far-reaching implications for prevention and treatment. As we find genes associated with risk for antisocial behavior and develop better understanding of the mechanics of the interplay between genes and the environment, we can expect to tailor prevention and treatment to serve the specific needs of etiologically distinct subgroups of children. Furthermore, researchers involved in sharpening the knowledge base have the responsibility of trying to ensure that such findings are not misused.

**KEYWORDS:** behavior genetics; antisocial behavior; etiology

## INTRODUCTION

The nature vs. nurture debate seems somewhat dated these days, as most scientists appear happy with the notion that both genetic predisposition and environmental risk/protective factors mould individuals throughout development. The interest has shifted to assessing the relative importance of genes and environmental influences on any given trait or ability and, more importantly, to discovering the developmental processes of genetic and environment interplay. In this article I will selectively summarize some research on antisocial and violent behavior that addresses etiological questions. I will first briefly and selectively outline quantitative genetic findings of antisocial and violent behavior, specifically highlighting our recent findings on etiological heterogeneity within early-onset antisocial behavior. Children who manifest behavior problems early in life are at the greatest risk for chronic, life-course persistent antisocial behavior, including violence.<sup>1</sup> However,

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these children do not appear to form an etiologically unitary group. In the following section I will then describe how genetic study designs can be used to answer specific questions about environmental risk factors for antisocial behavior. Next I will discuss the challenges for finding genes associated with antisocial behavior and violence. In the last section of this paper I will outline tentative implications of findings on genes and environment for both prevention and treatment.

### QUANTITATIVE GENETICS OF ANTISOCIAL AND VIOLENT BEHAVIOR

Rhee and Waldman<sup>2</sup> recently conducted a meta-analysis of behavior genetic studies on antisocial behavior and estimated that on average about 41% of the variance on antisocial behavior was due to genetic factors, about 16% to shared environmental factors, and about 43% to non-shared environmental factors. These estimates were based on findings from 51 studies that varied in sample size from very small (less than hundred participants) to large (thousands of participants), used different methods to infer estimates of heritability and environmental influence (twin, adoption, and sibling study designs), collected data on different age groups (children, juveniles, and adults), and applied different definitions of antisocial behavior using different informants. However, the basic finding is that antisocial behavior is moderately heritable. The same appears to hold for violence,<sup>3-5</sup> although studies using official conviction records have reported evidence of nil heritability.<sup>6,7</sup> There is a logical explanation to the nil heritability estimate, as using conviction records to measure violence will miss out on information on actual crimes committed, while including some individuals who have only committed a single, one-off violent act. Furthermore, small sample sizes of the studies relying on conviction records limits the statistical power of these analyses. Overall, the data appear to indicate moderate heritability for both antisocial and violent behavior.

If we use sufficiently large twin samples we can ask questions that go beyond demonstrating that antisocial behavior is heritable. We can investigate genetic and environmental influences on co-morbidity between antisocial behavior and other behavioral problems. For example, there is suggestive evidence of overlapping genetic influences for conduct disturbance and hyperactivity.<sup>8</sup> We can ask developmental questions by assessing antisocial behavior over time and investigate whether genetic or environmental variables contribute to its continuity or change by measuring twin 1's phenotypic score at age one, and twin 2's phenotypic score at age two. As an example, a recent study suggested that continuity of aggressive antisocial behavior is genetically mediated.<sup>9</sup> We can also use the twin design to divide groups of antisocial individuals on some distinct behavioral or personality marker to see whether etiological influences differ for different subtypes. For example, early-onset antisocial behavior appears to be more heritable than adolescence-limited antisocial behavior, a finding that has been argued to reflect the transient, almost normative nature of adolescent antisocial behavior.<sup>1</sup> On the other hand, the higher heritability estimate for antisocial behavior in young children may reflect a genetic predisposition to such behavior, particularly when antisocial behavior is pervasive over different settings.<sup>10,a</sup>

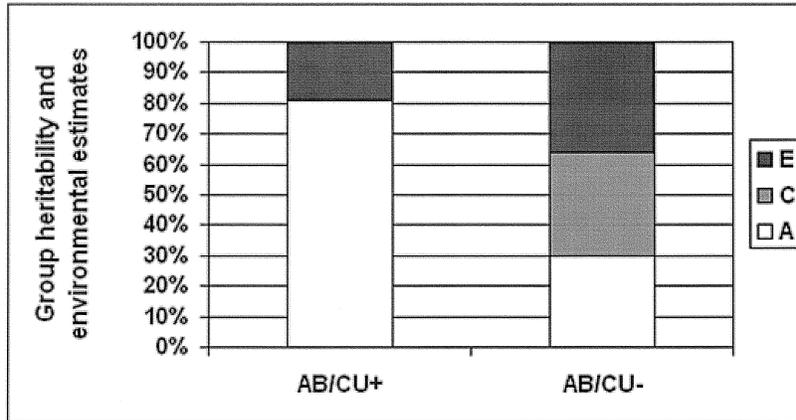
### QUANTITATIVE GENETICS HIGHLIGHTING ISSUES FOR PREVENTION

Our recent findings can serve as an example of investigation that goes beyond demonstrating heritability of a trait and that has potential to inform prevention. Previous research led us to ask whether there may be etiologically different subtypes within the early-onset subgroup of antisocial children.<sup>12</sup> This is an important question, as a demonstration of etiological differences in the early-onset group may index a need for differentiated prevention and treatment efforts that mirror the relevant risk factors for antisocial behavior in a particular sub-group.

Earlier research had demonstrated that callous–unemotional personality traits (lack of guilt and empathy) delineated a distinct group of antisocial children and adults with high levels of aggression (including proactive aggression), poor prognosis, and a neurocognitive profile characterized by dysfunctional processing of others' distress and hyper-reactivity to rewards.<sup>13–15</sup> In addition, antisocial children and adults with callous–unemotional personality traits did not show the traditional association between environmental risk factors (e.g., parenting or environment upon release from prison) and manifestation or maintenance of antisocial behavior.<sup>15,16</sup> In adults the combination of callous–unemotional traits and antisocial behavior is called psychopathy. It must be emphasized that psychopathy is an adult diagnosis and children should not be labeled as psychopaths. However, charting the callous–unemotional traits in children and investigating whether children with callous–unemotional personality traits differ in etiology from other children with early-onset antisocial behavior is an important enterprise with implications for prevention and treatment. Although previous research had strongly suggested that children with early-onset antisocial behavior coupled with callous–unemotional traits form a distinct subtype,<sup>13,14</sup> possible etiological differences between these children and others with early-onset antisocial behavior had not been assessed.

To address this question we studied teacher ratings of callous–unemotional traits and antisocial behavior in more than 7000 7-year-old twins from the Twins Early Development Study (TEDS). We found that exhibiting extreme levels of callous and

<sup>a</sup>The twin method is a natural experiment that relies on the different levels of genetic relatedness between MZ and DZ twin pairs to estimate the contribution of genetic and environmental factors to individual differences, or extreme scores in a phenotype of interest. Phenotypes include any behavior or characteristic that is measured separately for each twin, such as twins' score on an antisocial behavior checklist. Statistical model-fitting techniques and regression analyses methods incorporating genetic relatedness parameter are used to investigate the etiology of the phenotype of choice. I will not cover these techniques on this article and an interested reader should read Plomin's textbook *Behavioral Genetics*.<sup>11</sup> The basic premise of the twin method is this: If identical twins, who share 100% of their genetic material, appear more similar on a trait than fraternal twins, who share on average 50% of their genetic material (like any siblings), then we infer that there are genetic influences on a trait. Identical twins' genetic similarity is twice that of fraternal twins'. If nothing apart from genes influences behavior, then we would expect the identical twins to be twice as similar with respect to the phenotypic measure than fraternal twins. Shared environmental influences—environmental influences that make twins similar to each other—are inferred if fraternal twins appear more similar than is expected from sharing 50% of their genes. Finally, if identical twins are not 100% similar on a trait (as would be expected if only genes influenced a trait), non-shared environmental influences are inferred—in other words environmental influences that make twins different from each other. The non-shared environmental estimate also includes measurement error.



**FIGURE 1.** Estimates of genetic and environmental contribution to extreme levels of antisocial behavior (AB) in children with callous–unemotional traits (AB/CU+) and children without callous–unemotional traits (AB/CU–). A = genetic; C = shared environment; E = non-shared environment.

unemotional traits was under strong genetic influence (heritability of .67).<sup>12</sup> After establishing that the callous–unemotional traits were highly heritable in children, we conducted further research on children with antisocial behavior who either had or did not have elevated levels of these traits. When we separated children with antisocial behavior into these two groups, the most interesting result emerged (Fig. 1). Antisocial behavior in children with elevated levels of callous–unemotional traits was under extremely high genetic influence (heritability of .81) and no influence of shared environment. In contrast, antisocial behavior in children without elevated levels of callous–unemotional traits showed moderate genetic influence (heritability of .30) and substantial environmental influence (shared environmental influence = .34; non-shared environmental influence = .26). This is a novel finding and represents an etiologically based refinement for theories of antisocial behavior. Furthermore, it indicates that there is heterogeneity within the early-onset group that warrants further investigation.

#### WHAT ARE THE IMPLICATIONS OF ETIOLOGICAL HETEROGENEITY?

These findings of etiological heterogeneity have implications for future research on development of antisocial behavior. We are currently following up the twins at 9-years and will be able to assess whether the two subgroups of children with early-onset antisocial behavior show different patterns of stability for the antisocial behavior or differ in their environmental risk factors. As we are using a twin design, we can also address whether genetic or environmental effects are primarily responsible for any change or continuity in antisocial behavior in the two subgroups. The finding of high heritability in the callous–unemotional subgroup of children with early-onset antisocial behavior also suggests that molecular genetic research may be particularly

fruitful in this group and we are following this up (see the section entitled **FINDING GENES** in this article). In contrast, children with early-onset antisocial behavior, but without elevated levels of callous–unemotional trait, had a strong environmental influence on their antisocial behavior. Using theoretically defined environmental measures, as well as incorporating information about DNA polymorphisms, is particularly important for these future research efforts. The following sections will briefly discuss both environmental and molecular genetic research on antisocial and violent behavior.

#### **FINDING ENVIRONMENTS: HOW GENETIC STUDY DESIGNS CAN BE USED TO ANSWER SPECIFIC QUESTIONS ABOUT ENVIRONMENTAL RISK FACTORS FOR ANTISOCIAL BEHAVIOR**

The finding of substantial shared environmental influence for antisocial behavior in children without elevated levels of callous–unemotional traits indicates that family-wide environmental influences that are not acting on the child's genotype are important for the development of antisocial behavior in this subgroup.<sup>12</sup> In contrast, environmental influences acting in tandem with the genotype, as well as environmental influences unique to the child, appear more important for the development of antisocial behavior in children with elevated levels of callous–unemotional traits. We are in the process of charting common and unique environmental risk factors for antisocial behavior in these two etiologically distinct subtypes.

Behavioral genetic methods are powerful in informing the causal theory of the development of antisocial behavior. For example, comparing identical twins who differ on antisocial behavior outcome can enlighten us about child-specific nonshared environmental risk factors for antisocial behavior independent of genetic influence. Furthermore, given that genetic effects influence the likelihood of environmental risk, as well as the reaction to environmental risk, it is important to study measured, well-defined environmental factors within genetically sensitive designs where we can assess genetic–environmental correlation and genetic–environmental interaction, as well as environmental effects over and above gene–environment correlation and gene–environment interaction. By gene–environment correlation, behavioral geneticists can mean either passive, active, or evocative gene–environment relationships. A passive gene–environment correlation reflects genetic influence on exposure to environment, such as an adult's genetic propensity for antisocial behavior influencing the likelihood of that adult engaging in maltreatment—an environmental risk factor for the child. Evocative and active genetic–environmental correlations reflect experiences that are correlated with genetic propensities. An example of evocative gene–environment correlation might be a child's temperamental predisposition (which is partly under genetic influence) evoking a certain disciplinary reaction. An active gene–environment correlation could be a child's seeking out particular risk environments. This type of gene–environment correlation could be particularly relevant in adolescence or for children who receive inadequate parental supervision. By gene–environment interaction behavioral geneticists mean genetic sensitivity or susceptibility to environments, such that only children with certain genotypes will develop an antisocial outcome after exposure to environmental risk.

A study by Caspi and colleagues is a good example of using behavioral genetic methods to study the nature of association between child-specific environmental risk and antisocial behavior.<sup>17</sup> This study used a longitudinal design and compared identical twins discordant for teacher-rated antisocial behavior, demonstrating that mothers' negative emotional attitudes toward their children were associated with children's antisocial behavior. This association was not purely a child effect (i.e., an effect of children's behavior on parental treatment), as the longitudinal analyses documented that even after children's antisocial behavior at age 5 was controlled for, maternal negative expressed emotion still predicted increases in children's antisocial behavior at age 7. The discordant identical twins design ruled out that the association between maternal expressed emotion and children's antisocial behavior was genetically mediated, as maternal expressed emotion predicted differences between genetically identical individuals. These findings suggest maternal expressed emotion is an environmentally mediated risk factor, and possibly an environmental cause for antisocial behavior. Caspi and colleagues did not divide their sample of children to those with and without elevated levels of callous-unemotional traits. It might be interesting to include this distinction in future research. There is some suggestive evidence that children with early-onset antisocial behavior and elevated levels of callous-unemotional traits do not show the same association between poor parenting practices and increased antisocial behavior, as do children with early-onset antisocial behavior without elevated levels of callous-unemotional traits.<sup>16</sup>

Another recent twin study focused on maltreatment as an environmental risk factor.<sup>18</sup> This study supported the hypothesis that physical maltreatment is an environmental risk variable that is causally linked to children's antisocial behavior. Jaffee and colleagues found that physical maltreatment prospectively predicted antisocial outcome, that the extent of physical maltreatment was related to the level of antisocial behavior, and that physical maltreatment was followed by emergence of new antisocial behavior.<sup>18</sup> Controlling for parental history of antisocial behavior demonstrated that the association between physical maltreatment was not entirely accounted for by the parents' genotype—in other words, physical maltreatment had an effect on children's antisocial behavior over and above passive gene-environment correlation. Again, this study did not divide children with early-onset antisocial behavior to those with and without elevated levels of callous-unemotional traits. Given the different levels of genetic risk tentatively associated with the two subtypes, it may be interesting to see whether this particular environmental risk factor operates differently on the two subtypes.

The above examples provide a flavor of how environmental risk can be studied using behavioral genetic methodology. One further example, involving study of gene-environment interaction, will be provided in the section just below. An interested reader is referred to a paper by Rutter and Silberg for a more in-depth discussion of the issues raised in this article.<sup>19</sup> In concluding this section, I would like to stress that genetic study designs have very convincingly demonstrated that there are environmental risk factors that appear causal to the development of antisocial behavior while controlling for genetic factors. This is true for physical maltreatment that is a particularly extreme environmental risk factor, as well as for maternal expressed emotion—a risk factor that may be more common and does not on the surface appear so extreme.<sup>17,18</sup>

### FINDING GENES

When we find genes associated with antisocial behavior we will have a more powerful way of examining the questions of co-morbidity, developmental trajectory, and heterogeneity that I outlined earlier, in the twin method section of this article. For example, we can examine whether the same genes are the risk genes for both antisocial behavior and hyperactivity. We can also assess which genes are important at different points in development, and how risk genes may interact with risk environments throughout development.

Common behavioral disorders are currently proposed to be the quantitative extreme of the same genetic effects that operate throughout the distribution.<sup>20</sup> In this Quantitative Trait Loci (QTL) model, many genes are hypothesized to be involved in the development of any behavior pattern and these genes are thought to act in a probabilistic manner. There has been slow progress in identifying QTLs, as they are neither sufficient, nor necessary to cause extreme behavioral outcome. They can be said to act together with other risk or protective genes to increase or reduce the risk of disorder. Furthermore, a risk gene may have to be combined with environmental risk before a clinically significant outcome is produced. It is thus important to understand that genes are not a blueprint determining outcome.

There is now suggestive evidence that a number of serotonin pathway genes may be associated with impulsive antisocial, aggressive, and violent behavior.<sup>21</sup> Not all of these findings have been replicated, but this may not be so surprising given the likely heterogeneous nature of the samples under study and the fact that the genes act in a probabilistic, rather than deterministic manner. The molecular genetic research on antisocial behavior so far has primarily concentrated on impulsive antisocial and violent behavior. We are currently trying to find genes associated with callous-unemotional (CU) traits using our twin sample and a novel DNA pooling strategy comparing composite DNA from large samples of groups of individuals high versus low on the quantitative trait.<sup>22</sup> Pooled DNA will be genotyped on microarrays (gene chips) that can genotype more than 100,000 DNA markers in two days.<sup>23</sup>

If we find genes associated with CU traits, these genes can then be used to answer the questions I have highlighted earlier. For example, we can see whether the same genes associated with CU traits are also associated with some other phenotypes. We can also see whether these genes are associated with persistent risk. Perhaps the most exciting possibilities lie in the realm of using measured genes to study gene-environment interaction. The study of gene-environment interaction is possibly the most exciting follow-up for finding genes associated with behavior. As an example, a recent study by Caspi and colleagues demonstrated the involvement of a risk MAOA allele in antisocial behavior, but only if the carrier of the vulnerable genotype had also experienced childhood maltreatment.<sup>24</sup> MAOA is involved in regulating serotonin at key developmental stages, and this finding highlights the importance of genetically influenced levels of serotonin functioning in buffering/exposing individuals to environmental stress. It is interesting to note that a small subgroup of individuals in this study with both genetic and environmental risk present, did not go on to develop violent and antisocial behavior. This suggests the possibility of additional risk genes operating in tandem with the MAOA, or alternatively some pro-

tective genes modulating the impact of “MAOA–maltreatment” combination in some individuals.

### **IMPLICATIONS FOR PREVENTION AND TREATMENT**

The research reviewed above suggests that there may be a particularly genetically vulnerable group of youngsters for whom early intervention is likely to be extremely crucial to prevent a life course–persistent antisocial outcome. I would also like to highlight that prevention and treatment strategies should take into account the different etiologies of subgroups of antisocial and violent children. Etiologically heterogeneous samples may explain why intervention programs can sometimes have mixed results.<sup>25</sup> Some children seem to respond to well-timed, early prevention and treatment, while others do not. I would suggest that the root of this may lie in etiological differences. The modest to moderate success of intervention programs may actually reflect a high success rate for affecting the outcome for a particular etiological subtype. Frick has emphasized that while there are prevention programs available that address the needs of primarily impulsive antisocial behavior, less is known about possible prevention and treatment of antisocial behavior in the callous–unemotional subtype.<sup>25</sup>

Research on environmental risk factors within behavioral genetic designs has highlighted a number of important issues. It is more than likely that for children with a vulnerable genotype, this genotype will react with risk environments. Furthermore, at least one of the parents will share the risk genes for antisocial behavior and is thus more likely to either directly or indirectly contribute to a less-than-optimal rearing environment. As the parent or parents with the antisocial genotype are not often willing or capable of engaging in efforts toward prevention and treatment, these families present a particular challenge for professionals engaged in preventing future, ongoing cycles of violence. However, recent successes with nurse-visit programs in breaking the association between maltreatment and antisocial behavior suggest that genetic risk can be effectively moderated by environmental intervention.<sup>26,27</sup>

### **IS KNOWLEDGE OF ETIOLOGY NEEDED TO IMPROVE CLINICAL PRACTICE?**

Do the findings from behavioral genetic studies (both quantitative genetic studies and those including measured genes and environment) actually yield results relevant for the practitioner? I would argue that they do. As demonstrated in this article, genetic research is important for studying the causality and “mode of operation” of environmental risk factors in affecting antisocial behavior outcome. Behavioral genetic research also cautions against entertaining ideas of gene therapy for antisocial behavior. Genes that have variants that are common in the population are more than likely to have multiple functions, some of which are desirable, others not. Hence, a risk gene may have many functions over and above increasing risk for disorder. When this information is combined with the fact that genes interact in com-

plex systems, as well as with environmental risk factors, it seems pertinent to conclude that removing the effects of one gene via gene therapy is unlikely to be effective.<sup>28</sup>

This does not mean that genotype information will be irrelevant for therapeutic intervention. For example, demonstration of genetically heterogeneous subtypes of early-onset antisocial behavior suggests the possibility of subtype-specific risk gene variants. An early knowledge of such risk genes may come to guide prevention efforts prior to the emergence of clear, overt behavioral markers for the disorder. As cognitive-behavioral approaches are likely to feature strongly in the antisocial behavior intervention, developing better understanding of the genes-brain/cognition-behavior pathways for particular subtypes—especially within a longitudinal, developmental framework—could provide crucial insights for intervention. For example, using brain imaging techniques on genotypically discordant fraternal twins set to perform a theoretically meaningful cognitive task in the scanner could tell us about genetic contributions to functional brain differences associated with antisocial behavior. As there is ever-increasing knowledge about developmental vulnerability periods of particular brain areas, best timing for the environmental modulation of genetic risk could be cued with such study designs. Use of identical twins discordant for the disorder in a similar scanning experiment would yield information about non-shared environmental influences on brain/cognitive function crucial for the development of antisocial behavior. Aside from the obvious target of preventing environmental risk, knowledge of the nature of environmental risk that predisposes to brain functional and cognitive differences could direct efforts of “cognitive restructuring” in therapy. That is, it could cue the provision of counter-environments. Lastly, individuals with identical risk genes, but different levels of environmental risk could be scanned at different developmental stages to document developmental processes associated with maladaptive cognitions and antisocial outcome.

As a final argument, I would suggest that increased knowledge about genetic influences on antisocial behavior will eventually lead to etiology-based diagnosis in antisocial behavior disorders. Current diagnostic systems rely on overt behaviors, thus yielding diagnostic categories comprising etiologically heterogeneous groups of individuals who, unsurprisingly, do not respond in a similar manner to prevention and treatment efforts.<sup>29</sup>

### SUMMARY

In this article I have argued that genetic research provides valuable information about the relative importance of genetic and environmental influences to antisocial and violent behavior. Such research also yields knowledge about the etiology of comorbidity, as well as developmental change and continuity. Perhaps most interestingly, recent twin research has demonstrated etiological differences between distinct subtypes of children with early-onset antisocial behavior, raising the possibility that successful intervention may need to integrate information about differential genetic risk. Genetic study designs are crucial for understanding the mechanisms by which environmental risk factors operate. Recent research has demonstrated environmentally mediated risk effects over and above genetic effects. Combining measured genes and environments in the study of antisocial behavior enables direct assessment

of gene–environment interaction, and has illuminated the importance of studying risk genes in conjunction with risk environments. Genetic risk may not be expressed at the behavioral level unless combined with a toxic environment. As we develop better understanding of genes–brain/cognition–behavior pathways, we will be more able to tailor individualized treatments. Diagnostic boundaries may also be revised in the light of genetic information.<sup>29</sup> Future possibilities include collection of DNA through noninvasive cheek-swabs and using genotype information to provide prevention that is tailored to suit the client’s genotype. Genetic information should never be used to dictate who gets to reproduce (aside from the obvious ethical reprehensibility of eugenics, the genetic information it is much too probabilistic to predict outcome), nor should genetic information be used to decide who gets access to treatment. However, genetic information should be used to provide people with the most effective and least invasive means of prevention and treatment—means that take into account the person’s specific vulnerabilities and strengths.

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