The Genetics of Antisocial Behavior

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Overall, the evidence from over 100 twin and adoption studies of antisocial behavior suggests that genetic factors account for about half of the variation in risk. However, behavioral genetic studies of antisocial behavior still tend to produce far-ranging estimates of heritability, suggesting that there may be important moderators of these genetic risk factors. In this review, the results of some recent behavioral genetic studies of antisocial behavior that focus on the following issues are examined: 1) developmental changes in the heritability of antisocial behaviors, 2) developmental subtypes of antisocial behavior disorders, 3) sex differences in the heritability of antisocial behavior, 4) cohort differences in the heritability of antisocial behavior, and 5) the genetics of antisocial behavior comorbidity.

Introduction

More than 100 twin and adoption studies have been conducted, most within the past decade, in an effort to determine whether, and to what extent, genetic factors play a role in the development of antisocial behavior [1••]. Overall, the evidence from this large body of data strongly suggests that antisocial behavior runs in families, in part due to the transmission of genes that increase the propensity to become antisocial. A quantitative review of 51 such studies suggests that 41% of the variation in risk for becoming antisocial is due to genetic factors, 16% is due to shared family experiences, and the remainder of the variation in risk (43%) is due to experiences specific to an individual [1••]. Compared with other psychiatric disorders such as schizophrenia [2], depression [3], alcoholism [4], and attention deficit hyperactivity disorder (ADHD) [5], for which recent behavioral genetic studies have tended to converge upon similar estimates of the magnitude of genetic influences, behavioral genetic studies of antisocial behavior still tend to produce far-ranging estimates of heritability. Perhaps this is to be expected, because antisocial behavior is a much more heterogeneous concept than schizophrenia, depression, alcoholism, and ADHD. Differences between studies in heritability estimates may be attributable to the definition of antisocial behavior used (*eg*, psychiatric, legal, or the personality trait of aggression), the method of assessment (*eg*, questionnaire, interview, or official record), or the source of information (*eg*, self-report or parent-report). Cross-study differences may provide clues to real differences in the underlying causes of the propensity to engage in different forms of antisocial behaviors. Cross-study differences may also be due to differences in the underlying causes of antisocial behaviors during different developmental periods (*eg*, childhood, adolescence, adulthood), for the two sexes, or for individuals from different cultures or eras. In other words, there may be some contexts in which the genes predisposing one to engage in antisocial behaviors are more likely to be "switched on." In this review, I present the results of some recent behavioral genetic studies of antisocial behavior that, by making within-study comparisons, may reconcile some of the observed cross-study differences.

Developmental Changes in the Heritability of Antisocial Behaviors

One factor that may account for the variability in findings from behavioral genetic investigations is the age at which antisocial acts are exhibited. Earlier reviews of delinquency and crime suggested that there were genetic influences on adult criminality, but not juvenile delinquency [6]. An influential report from the large Vietnam Era Twin (VET) Registry cohort, a sample of over 3300 twin pairs identified from military records of men who served during the Vietnam era, reported results consistent with these earlier studies [7]. Lyons *et al*. [7] obtained a lower heritability estimate for a conduct disorder (CD) symptoms scale than for a scale of adult antisocial behavior (AAB) symptoms, although heritabilities for both were significantly greater than zero (Table 1). This study is especially important because it is one of the few studies that has compared the etiology of juvenile versus adult antisocial behaviors in the same subjects. A more recent study [8] in which childhood, adolescent, and adult antisocial behaviors were assessed cross-sectionally in the same subjects obtained results consistent with Lyons *et al.* [7], but also extended these findings by demonstrating higher heritability of adolescent (ages 15–17) versus childhood (prior to age 15) antisocial behaviors, and also including women. This difference in heritability for juvenile versus adult antisocial behaviors is not always found, however. For example, in the VET Registry cohort, a legal definition of antisocial behavior was as heritable in childhood as in adulthood (Table 1) [9].

Table 1. Results from the Vietnam Era Twin study on the heritability of antisocial behavior exhibited in childhood versus adulthood by legal versus psychiatric definitions

	Heritability estimate, variation accounted for by genetic factors, %		
Definition of antisocial behavior	**Child**	**Adult**	**Child + adult**
Legal, arrested	39	30	—
Psychiatric, number of symptoms	7	43	—
Psychiatric, diagnoses	23	54	67

Data from Lyons *et al.* [7], Lyons [9], and Slutske *et al.* [25].

Measuring both juvenile and adult antisocial behaviors in the same subjects allows one to examine the extent to which the genetic risk factors for juvenile versus adult antisocial behaviors are distinct or overlapping by simultaneously fitting models to both types of antisocial behaviors. Both Lyons *et al.* [7] and Jacobson *et al.* [8] found that the genetic influences that contribute to risk for childhood antisocial behaviors also contribute to the risk for engaging in later antisocial behaviors in adulthood, and that additional genetic risk factors come into play in adolescence and adulthood. This is an important result that will need to be reexamined prospectively in future studies.

Developmental Subtypes of Antisocial Behavior Disorders

Another developmental consideration in studies of antisocial behavior concerns the age of onset or persistence of the behaviors. For example, the fourth edition of *The Diagnostic and Statistical Manual of Mental Disorders*, (DSM-IV) [10] distinguishes between two types of CD based on the age of onset of symptoms. Childhood-onset–type CD is diagnosed when symptoms are evident prior to age 10, and adolescent-onset–type CD is diagnosed when symptoms are not observed until age 10 or later. As children, individuals with childhood-onset–type CD are more likely to have a history of ADHD and oppositional-defiant disorder, to have disturbed peer relationships, and to be more physically aggressive compared with those with adolescent-onset–type CD. As adults, individuals with a history of childhood-onset type CD are more likely to persist in their antisocial behaviors and to meet the diagnostic criteria for antisocial personality disorder (ASPD) than those with adolescent-onset type CD [10–14]. These two developmental subtypes of CD may represent the outcomes of distinct etiologic mechanisms.

Moffitt [15] has labeled these divergent developmental pathways for antisocial behavior *adolescence-limited* and *life-course persistent*. Individuals with life-course persistent antisocial behavior, as the label implies, commit antisocial acts starting early in life and persist in their antisocial behavior into adulthood. According to Moffitt [15], life-course persistent antisocial behavior "has a basis in subtle dysfunctions in the nervous system" that lead to deficits in cognitive functioning and difficult temperaments. Children with a neurologic vulnerability in combination with an adverse rearing environment are at risk for developing life-course persistent antisociality. Adolescence-limited antisociality, in contrast, is described as a normal phase of development that is caused by social modeling of peer antisocial behaviors and positive reinforcement of antisocial acts. Life-course persistent antisociality has its roots in the individual and family, whereas adolescence-limited antisociality has its roots in society. A similar developmental taxonomy of antisocial behavior considers whether problems occur only during childhood or adolescence, only during the adult years, or during both phases of life. Again, persistent or continuous antisociality is considered to be etiologically distinct from transitory antisociality that is limited to a single phase of life. Based on their review of the behavioral genetic literature, DiLalla and Gottesman [16] described three etiologically distinct patterns of antisocial behavior: continuous, transitory, and late-blooming. Transitory antisociality, *ie*, antisociality that occurs only during adolescence, is hypothesized to be largely environmentally-influenced. Genetic factors are hypothesized to play a relatively greater role in the development of late-blooming and especially continuous antisociality.

There have not been good behavioral genetic investigations into the early- versus late-onset, or transitory versus persistent antisocial behavior distinction. In the VET Registry cohort, there was no difference in the heritability of early- versus late-onset CD [17]. However, because this distinction was made based on retrospective reports of age-of-onset, not much confidence can be placed upon this result. Persistent antisocial behavior was operationalized in the VET Registry cohort as meeting the criteria for both CD and AAB, that is, meeting the criteria for ASPD. ASPD was the most heritable definition of antisocial behavior in the VET Registry cohort (Table 1), suggesting that there might be merit to DiLalla and Gottesman's [16] proposal that a pattern of antisocial behavior that persists from childhood into adulthood may be more heritable than antisocial behaviors limited to just childhood or just adulthood. Again, these theories await more rigorous testing with prospective behavioral genetic investigations.

Sex Differences in the Heritability of Antisocial Behavior

A very consistent, but as yet unexplained, finding is the marked sex difference in the rates of antisocial behaviors. This prevalence difference may reflect sex differences in the causes of antisocial behaviors. So far, however, there is not convincing evidence that there are major differences in the contribution of genetic, shared family experiences, and individual-specific experiences in the etiology of antisocial behavior for men as compared with women. In their quantitative review of 51 twin and adoption studies, Rhee and Waldman [1••] obtained very similar estimates of the heritability of antisocial behavior for men (44%) and women (41%), although the difference between these estimates was statistically significant. However, when analyses were limited to those studies that included both men and women, the difference was no longer statistically significant. Miles and Carey [18••] also obtained slightly but significantly greater heritability estimates for men than for women in their quantitative review of 24 twin and adoption studies of aggression.

Studies that include male, female, and unlike-sex twin pairs can also test the degree to which the genetic risk factors (and other nongenetic familial risk factors) for antisocial behavior are distinct or overlapping by comparing the similarity of unlike-sex dizygotic twin pairs to the same-sex dizygotic pairs. Two recent studies carried-out such an analysis [8,19], and both concluded that the genetic risk factors for antisocial behavior disorders are the same for the two sexes. For example, in Slutske *et al*. [19], the dizygotic twin correlations for CD were 0.37 for male-male dizygotic twin pairs, 0.48 for female-female dizygotic pairs, and 0.34 for unlike-sex dizygotic pairs.

It has been suggested that the familial causes of antisocial behavior disorders are largely overlapping in men and women, but that women require more familial risk factors before they will become antisocial [20]. Twin studies that include male, female, and unlike-sex twin pairs can test this hypothesis as well, by comparing the risk of antisocial behavior disorder among, for example, the male dizygotic cotwins of male probands (affected individuals) with the risk to male dizygotic cotwins of female probands. A recent twin study found that 37% of the male dizygotic cotwins of male probands had a history of childhood CD, compared with 45% of the male dizygotic cotwins of female probands (Table 2) [19]. Among women, 8% of the dizygotic cotwins of male probands and 18% of the female dizygotic cotwins of female probands had a history of childhood CD. These results are consistent with the hypothesis that, on average, women with an antisocial behavior disorder have more genetic (and other nongenetic familial) risk factors than men with an antisocial behavior disorder, and so the family members of such affected women are at greater risk for becoming antisocial than the family members of affected men. Thus, part of the explanation for the ubiquitous sex difference observed for nearly all forms of antisocial behaviors may be that women require more familial risk factors (genetic and nongenetic) before they become antisocial. In other words, it may be more difficult to produce an antisocial woman than an antisocial man.

Cohort Differences in the Heritability of Antisocial Behavior

Another consistently observed phenomenon is the higher rate of antisocial behaviors among more recently born cohorts [19,21]. The same factors that have led to the higher rates of participation in antisocial activities may also have led to differences in the contribution of genetic and environmental factors. Studies that include subjects born at different periods in time can test the extent to which the contribution of genetic factors to the risk for becoming antisocial have changed with more recently born cohorts. One hypothesis is that genetic factors should become more important because adolescents and young adults in more recently born cohorts have more personal freedom and opportunities to engage in antisocial activities. For example, more recently born adolescents may be less likely to be supervised by their parents than adolescents born at earlier points in history. Thus, it should be more likely that the genetic predisposition for becoming antisocial will be actualized in the more recently born than in the earlier-born adolescents.

Two recent studies, one of men in the United States born between 1940 and 1974 [21], another of men and women in Australia born between 1902 and 1964 [19], have examined cohort differences in the genetic and environmental contributions to the risk for CD. Surprisingly, given the broad age ranges studied, neither study was able to detect either an increase or decrease in the contribution of genetic factors with more recently born cohorts, although the US-based study found that the contribution of shared family experiences was more important among the more recently born men. A more rigorous approach to studying this question would be to study individuals born in different years but assessed at the same age, so that the effect of birth year and age will be unconfounded with each other. So far, no behavioral genetic studies have used this approach to examine cohort differences.

The Genetics of Antisocial Behavior Comorbidity

Once it is established that there are genetic influences for a disorder, the magnitude of the heritability may not be, in itself, particularly useful. The heritability estimate obtained from behavioral genetic investigations can be used, however, as a guide in molecular genetic investigations to understand the proportion of total genetic variation any given susceptibility gene accounts for, and whether there is any remaining genetic variation to be explained. Molecular

Table 2. Results from the Australian Twin Registry study on the risk of conduct disorder among the dizygotic cotwins of male and female probands with conduct disorder

Cotwin sex	Proband sex	Population prevalence, %	Risk to cotwin, %	Risk ratio
Male	Male	19	37	1.9
Male	Female	19	45	2.4
Female	Male	3	8	2.7
Female	Female	3	18	6

From Slutske *et al.* [19].

genetic investigation of antisocial behaviors has not been pursued as vigorously as the search for genes associated with other psychiatric syndromes, perhaps because of the controversy surrounding the genetics of crime. However, genes do not cause antisocial behaviors directly, but rather contribute to more general dispositions that increase the likelihood of an individual engaging in antisocial behaviors. Thus, molecular genetic investigations of other related syndromes may eventually indirectly uncover susceptibility genes for antisocial behaviors.

Behavioral genetic studies of antisocial behavior that also include measures of other domains can explore the extent to which the genetic factors that increase the risk for antisocial behavior are distinct or overlapping with those for other associated traits. Recent work in this area suggests that the genetic risk for antisocial behavior is significantly associated with the risk for ADHD [22], alcoholism [23,24], and pathologic gambling disorder [25]. Young *et al.* [26•] found that 22% of the variation in CD symptoms could be explained by a higher level trait they termed *behavioral disinhibition*, which was highly heritable (84% of the variation in the trait of behavioral disinhibition was due to genetic factors) and also explained to varying degrees the variation in substance experimentation, ADHD symptoms, and the personality trait of novelty-seeking. Thus, progress in identifying susceptibility genes for the disposition of behavioral disinhibition will likely be informative for understanding the genetics of antisocial behaviors.

Conclusions

Recent meta-analytic reviews have been extremely valuable in bringing order to the diverse array of studies comprising the behavioral genetic literature on antisocial behavior. Yet it is sometimes difficult to disentangle the causes of heterogeneity across studies because they can differ on so many characteristics. In this brief review, the value of within-study comparisons was highlighted with respect to 1) developmental changes in the heritability of antisocial behaviors, 2) developmental subtypes of antisocial behavior disorders, 3) sex differences in the heritability of antisocial behavior, 4) cohort differences in the heritability of antisocial behavior, and 5) the genetics of antisocial behavior comorbidity. Although there are many twin and adoption studies of antisocial behavior, only a minority of studies provide information relevant to these five topics. More research in which within-study comparisons (*eg*, of the heritability of antisocial behavior among men versus women or for early-onset versus later-onset antisocial behavior) is required in order to draw firmer conclusions about the issues raised in this review. Such studies will provide a richer database concerning the similarities and differences of the genetic effects on antisocial behavior risk in differ populations and contexts.

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