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Genetics and Crime

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The idea that inherited genetic predispositions may underlie the risk for engaging in criminal behavior is not exactly new. Perhaps most convincingly, several adoption studies in the 1970s and 1980s provided powerful evidence that having an incarcerated birth parent raised one's own risk of earning a criminal conviction as an adult, even if reared by pro-social, law-abiding – but genetically unrelated – foster parents. This remarkable finding was replicated in adoptive cohorts across cultures, including two Scandinavian studies (Cloninger *et al*., 1982; Mednick *et al*., 1984) as well as in the United States (Cadoret *et al*., 1983). Based on these adoption studies, the genetic effect on criminal outcomes appears important for both sexes, although individual genetic risk is typically more extreme for female than male offenders (Baker *et al*., 1989). Heritable influences also differ in these studies according to the type of crimes committed, with petty, non-violent offending showing larger genetic influence than violent offenses (Mednick *et al*., 1984).

Most importantly, however, these early adoption studies shared one other remarkable and profound result, which is that the genetic risk for criminal behavior could be exacerbated by adverse environmental circumstances, such as coming from a low socioeconomic background (Van Dusen, 1983), or being raised in a family with at least one criminal adoptive parent (Cloninger *et al*., 1982; Mednick *et al*., 1984). Such effects fall under the realm of genotype by environment (GxE) interactions, and highlight the complexity of the genetic and environmental effects in criminal outcomes.

The provocative findings from these early adoption studies have since sparked numerous lines of research attempting to replicate and further refine our understanding of both genetic and environmental causes of crime and violence. A plethora of twin and adoption studies subsequently confirmed the genetic effect on criminal outcomes, and on the wider constructs of antisocial behavior (see Rhee and Waldman, 2002) and externalizing behavior disorders. Although dozens of studies have replicated the genetic effect in antisocial behavior across a variety of cultures, the genetic

influences have been almost entirely unspecified, with little understanding about how many genes, their location in the human genome, or the specific environments or experiences that lead to gene expression.

So what is new in research on genetics of crime? Current genetic research on antisocial behavior – including criminal offending – aims to specify the nature of both genetic and environmental influences, and how they may interact with one another to lead to criminal outcomes. This is being done in a variety of ways, including molecular genetic studies attempting to identify specific genes which increase risk for criminal behavior or its correlates such as impulsivity, risky decision making, and aggression, as well as investigations of biological and social risk factors and how their relations to crime may be mediated by genes and environment. Our goal in this chapter is to review the ways in which we have begun to unpack the black boxes of genetic and environmental influences in antisocial behavior, with a focus on studies that include criminal offending. We first briefly review the evidence for (anonymous) genetic influences and gene–environment interactions in antisocial behavior, including the various ways in which these effects have been shown to vary – across type of crime, gender, and development. This is followed by a review of recent studies attempting to identify specific genes and the factors that may potentially modify their expression.

UNSPECIFIED GENETIC AND ENVIRONMENTAL EFFECTS ON CRIMINAL BEHAVIOR

The strongest evidence for a genetic effect on criminality comes from the early twin and adoption studies conducted in several countries, including the United States (Cadoret *et al*., 1995), Sweden (Sigvardsson *et al*., 1982), Denmark (Mednick *et al*., 1984), and Norway (Torgersen *et al*., 1993). These effects are especially strong for crimes against property, including theft, vandalism, and property damage. Twin concordance for convictions is consistently greater for genetically identical (monozygotic) than for non-identical/fraternal (dizygotic) twin pairs for property crimes such as vandalism and theft (Cloninger and Gottesman, 1987). For adopted individuals, there is increased risk for property crime convictions when his or her birth parent evidenced a similar conviction, further suggesting the importance of genetic influences on property offending. In the absence of birth parent convictions, however, there is little or no increase in risk when raised by adoptive parents with property crime convictions (Mednick *et al*., 1984), indicating little importance of shared family environment, at least when genetic risk is low. Environmental influences on non-violent criminality thus appear largely non-familial and specific to the individual rather than shared by relatives living together.

Further, it is well documented that males are much more likely than females to engage in most forms of criminal behavior (Junger-Tas *et al*., 1994; Moffitt *et al*., 2001; Rutter *et al*., 2003). This sex difference is widest for violent offending (Rutter *et al*., 1998; Smith and Visher, 1980), and narrowest for drug and alcohol related crimes (Moffitt *et al*., 2001). Although males are arrested and convicted far more often than females the heritability of non-violent criminality is comparable for the two sexes (Baker *et al*., 1989; Rhee and Waldman, 2002). Twin studies have shown

greater identical (monozygotic) than fraternal (dizygotic) concordance for non-violent criminal convictions in both male and female same-sex pairs. Nonetheless, there is some evidence for sex-limited genetic effects, whereby different genetic or environmental factors may be important in males and females, in that opposite-sex fraternal (dizygotic) twins are often less similar than same-sex fraternal twin pairs (Cloninger and Gottesman, 1987).

The broader construct of antisocial behavior – which includes criminal offending, as well as aggression – also shows substantial genetic influence. In a meta-analysis combining effect sizes in 51 twin and adoption studies, Rhee and Waldman (2002) reported a heritability estimate of 41 per cent, with the remaining 59 per cent of variance being due to environmental factors. Interestingly, when comparing results for various definitions of antisocial behavior, only criminal offending appeared to be influenced by both additive genetic effects and non-additive genetic effects – possibly due to genetic dominance and epistatic interactions between genes – based on a pattern of results whereby, on average, identical (monozygotic) twin correlations are more than twice the value of fraternal (dizygotic) twin correlations, and also that biological parent–offspring correlations are less than fraternal twin correlations. Such non-additive genetic effects could arise if one or more high risk alleles act in a recessive fashion, or if certain alleles at one locus affect gene expression at other loci (epistasis).

One intriguing aspect of the literature on genetics and crime is that the strong and consistent genetic influence seen for property offending does not hold true for violent criminal convictions. None of the major adoption studies in Scandinavia or the United States found any elevated risk for violent convictions as a function of either biological or adoptive parent criminal offending, although one early twin study did find greater identical (monozygotic) than fraternal (dizygotic) concordance for violent convictions (see Cloninger and Gottesman, 1987). This pattern of twin, but not parent-offspring, similarity for violent criminal behavior suggests the possibility of non-additive genetic effects due to dominance or epistasis, which would result in increased resemblance for siblings (and twins), but not for parents and offspring. Thus, there may be genetic risk for violent crimes such as murder and rape, which may stem from rare recessive genes, or specific combinations of alleles that do not appear in studies of vertical transmission across generations.

Developmental effects

How early in life do genetic influences for criminal offending appear? Limited access to official court records for crimes committed prior to age 18 have made it difficult to investigate the etiology of law-breaking behaviors in youth in the same manner as in the large twin and adoption studies. Nonetheless, a large literature exists for studies using parent and teacher ratings of children and adolescents and youth self-report methods, which aim to understand early rule-breaking and other behavior problems in childhood and adolescence that may give way eventually to law-breaking behaviors in adulthood.

In the aforementioned meta-analysis review of twin and adoption studies of the wider construct of antisocial behavior (Rhee and Waldman, 2002) genetic influences appear to be at least as important (if not more so) in children and adolescents

compared to adults. In fact, their meta-analysis suggested a significant decrease in genetic influences across age, although sample differences in age across studies are confounded with the definition and method of assessment of antisocial behavior. Studies of younger children tend to rely more often on parent and teacher ratings of children's aggressive and rule-breaking behaviors, while studies of adults are more apt to use self-report or official records of convictions. Thus, it is difficult to know exactly whether and how genetic influences on criminal offending might emerge across development. This highlights the importance of using multi-method assessments in longitudinal studies, in which narrow age bands are studied. A few such longitudinal studies of antisocial behavior have begun to shed more light on the developmental course of genetic etiologies.

In our own longitudinal twin study of antisocial behavior we have found a high heritability (over 90 per cent) of a common view obtained by ratings of childhood antisocial behavior from both the parents and teachers, as well as through self-reports from the children themselves (Baker *et al*., 2007). In addition to the genetic effects common to all three reporters of the child's antisocial behavior, there appear to be additional genetic influences specific to a given reporter (Baker *et al*., 2007; Baker *et al*., 2008). The genetic influences also appear to be quite stable from childhood to adolescence both for a general antisocial behavior factor (Baker *et al*., 2009) as well as narrower measures of proactive and reactive aggression (Tuvblad *et al*., 2009).

Age of onset is often considered as a moderator of genetic effects in criminal behavior. Official statistics and victim surveys consistently show that adolescents account for a large proportion (approximately one fourth to one third) of all crimes. From self-report studies we also know that between 50 and 80 per cent of all juveniles participate in antisocial behavior at some time during childhood or adolescence. However, a small proportion of all antisocial individuals (5–7 per cent) accounts for approximately half of all antisocial acts (Loeber and Farrington, 1998; Rutter *et al*., 1998; Vermeiren, 2003). Most of the antisocial acts committed are theft-related, and only a small proportion is aggressive and violent (Farrington and Loeber, 2000). There are also some well-established developmental patterns in antisocial behavior. For example, individuals with an early age of onset are more likely to persist in antisocial behavior (Loeber and Farrington, 2000; Robins, 1978; Simonoff *et al*., 2004; Stouthamer-Loeber and Loeber, 2002; Tremblay *et al*., 1994). Finally, antisocial behavior has been found to increase in early adolescence, to peak in mid-adolescence, and then to drop sharply in young adulthood (Moffitt, 1993). The fact that antisocial behavior peaks in adolescence and that age of onset is related to persistence is the starting point for a developmental taxonomy of antisocial behavior that differentiates the most deviant over the life course from those likely to show temporary difficulties during adolescence. The theory proposes that 'life-course persistent' and 'adolescent-limited' antisocial behavior differs in terms of etiology, developmental course, prognosis, and classification of behavior as pathological versus normative. Life-course persistent antisocial behavior is thought to have a neuro-developmental origin, and to begin at a very young age and continue from adolescence into adulthood. In contrast, adolescence-limited antisocial behavior is thought to be limited to adolescent years and to be more influenced by social peer pressure (Moffitt, 1993). DiLalla and Gottesman (1989) had previously suggested a similar theory, but they referred to life-course persistent as continuous antisocials, and 'adolescent-limited' antisocial behavior as transitory delinquents. They further

suggested a third group called 'late bloomers', who are thought to begin their offending in adulthood (DiLalla and Gottesman, 1989). Genetic influences are generally thought to contribute more to persistent antisocial behavior, than to adolescent onset or transitory antisocial behavior (Moffitt, 2005a). Only a few behavioral genetic studies have reported findings that can be interpreted in support of these developmental theories, or at least in support of different aspects of these theories. A recent study showed that a common genetic factor was influencing antisocial behavior in males beginning at age ten and through young adulthood, hence, reflecting persistent antisocial behavior. Whereas a common shared environmental factor was found for adolescent and adult antisocial behavior, this was interpreted by the authors to reflect adolescent onset or transitory antisocial behavior (Silberg *et al*., 2007).

Genetics of violent vs. non-violent behavior in children

Although the distinction between violent and non-violent *criminal offending* is more difficult to make in children and adolescents compared to adults, a number of studies have compared the genetic etiologies for different forms of aggressive and antisocial behavior in younger subjects. Researchers often delineate between overt, physical and possibly violent behaviors (referred to as 'aggression') and covert antisocial behavior which includes property damage and theft (referred to as 'delinquency') (Achenbach, 1991; Frick *et al*., 1993; Loeber and Hay, 1997). Longitudinal studies have shown differences in violent and non-violent behavior, with violent and aggressive behavior generally being more stable across time, compared with non-violent and delinquent behaviors (Stanger *et al*., 1997; Tolan and Gorman-Smith, 1998). Further support for this distinction is provided by twin studies, in that aggressive and violent behavior has been found to be highly heritable (Edelbrock *et al*., 1995; Eley *et al*., 1999; Ghodesian-Carpey and Baker, 1987; Hudziak *et al*., 2003), whereas non-violent behavior shows a roughly equal influence of genes and shared environment (Bartels *et al*., 2003; Edelbrock *et al*., 1995; Eley *et al*., 2003). Twin studies have also demonstrated that aggressive and violent behavior and non-violent behavior share common genetic factors and environmental influence, but there are also genetic and environmental factors unique to each type of behavior (Button *et al*., 2004; Gelhorn *et al*., 2006).

Peer and sibling influences: partners in crime?

Numerous studies have shown that to have antisocial peers or siblings is a strong risk factor for antisocial behavior (Farrington and Loeber, 2000; Hawkins *et al*., 1998). In other words, antisocial individuals tend to have antisocial friends. This could be due to a selection process, but it could also be explained by an influence process. Regardless of the causal direction involved, most antisocial activities are not perpetuated by individuals acting alone, but rather are undertaken together with others. Differential association theorists argue that antisocial behavior is largely learned through personal interactions in the peer group (Sutherland and Cressey, 1978). Through interaction with others, individuals learn the values, attitudes, techniques, and motives for criminal and antisocial behavior. Peer influences have a large impact during adolescence (Lipsey and Derzon, 1998), and this is also when the nature of the peer group changes and an individual tends to spend more time with his/her peers, compared with a younger child (Rutter *et al*., 1998). Related to this is the fact

that antisocial behavior is most prevalent in adolescence. It increases in early adolescence, reaches its peak in mid-adolescence, and then largely disappears by young adulthood (Moffitt, 1993).

Regardless, peers and siblings may influence one another and the effect of such phenotypic reciprocal interaction can be investigated using a twin sample. Siblings may either imitate each other's behavior, that is, the behavior in one twin leads to the behavior in the other twin. Or they may take on opposite or competing behaviors. In other words, the behavior of one twin has an inhibitory effect on the behavior of the other twin (Carey, 1986, 1992). This type of competing or contrast interaction effect has been repeatedly found in studies investigating symptoms of attention-deficit hyperactivity disorder (ADHD) (van Beijsterveldt *et al*., 2004; Vierikko *et al*., 2004). However, many of these studies used parent reported data, which make it difficult to determine if the observed interaction effect is a true contrast effect or if it is due to rater bias. An imitation effect is confounded in the shared environment, and a contrast effect is confounded within dominant non-additive genetic effects (Rietveld *et al*., 2003). However, if there is sibling interaction, variance differences between monozygotic and dizygotic twins are expected.

Several studies investigating antisocial behavior have found a positive interaction between twins, indicating that siblings sometimes co-operate and 'become partners in crime' (Carey, 1992; Rowe, 1983; Rowe *et al*., 1992). An early study by Rowe (1983) found that genetic, as well as shared environmental influences were important in the development of adolescent antisocial and delinquent behavior. Further analyses showed however, that monozygotic twins were more likely to commit delinquent acts together, compared to dizygotic twins. It was therefore concluded that since twins may influence one another, this may partly explain the shared environmental influences. In another study, Carey (1992) investigated sibling interaction effects for antisocial behavior in a large set of Danish twins. The twins were followed through official police and court records. A modest heritability and positive sibling interaction effect was found, indicating that the combination of heritability and sibling imitation processes contribute to liability toward antisocial and criminal behavior.

GxE INTERACTIONS IN CRIMINAL BEHAVIOR

The complex interplay between genes and environment must also be considered, in addition to their main effects in criminal behavior. Genetic influences, for example, can be exacerbated through certain experiences or exposure to specific circumstances. Likewise, individuals with different genotypes may respond differently to the same environmental exposure. Conversely, some environments may serve as protective factors, such that the genetic effects on criminal outcomes are reduced or eliminated for some individuals. The dependence of genetic effects on different environments or vice versa is referred to as gene–environment interaction. Although gene–environment interactions have become of particular interest in recent research on psychopathology (Moffitt *et al*., 2005; Rutter *et al*., 2006), these complex effects have long been known to occur for criminal behavior.

There are a number of ways in which gene–environment interactions may be tested in genetically informative studies. The classic approach is based on analysis

of variance (ANOVA) in adoption studies in which mean levels of criminal outcomes (e.g., conviction rates) are examined as a function of genetic risk (i.e., criminal background in biological parents) and environmental risk (i.e., criminal background in adoptive parents). Some of the strongest evidence for gene–environment interaction in criminal behavior comes from the early adoption studies using this approach. It has been repeatedly found that genetic predisposition for crime (e.g., crime or psychopathology in biological parents) combined with a high risk environment (i.e., adoptive home environment) leads to greater risk for criminal offending the offspring than what would be expected from the (additive) main effects of genes and environment. This synergistic effect (illustrated in Figure 1.1) has been replicated for property offending in each of the major adoption studies of criminal behavior (Bohman *et al*., 1982; Cadoret *et al*., 1983, 1995; Cloninger *et al*., 1982; Crowe, 1974; Mednick *et al*., 1984).

A similar gene–environment interaction has been reported for childhood conduct problems, using a measured environmental risk factor approach in twins. Jaffee *et al*. (2005) found that measured environmental risk (childhood maltreatment) exacerbated the genetic risk for conduct problems (based on the co-twin's conduct problems). That is, the (environmental) effect of maltreatment on the risk for conduct problems was greater among those children who had higher genetic liability for conduct disorder, compared to those who had a low genetic liability. This finding parallels the gene–environment interaction consistently reported in the early adoption studies of criminal offending, in which genetic effects were more severe in adverse environments. This gene–environment interaction can also be framed in a more positive manner, such that favorable genotypes may provide the greatest protection against problem behaviors in adverse circumstances such as maltreatment during childhood (Jaffee *et al*., 2005).

Another major approach for studying gene–environment interactions is through the estimation of genetic *variance* (and its relative importance, i.e., heritability)

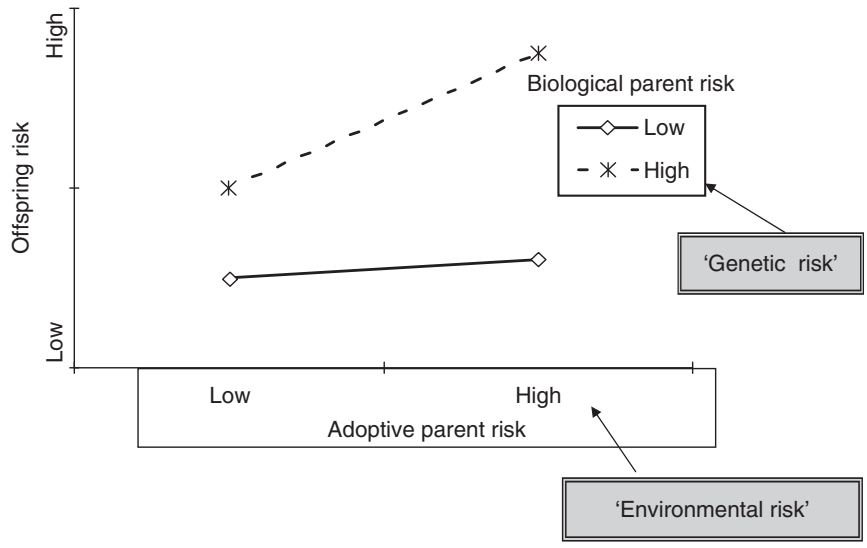

Figure 1.1 GxE interaction.

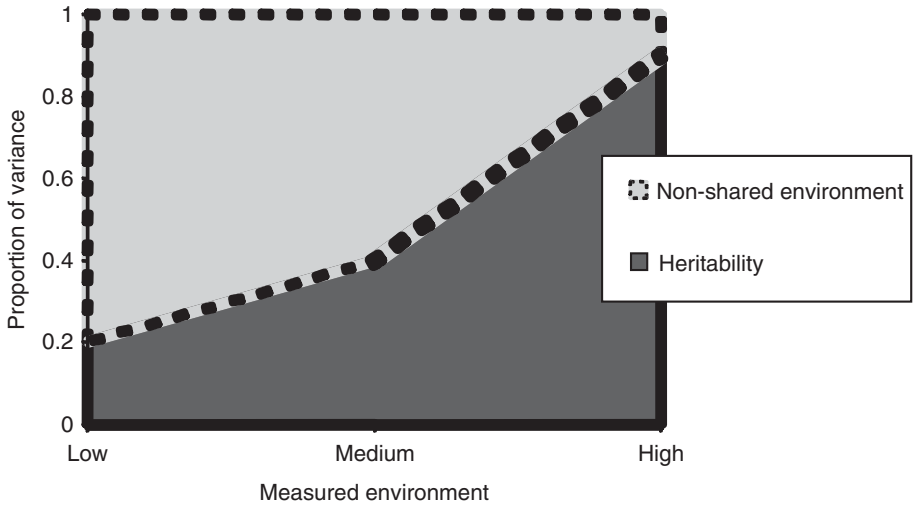

Figure 1.2 GxE Interaction: differential heritability across measured environments.

across a range of different environments. For example, genetic influences on the propensity toward criminal offending could be of lesser or greater importance for individuals raised in favorable versus impoverished surroundings, such as neighborhood or schools. Figure 1.2 illustrates this type of gene–environment interaction, whereby heritability increases across the favorability of the environment. This approach to studying gene–environment interactions does not require adoption designs, but can be made in twin studies as well.

One recent twin study using this differential heritability across environments approach found that relative importance of genetic influences on antisocial behavior in 16 to 17-year-old boys to be *larger* for more advantaged (h^2 = 0.37) than less advantaged (h^2 = 0.01) neighborhoods in Sweden (Tuvblad *et al*., 2004). Family environmental effects, on the other hand, were of greater importance in less advantaged neighborhoods (c^2 = 0.69) than in more advantaged ones (c^2 = 0.13). Although the differences across environments were less marked for girls, the same pattern was found with greater heritability (h^2 = 0.69) and smaller shared environment (c^2 = 0.06) for more advantaged environments compared to relative effects in less advantaged neighborhoods (h^2 = 0.61, c^2 = 0.16), see Figure 1.3.

At a glance, the nature of this gene–environment interaction – with larger genetic variance in favorable environments – may seem at odds with the well-replicated finding using the ANOVA approach in adoption studies, in which larger genetic effects are evident in more adverse environments. Certainly the studies using the different approaches to study gene–environment interactions differ in the measures of antisocial behavior (i.e., criminal offending vs. the broader construct of aggression and delinquency), which might explain some of the discrepancy. More importantly, however, the two approaches for studying gene–environment interaction differ in that the differential heritability approach (e.g., Tuvblad *et al*.) focuses on *variance* of antisocial outcomes, while mean levels of deviant behavior are the focus of the ANOVAs in the adoption studies (e.g., Mednick *et al*., 1984). What the adoption studies demonstrate is that the greatest overall *incidence* of criminal offending

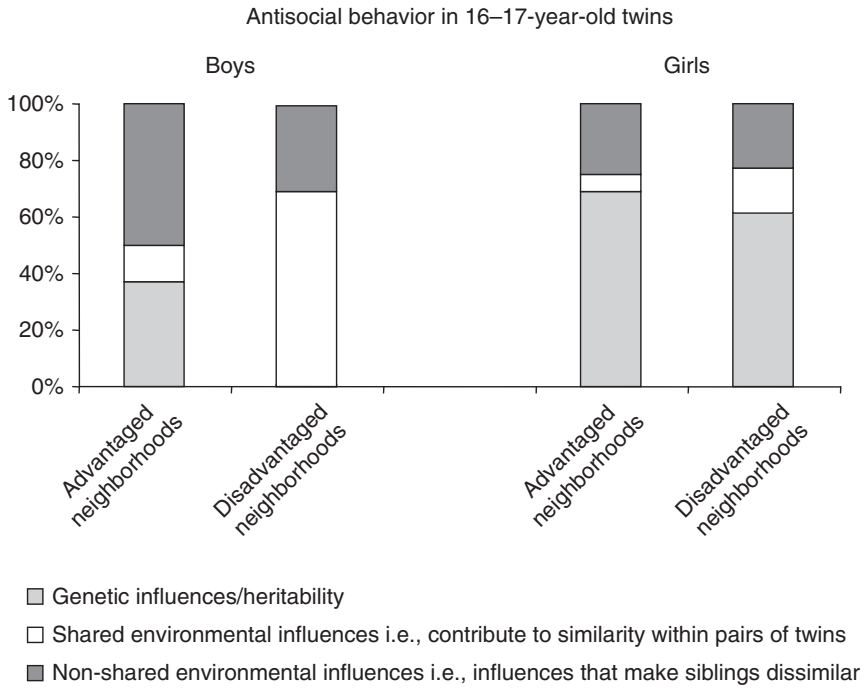

Figure 1.3 The heritability of antisocial behavior differs with socioeconomic status. (From Tuvblad *et al.* (2006) *Journal of Child Psychology and Psychiatry*.)

occurs when both genetic and environmental adversity occur together, while the twin study findings such as those of Tuvblad *et al*. suggest that individual differences in deviant behavior are explained by genetic factors differently according to the environmental circumstances. The latter findings are consistent with Raine's (2002a) 'social push hypothesis', in which biological risk factors (which includes genetic predisposition) appear less influential (i.e., explain less variance) in individuals from adverse environments, since environmental risks *push* him or her toward antisocial behavior and biological risk factors are masked in these disadvantaged individuals. Conversely, individuals from favorable environments lack the social push, and effectively have less environmental variation, so that biological risk factors are more likely to be revealed and thus explain individual differences in deviant outcomes (see Raine, 2002b; Tuvblad *et al*., 2004).

In spite of the well-replicated findings of both main effects for genetics and gene–environment interaction in criminal behavior, for many years both the genetic and environmental risk factors have remained anonymous. That is, the nature of the genetic influences are unspecified in the classic approach, with no information being provided about the number of genes involved, their location in the genome, or the specific alleles which contribute to the highest risk for criminal offending. Similarly, environmental risk factors as defined by adoptive parent criminal background in no way specifies the child's experiences or how these may lead to criminal offending. With advances in molecular genetics and gene identification methods, however, research is beginning to give identity to both genetic and environmental risk factors for criminal and other antisocial behavior, as discussed next.

UNPACKING THE BLACK BOXES OF GENES AND ENVIRONMENT

The holy grail in genetic research on pathological behavior in general is twofold: (1) to identify specific allelic variations that explain observable phenotypic variation or an increase in the risk for deviant outcomes, and (2) to understand the conditions under which such high risk alleles have the greatest or least effect. With the rapid technological developments in molecular genetics and methods for genotyping large groups of individuals using hundreds of DNA markers, researchers in the social sciences are becoming immersed in the search for the holy grail in both normal and abnormal trait variation in a wide array of behaviors. Although to date there are no genome wide studies of criminal behavior *per se*, there are a handful of studies that have specifically focused on antisocial behavior (including criminal offending) and several others studying many correlates of criminal outcomes. In spite of the surge of interest in 'gene identification' studies of human behavior, this research is still in its infancy, particularly with regard to studies of antisocial behavior and especially criminal offending. The few studies to date are reviewed here.

Other attempts to unpack both the genetic and environmental black boxes in antisocial behavior involve the study of measured risk factors, which may include both biological and social variables which predict antisocial outcomes. The extent to which these risk factor–antisocial behavior outcomes may be genetically and environmentally mediated can be understood in the context of genetically informative designs, such as twin and adoption studies, even when DNA markers are not studied. The risk factor approach is tantamount to studying endophenotypes, which are other measurable variables with significant genetic covariation with antisocial outcomes. An endophenotype is a type of biomarker, but with more strict criteria. An endophenotype has to be associated with the outcome in the population, it has to be heritable, and to be found in an individual regardless of the outcome it manifested. Also, an endophenotype is expected to be found in non-affected family members at a higher rate than in the general population (Gottesman and Gould, 2003). Genetically informative studies of risk factors for criminal offending are thus also reviewed following the review of gene identification studies.

Gene identification

In spite of the overwhelming evidence for genetic influences in criminal and other antisocial behaviors, research attempting to identify specific genes that increase risk for criminal offending and the biochemical pathways between genes and behavior is still relatively rare. The first study to demonstrate a link between a specific genotype and antisocial behavior (Brunner *et al*., 1993) investigated a large, multigeneration Dutch family that had several members (particularly males) who were prone to violent, aggressive, and impulsive behavior, with histories of fighting, arson, attempted rape, and exhibitionism. Through analyses of DNA samples in this large pedigree, it was shown that the aggressive males shared a mutant form of the gene that codes for the enzyme MAO-A (monoamine oxidase A). MAO-A breaks down neurotransmitters (including serotonin, noradrenaline, and dopamine) which are known to be important in impulsive behaviors and reward dependence. The mutant alleles inherited by the

aggressive and antisocial males, however, resulted in deficient production of the MAO-A enzyme, which in turn led to large quantities in the blood and ineffective functioning of the neurotransmitters necessary for proper impulse control and reward pathways in the brain (Brunner *et al*., 1993). This finding of increased aggression being associated with MAO deficiency produced by a genetic mutation in the MAO-A allele coincides with animal research using knockout strains of mice (Shih, 2004), where the same finding has been well-replicated. Moreover, associations between aggression with neurotransmitters serotonin, dopamine, and noradrenaline have been found in both humans and animals (Arce and Santisteban, 2006). Although the main effects of the MAO-A mutation have not replicated yet in any other large human pedigrees, this genetic defect remains the first such link to aggressive behavior in humans.

It has also been suggested that environmental factors may moderate the effects of the MAO-A mutations on aggression, which one could easily predict given the well-replicated finding of gene–environment interactions in criminal behavior in adults and conduct problems and antisocial behavior in youth. One highly cited finding illustrates such a gene–environment interaction in antisocial behavior using a measured gene/measured environment approach (Caspi *et al*., 2002). A functional polymorphism in the MAO-A gene was found to increase the risk for conduct problems (including violence) in adolescent males, but only in conjunction with early childhood maltreatment. More specifically, maltreated boys (i.e., with adverse environmental) who had the adverse genotype conferring MAO deficiency (due to inheritance of the mutant allele) were more likely to develop conduct disorder as a youth, and antisocial personality disorder and violent criminal behavior as adults (see Figure 1.4). The fact that the main effect of the MAO-A mutation as found in Brunner *et al*. (1993) was not found in the Caspi *et al*. (2002) study underscores the

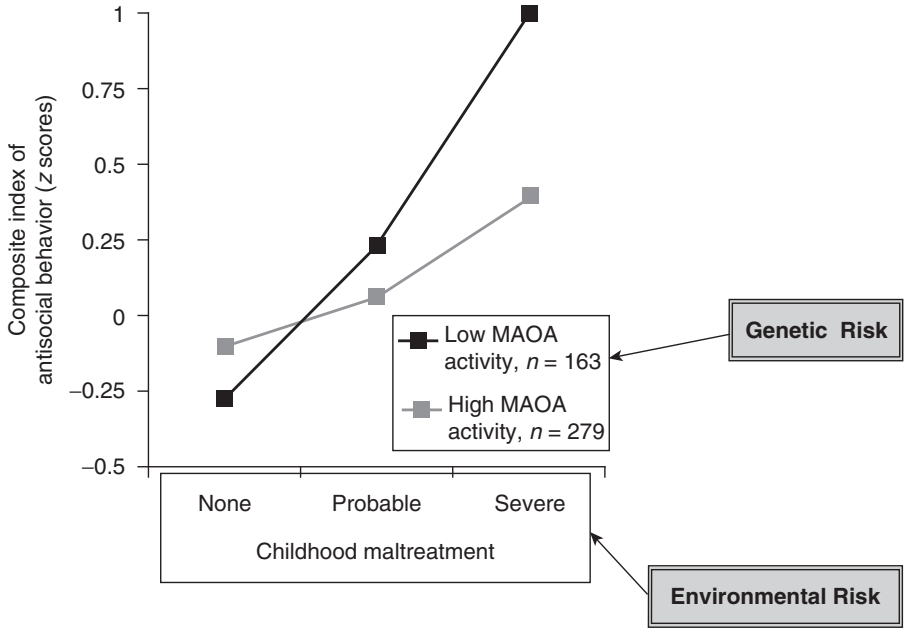

Figure 1.4 Genotype (MAO-A) x environment (maltreatment) interaction in antisocial behavior. (From Caspi *et al.* (2002) *Science*.)

importance of investigating specific genetic effects under a variety of environmental circumstances in order to fully understand the risk for criminal offending and other antisocial behavior. The Caspi *et al*. (2002) finding is particularly intriguing since it was one of the first studies to illustrate the well-replicated GxE interaction in criminal behavior using a measured gene/measured environment approach. To date, there has only been a few replications of this interesting finding (Foley *et al*., 2004; Kim-Cohen *et al*., 2006; Nilsson *et al*., 2006) and one published failure to replicate (Haberstick *et al*., 2005). For instance, Kim-Cohen and colleagues (2006) found that the MAOA polymorphism moderated the development of psychopathology after exposure to physical abuse in a sample of 975 seven-year-old boys. This finding was extended to the maltreatment experience closer in time compared with previous work by Caspi *et al*. (2002), and therefore the possibility of a spurious finding by accounting for passive and evocative gene–environment correlation could be ruled out. It should be mentioned that passive gene–environment correlation refers to the association between the genotype a child inherits from her parents and the environment in which the child is raised, and evocative gene–environment correlation occurs when an individual's (heritable) behavior evokes an environmental response. Moreover, the authors also conducted a meta-analysis. Across five included studies (Caspi *et al*., 2002; Foley *et al*., 2004; Haberstick *et al*., 2005; Kim-Cohen *et al*., 2006; Nilsson *et al*., 2006) the adverse mental health problems were greatest for maltreated boys with the genotype conferring low MAOA activity. It was concluded that these findings provide strong evidence suggesting that the MAOA gene influences vulnerability to environmental stress, and that this biological process can be initiated early in life. However, these findings need to be replicated in samples including females.

A gene–environment interaction has also been identified for variation in the *age of onset* for criminal offending using a measured gene/measured environment approach. Based on data from the National Longitudinal Study of Adolescent Health (AdHealth), DeLisi *et al*. (2008) found that polymorphisms in genes related to the neurotransmitter dopamine were associated with age of first police contact and arrests, but only for youth from *low risk* family environments. More specifically, among those adolescents with a history of criminal offending, those at greatest risk for later onset were those with the A1 allelic form of the DRD2 gene, in combination with favorable home environments as defined by maternal attachment, involvement, and engagement (DeLisi *et al*., 2008). It is important to emphasize that the DeLisi *et al*. (2008) finding involves the age of onset of first police contact, and not the overall risk for offending vs. not offending. However, different forms of the DRD2 allele have demonstrated associations with criminal victimization (Beaver *et al*., 2007) and age of first sexual intercourse (Miller *et al*., 1999), as well as normal personality variation (Munafò *et al*., 2003). Other studies have also found gene–gene interactions between DRD2 and DRD4 in predicting conduct disorder in childhood and criminal offending in adults (Beaver *et al*., 2007). Still, the finding of enhanced risk for later onset criminal offending as a function of high genetic risk combined with low environmental risk is contrary to predictions from other developmental models of antisocial behavior. Both Moffitt (2005) and Lahey *et al*. (1999) have suggested that early-onset forms should be more pervasive over time and more influenced by genetic factors than late-onset, transient forms of delinquency, yet the

converse pattern was found by DeLisi *et al*. (2008). Nonetheless, the DeLisi *et al*. (2008) findings are particularly interesting in that they also demonstrate a GxE interaction in criminal offending using specific genetic markers and well-defined measures of the environment.

Further, several behaviors and disorders are associated with antisocial and criminal behavior; these include, for example, attention deficit hyperactivity disorder (ADHD) and drug and alcohol abuse. A few genes related to some of these associated behaviors and disorders have been identified. Hence, some of these genes could either be considered as candidate genes for antisocial and criminal behavior or as predictors. A recent study found evidence of linkage to a region of chromosome 7, which appears to contain genes conferring risk to externalizing behaviors, including alcohol, drug dependence, conduct disorder, antisocial personality disorder, novelty and sensation seeking (Dick *et al*., 2008). It may be that a broader spectrum of externalizing behaviors and disorders should be studied in order to identify susceptibility genes. As previously mentioned, ADHD often co-occurs with antisocial and criminal behavior (Hechtman, 1999) and longitudinal studies show that ADHD leads to antisocial behavior not the other way around (Kutcher *et al*., 2004). Using our longitudinal sample we were able to show that the covariation between antisocial behavior and ADHD is in part explained by common genes (Tuvblad *et al*., 2009). Related to this, a recent study by Caspi and colleagues found evidence that the COMT valine/methionine polymorphism at codon 158 (COMT Val158Met) was associated with phenotypic variation among children with ADHD. Valine/valine homozygotes also had more symptoms of conduct disorder, were more aggressive, and were more likely to be convicted of criminal offenses. However, COMT was not a susceptibility gene for aggression and antisocial behavior, rather COMT influenced the phenotypic variation in ADHD and predicted which children would engage in antisocial behavior (Caspi *et al*., 2008).

Risk factors (endophenotypes) for criminal behavior

Empirical research has established a number of risk factors that are associated with the risk for engaging in criminal behavior. These factors include broad biological and social risk factors (e.g., low resting heart rate) (Ortiz and Raine, 2004); prenatal factors (e.g., fetal exposure to alcohol, smoking and/or malnutrition) (Raine, 2002a); personality factors (e.g., impulsivity, callous and unemotional traits, stimulation-seeking, fearlessness) (Lahey *et al*., 2003; Raine, 2002a; Viding *et al*., 2005); family factors (e.g., parental criminality, poor child rearing practices, parental substance use, low socioeconomic status, maltreatment) (Caspi *et al*., 2002; Farrington *et al*., 1996; Lipsey and Derzon, 1998; Loeber and Dishion, 1983); school related factors (e.g., poor academic performance, weak bonding to school) (Hawkins *et al*., 1998; Loeber and Farrington, 2000); peer factors (e.g., delinquent peers and siblings, gang membership, peer rejection) (Farrington and Loeber, 2000); and contextual factors (e.g., neighborhood poverty, availability of weapons) (Beyers *et al*., 2001; Brooks-Gunn *et al*., 1993; Sampson *et al*., 1997).

Even though it is well-known which risk factors are related to criminal behavior, less is known about the underlying mechanisms of how these factors are related to the development of criminal behavior. This has led several researchers to conclude

that the study of criminal behavior is 'stuck in the risk factor stage' (Hinshaw, 2002; Moffitt, 2005b; Rutter, 2003). One way to further examine how some of these factors are associated with antisocial behavior is to use a genetic informative design. For example, the family concentration of criminal behavior (Putkonen *et al*., 2007) may be explained by common genetic influences across generations, but it may also be due to an environmental mediation, or a combination of both. Behavior genetic studies may be helpful here; by using twin and family studies it is possible to disentangle such effects. Another important area where behavior genetic research can be useful is in the field of endophenotypes (Moffitt, 2005a). Research examining the relationship between endophenotypes and criminal behavior may increase the understanding of the underlying genetic mechanism in antisocial behavior. In our own longitudinal twin study of antisocial behavior we examined a particularly robust endophenotype for antisocial behavior: resting heart rate. The results showed that the association between low resting heart rate and antisocial behavior was significantly and entirely explained by common genetic factors, although the heritable component of heart rate explained only a small portion (1–4 per cent) of the substantial genetic variance in antisocial behavior. Despite the effect size being small, children with low resting heart rate appear to be genetically predisposed towards externalizing behavior problems as early as age 9 years old (Baker *et al*., 2009).

SUMMARY AND FUTURE DIRECTIONS

There is no question that genetic influences are important to criminal and other forms of antisocial behavior, with twin and adoption studies convincingly showing strong heritability for both law breaking offenses and various forms of aggression. It appears, however, that the magnitude and nature of genetic effects may well vary across types of criminal offending in adults, with crimes against property showing greater heritability compared to violent crimes against persons. Conversely, aggressive behaviors (which generally involve actions against others) in children and adolescents tend to show stronger genetic effects than delinquent behaviors (which involve actions against property). The relative importance of genes and environment do thus clearly vary across both age as well as the type of antisocial behavior. Moreover, the exact nature of these genetic influences is largely unspecified in twin and adoption studies, as is the specific ways in which environmental factors influence criminal and other antisocial outcomes.

Two avenues of research show promise for elucidating these specific genes and their pathways leading to crime and aggression. First, molecular genetic studies have the potential to identify specific gene variants that increase risk for criminal offending, aggressive behavior, and externalizing behavior disorders. Some polymorphisms, such as mutations in the MAO-A gene, have already shown relationships to antisocial behavior, including violence, across several studies. Second, risk factor research enables identification of measurable biological variables that may have genetic overlap with criminal behavior. However, even well-known and highly heritable risk factors such as resting heart rate, for example, are being shown to explain only very small portions of the large genetic effects in antisocial behavior. Future studies may require comprehensive investigations of many measured risk factors and specific genetic markers in order to explain these genetic effects more extensively.

One of the most important findings to emerge in genetic research on criminal behavior is the fact that the magnitude of genetic effects depends on social circumstances and other environmental factors. All major adoption studies of criminal behavior have shown that genetic risk is amplified in the presence of adverse environmental factors – e.g., defined as measurable, specific social variables such as childhood maltreatment, or as something as general as the presence of a convicted adoptive parent in the home. Viewed differently, the substantial genetic risk for criminal offending may be ameliorated in low risk environments. The nature of these environmentally based protective factors need greater attention in future genetic research on crime and aggression.

The fact that genetic predispositions exist for criminal offending does in no way imply that environmental factors are unimportant, nor that effective treatment and prevention programs cannot be developed and implemented to reduce the chances that an individual will engage in law-breaking or other antisocial behaviors. Heritability estimates for criminal behavior are only moderate at best, according to the Rhee and Waldman (2002) meta-analysis, with that of environmental influences for criminal outcomes being equally strong. Yet, even for disorders completely determined by genetic mutations, such as PKU, environmental interventions such as dietary modifications can ameliorate the adverse effects on cognitive and behavioral outcomes. It is reasonable to assume that detailed understanding of the genetically-based mechanisms underlying risk for criminal behavior could lead to effective social interventions and prevention programs. Given the well-replicated GxE interaction found in behavior genetic studies of criminal behavior, it is important to consider the possibility that certain treatments and interventions may have varying effects for different individuals based on their genetic inheritance.

REFERENCES

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 Profile*. Burlington, VT: University of Vermont Department of Psychiatry.

Arce, E. and Santisteban, C. (2006). Impulsivity: a review. *Psicothema* 18(2): 213–220.

Baker, L. A., Mack, W., Moffitt, T. E. and Medncik, S. A. (1989). Sex differences in property crime in a Danish adoption cohort. *Behavior Genetics* 19: 355–370.

Baker, L. A., Jacobson, K. C., Raine, A., Lozano, D. I. and Bezdjian, S. (2007). Genetic and environmental bases of childhood antisocial behavior: A multi-informant twin study. *Journal of Abnormal Psychology* 116(2): 219–235.

Baker, L. A., Raine, A., Liu, J.-H., and Jacobson, K. C. (2008). Genetic and environmental influences in reactive and proactive aggression in children. *Journal of Child Abnormal Psychology* 36: 1265–1278.

Baker, L. A., Tuvblad, C., Reynolds, C., Zheng, M. and Raine, A. (2009). Resting heart rate and the development of antisocial behavior from age 9–14: Genetic and environmental influences. *Development and Psychopathology* 21: 939–960.

Bartels, M., Hudziak, J. J., van den Oord, E. J. C. G., van Beijsterveldt, C. E. M., Rietveld, M. J. H. and Boomsma, D. (2003). Co-occurence of aggressive behavior and rule-breaking behavior at age 12: multi-rater analyses. *Behavior Genetics* 33: 607–621.

Beaver, K. M., Wright, J. P., Delisi, M., Daigle, L. E., Swatt, M. L. and Gibson, C. L. (2007). Evidence of a gene x environment interaction in the creation of victimization: results from a longitudinal sample of adolescents. *International Journal of Offender Therapy and Comparitive Criminology* 51(6): 620–645.

Beyers, J. M., Loeber, R., Wikström, P.-O. H. and Stouthamer-Loeber, M. (2001). What predicts adolescent violence in better-off neighbourhoods? *Journal of Abnormal Child Psychology* 29(5): 379–381.

Bohman, M., Cloninger, C. R., Sigvardsson, S. and von Knorring, A.-L. (1982). Predisposition to petty criminality in Swedish adoptees. *Archives of General Psychiatry* 39: 1233–1241.

Brooks-Gunn, J., Duncan, G., Klebanov, P. K. and Sealand, N. (1993). Do neighbourhoods influence child and adolescent development? *American Journal of Sociology* 99: 353–395.

Brunner, H. G., Nelen, M., Breakefield, X. O., Ropers, H. H. and van Oost, B. A. (1993). Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science* 262(5133): 578–580.

Button, T. M. M., Scourfield, J., Martin, N. and McGuffin, P. (2004). Do aggressive and non-aggressive antisocial behaviors in adolescents result from the same genetic and environmental effects? *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)* 129 B: 59–63.

Cadoret, R. J., Cain, C. A. and Crowe, R. R. (1983). Evidence for gene–environment interaction in the development of adolescent antisocial behavior. *Behavior Genetics* 13: 301–310.

Cadoret, R. J., Yates, W. R., Troughton, E., Woodworth, G. and Stewart, M. A. (1995). Genetic–environmental interaction in the genesis of aggressivity and conduct disorders. *Archives of General Psychiatry* 52(11): 916–924.

Carey, G. (1986). Sibling imitation and contrast effects. *Behavior Genetics* 16: 319–341.

Carey, G. (1992). Twin imitation for antisocial behavior: implications for genetic and family environment research. *Journal of Abnormal Psychology* 101(1): 18–25.

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A. and Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science* 297(5582): 851–854.

Caspi, A., Langley, K., Milne, B., Moffitt, T. E., O'Donovan, M., Owen, M. J., Polo Tomas, M., Poulton, R., Rutter, M., Taylor, A., Williams, B. and Thapar, A. (2008). A replicated molecular genetic basis for subtyping antisocial behavior in children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry* 65(2): 203–210.

Cloninger, C. R. and Gottesman, I. I. (1987). Genetic and environmental factors in antisocial behavior disorders. *The Causes of Crime*: *New Biological Approaches* (pp. 96–100).

Cloninger, C. R., Sigvardsson, S., Bohman, M. and von Knorring, A. L. (1982). Predisposition to petty criminality in Swedish adoptees. II. Cross-fostering analysis of gene–environment interaction. *Archives of General Psychiatry* 39: 1242–1247.

Crowe, R. R. (1974). An adoption study of antisocial personality. *Archives of General Psychiatry* 31: 785–791.

DeLisi, M., Beaver, K. M., Wright, J. P. and Vaughn, M. G. (2008). The etiology of criminal onset: the enduring salience of nature and nurture. *Journal of Criminal Justice* 36: 217–223.

Dick, D. M., Aliev, F., Wang, J. C., Grucza, R. A., Schuckit, M., Kuperman, S., Kramer, J., Hinrichs, A., Bertelsen, S., Budde, J. P., Hesselbrock, V., Porjesz, B., Edenberg, H. J., Bierut, L. J. and Goate, A. (2008). Using dimensional models of externalizing psychopathology to aid in gene identification. *Archives of General Psychiatry* 65(3): 310–318.

DiLalla, L. F. and Gottesman, I. (1989). Heterogeneity of causes for delinquency and criminality: Lifespan perspectives. *Development and Psychopathology* 1: 339–349.

Edelbrock, C., Rende, R., Plomin, R. and Thompson, L. A. (1995). A twin study of competence and problem behavior in childhood and early adolescence. *Journal of Child Psychology and Psychiatry* 36(5): 775–785.

Eley, T. C., Lichtenstein, P. and Stevenson, J. (1999). Sex differences in the etiology of aggressive and nonaggressive antisocial behavior: results from two twin studies. *Child Development* 70(1): 155–168.

Eley, T. C., Lichtenstein, P. and Moffitt, T. E. (2003). A longitudinal behavioral genetic analysis of the etiology of aggressive and non-aggressive antisocial behavior. *Development and Psychopathology* 15(2): 383–402.

Farrington, D. P. and Loeber, R. (2000). Epidemiology of juvenile violence. *Child and Adolescent Psychiatric Clinics of North America* 9(4): 733–748.

Farrington, D. P., Barnes, G. C. and Lambert, S. (1996). The concentration of offending in families. *Legal and Criminological Psychology* 1: 47–63.

Foley, D. L., Eaves, L., Wormley, B., Silberg, J., Maes, H., Kuhn, J. and Riley, B. (2004). Childhood adversity, monoamine oxidase a genotype, and risk for conduct disorder. *Archives of General Psychiatry* 61: 738–744.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., Hart, E. A. and Hanson, K. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinic sample. *Clinical Psychology Review* 13(4): 319–340.

Gelhorn, H. L., Stallings, M. C., Young, S. E., Corely, R. P., Rhee, S. H., Hopfer, C. and Hewitt, J. K. (2006). Common and specific genetic influences on agressive and nonaggressive conduct disorder domains. *Journal of American Academy of Child and Adolescent Psychiatry* 45(5): 570–577.

Ghodesian-Carpey, J. and Baker, L. A. (1987). Genetic and environmental influences on aggression in 4- to 7-year-old twins. *Aggressive Behavior* 13: 173–186.

Gottesman, I. I. and Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *American Journal of Psychiatry* 1160: 636–645.

Haberstick, B. C., Lessem, J. M., Hopfer, C. J., Smolen, A., Ehringer, M. A., Timberlake, D. and Hewitt, J. K. (2005). Monoamine oxidase A (MAOA) and antisocial behaviors in the presence of childhood and adolescent maltreatment. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)* 135B(1): 59–64.

Hawkins, J. D., Herrenkohl, T., Farrington, D. P., Brewer, D., Catalano, R. F. and Harachi, T. W. (1998). A review of predictors of youth violence. In Loeber, R. and Farrington, D.P. (eds), *Serious and Violent Juvenile Offenders*: *Risk Factors and Successful Interventions* (pp. 106–146). Sage CA: Thousands Oaks.

Hechtman, L. (1999). Predictors of long-term outcome in children with attention-deficit/hyperactivity disorder. *Pediatric Clinics of North America* 46(5): 1039–1052.

Hinshaw, S. P. (2002). Intervention research, theoretical mechanisms, and causal processes related to externalizing behavior patterns. *Development and Psychopathology* 14: 789–818.

Hudziak, J. J., van Beijsterveldt, C. E. M., Bartels, M., Rietveld, M. J. H., Rettew, D. C., Derks, E. M. and Boomsma, D. (2003). Individual differences in aggression: Genetic analyses by age, gender, and informant in 3-, 7-, and 10-year-old Dutch twins. *Behavior Genetics* 33(5): 575–589.

Jaffee, S. R., Caspi, A., Moffitt, T. E., Dodge, K. A., Rutter, M., Taylor, A. and Tully, L. A. (2005). Nature X nurture: genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology* 17(1): 67–84.

Junger-Tas, J., Terlouw, G.-J. and Klein, M. W. (1994). *Delinquent Behavior Among Young People in the Western World*. Amsterdam: Kugler Publications.

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W. and Moffitt, T. E. (2006). MAOA, maltreatment, and gene–environment interaction predicting children's mental health: new evidence and a meta-analysis. *Molecular Psychiatry*, 1–11.

Kutcher, S., Aman, M., Brooks, S. J., Buitelaar, J., van Daalen, E., Fegert, J., Findling, R. L., Fisman, S., Greenhill, L. L., Huss, M., Kusumakar, V., Pine, D., Taylor, E. and Tyano, S. (2004). International consensus statement on attention-deficit/hyperactivity disorder (ADHD) and disruptive behaviour disorders (DBDs): clinical implications and treatment practice suggestions. *European Neuropsychopharmacology* 14(1): 11–28.

Lahey, B. B., Waldman, I. D., and McBurnett, K. (1999). The development of antisocial behavior: An integrative causal model. *Journal of Child Psychology and Psychiatry*, 40(5): 669–682.

Lahey, B. B., Moffitt, T. E. and Caspi, A. (eds), (2003). *Causes of Conduct Disorder and Juvenile Delinquency*. New York: Guilford Press.

Lipsey, M. W. and Derzon, J. H. (1998). Predictors of violence or serious delinquency in adolescence and early adulthood. In Loeber, R. and Farrington, D. P. (eds), *Serious and Violent Juvenile Offenders* (pp. 86–105). Sage CA: Thousand Oaks.

Loeber, R. and Dishion, T. J. (1983). Early predictors of male adolescent delinquency: A review. *Psychological Bulletin* 94: 68–99.

Loeber, R. and Farrington, D. P. (1998). *Serious and Vilolent Juvenile Offenders. Risk Factors and Successful Interventions.* Thousands Oaks, California: SAGE Publications, Inc.

Loeber, R. and Farrington, D. P. (2000). Young children who commit crime: Epidemiology, development, origins, risk factors, early interventions, and policy implications. *Developmental Psychology* 12: 737–762.

Loeber, R. and Hay, D. (1997). Key issues in the development of aggression and violence from childhood to early adulthood. *Annual Review of Psychology* 48: 371–410.

Mednick, S. A., Gabrielli, W. F. and Hutchings, B. (1984). Genetic influence in criminal convictions: evidence from an adoption cohort. *Science* 224: 891–894.

Miller, W. B., Pasta, D. J., MacMurray, J., Chiu, C., Wu, H. and Comings, D. E. (1999). Dopamine receptor genes are associated with age at first sexual intercourse. *Journal of Biosocial Science* 31(1): 43–54.

Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: a developmental taxonomy. *Psychological Review* 100(4): 674–701.

Moffitt, T. E. (2005a). Genetic and environmental influences on antisocial behaviors: evidence from behavioral-genetic research. *Advances in Genetics* 55: 41–99.

Moffitt, T. E. (2005b). The new look of behavioral genetics in developmental psychopathology: gene–environment interplay in antisocial behaviors. *Psychological Bulletin* 131(4): 533–554.

Moffitt, T. E., Caspi, A., Rutter, M. and Silva, P. (2001). *Sex Differnces in Antisocial Behaviour: Conduct Disorder, Delinquency and Violence in the Dunedin Longitudinal Study*. Cambridge: Cambridge University Press.

Moffitt, T. E., Caspi, A. and Rutter, M. (2005). Strategy for investigating interactions between measured genes and measured environments. *Archives of General Psychiatry* 62(5): 473–481.

Munafò, M. R., Clark, T. G., Moore, L. R., Payne, E. R. W. and Flint, J. (2003). Genetic polymorphisms and personality in healthy adults: a systematic review and meta-analysis. *Molecular Psychiatry* 8(5): 471–484.

Nilsson, K. W., Sjöberg, R. L., Damberg, M., Leppert, J., Ohrvik, J., Alm, P. O., Lindström, L. and Oreland, L. (2006). Role of monoamine oxidase A genotype and psychosocial factors in male adolescent criminal activity. *Molecular Psychiatry* 10(11): 903–913.

Ortiz, J. and Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of American Academy of Child and Adolescent Psychiatry* 43(2): 154–162.

Putkonen, A., Ryynänen, O. P., Eronen, M. and Tiihonen, J. (2007). Transmission of violent offending and crime across three generations. *Social Psychiatry and Psychiatric Epidemiology* 42(2): 94–99.

Raine, A. (2002a). Annotation: The role of prefrontal deficits, low autonomic arousal, and early health factors in the developmetn of antisocial and aggressive behavior in children. *Journal of Child Psychology and Psychiatry* 43(4): 417–434.

Raine, A. (2002b). Biosocial studies of antisocial and violent behavior in children and adults: a review. *Journal of Abnormal Child Psychology* 30(4): 311–326.

Rhee, S. H. and Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: a meta-analysis of twin and adoption studies. *Psychological Bulletin* 128: 490–529.

Rietveld, M. J., Hudziak, J. J., Bartels, M., van Beijsterveldt, C. E. and Boomsma, D. I. (2003). Heritability of attention problems in children: I. cross-sectional results from a study of twins, age 3–12 years. *American Journal of Medical Genetics Part B (Neuropsychiatric Genetics)* 117(1): 102–113.

Robins, L. (1978). Sturdy childhood predictors of adult antisocial behaviour: replications from longitudinal studies. *Psychological Medicine* 8: 611–622.

Rowe, D. C. (1983). Biometrical genetic models of self-reported delinquent behavior: a twin study. *Behavior Genetics* 13(5): 473–489.

Rowe, D. C., Rodgers, J. L. and Meseck-Bushey, S. (1992). Sibling delinquency and the family environment: shared and unshared influences. *Child Development* 63(1): 59–67.

Rutter, M. (2003). Crucial paths from risk indicator to causal mechanism. In Lahey, B.B., Moffitt, T.E. and Caspi, A. (eds), *Causes of Conduct Disorder and Juvenile Delinquency.* New York: The Guilford University Press.

Rutter, M., Caspi, A. and Moffitt, T. E. (2003). Using sex differences in psychopathology to study causal mechanisms: unifying issues and research strategies. *Journal of Child Psychology and Psychiatry* 44(8): 1092–1115.

Rutter, M., Giller, H. and Hagell, A. (1998). *Antisocial Behavior by Young People*. Cambridge, UK: Cambridge University Press.

Rutter, M., Moffitt, T. E. and Caspi, A. (2006). Gene–environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry* 47(3/4).

Sampson, R. J., Raudenbush, S. W. and Earls, F. (1997). Neighbourhoods and violent crime: A multilevel study of collective efficacy. *Science* 277: 918–924.

Shih, J. C. (2004). Cloning, after cloning, knock-out mice, and physiological functions of MAO A and B. *Neurotoxicology* 25(1–2): 21–30.

Sigvardsson, S., Cloninger, C. R., Bohman, M. and von Knorring, A. L. (1982). Predisposition to petty criminality in Swedish adoptees. III. Sex differences and validation of the male typology. *Archives of General Psychiatry* 39(11): 1248–1253.

Silberg, J. L., Rutter, M., Tracy, K., Maes, H. H. and Eaves, L. (2007). Etiological heterogeneity in the development of antisocial behavior: the Virginia Twin Study of Adolescent Behavioral Development and the Young Adult Follow-Up. *Psychological Medicine* 37(8): 1193–1202.

Simonoff, E., Elander, J., Homshaw, J., Pickles, A., Murray, R. and Rutter, M. (2004). Predictors of antisocial personality. *British Journal of Psychiatry* 184: 118–127.

Smith, D. A. and Visher, C. A. (1980). Sex and involvement in deviance/crime: A quantitativereview of the empirical literature. *American Sociological Review* 45: 691–701.

Stanger, C., Achenbach, T. M. and Verhulst, F. C. (1997). Accelerated longitudinal comparisons of aggressive versus delinquent syndromes. *Development and Psychopathology* 9: 43–58.

Stouthamer-Loeber, M. and Loeber, M. (2002). Lost opportunities for intervention: undetected markers for the development of serious juvenile delinquency. *Criminal Behaviour and Mental Health* 12: 69–82.

Sutherland, E. H. and Cressey, D. R. (1978). *Criminology*. Philadelphia: J. B. Lippincott.

Tolan, P. H. and Gorman-Smith, D. (1998). Development of serious and violent offending careers. In Loeber, R. and Farrington, D. P. (eds), *Serious and Violent Juvenile Offenders. Risk Factors and Successful Interventions* (pp. 68–85). SAGE Publications.

Torgersen, S., Skre, I., Onstad, S., Edvardsen, J. and Kringlen, E. (1993). The psychometric–genetic structure of *DSM–III–R* personality disorder criteria. *Journal of Personality Disorders* 7: 196–213.

Tremblay, R. E., Pihl, R. O., Vitaro, F. and Dobkin, P. L. (1994). Predicting early onset of male antisocial behavior from preschool behavior. *Archives of General Psychiatry* 51: 732–739.

Tuvblad, C., Grann, M. and Lichtenstein, P. (2004). Heritability for adolescent antisocial behavior differs with socioeconomic status: Gene–environment interaction. *Behavior Genetics* 34(6): 662.

Tuvblad, C., Raine, A., Zheng, M. and Baker, L. A. (2009). The genetic and environmental stability differs in reactive and proactive aggression. *Aggressive Behavior* 35(6): 437–452.

Tuvblad, C., Zheng, M., Raine, A. and Baker, L. A. (2009). A common genetic factor explains the covariation among ADHD ODD and CD symptoms in 9–10-year-old boys and girls. *Journal of Abnormal Child Psychology* 37: 153–167.

van Beijsterveldt, C. E. M., Verhulst, F. C., Molenaar, P. C. M. and Boomsma, D. (2004). The genetic basis of problem behavior in 5-year-old Dutch twin pairs. *Behavior Genetics* 34(3): 229–242.

Van Dusen, K. T., Mednick, S. A. and Gabrielli, W. (1983). Social class and crime in an adoption cohort. In Van Dusen, K. T. and Mednick, S. A. (eds), *Prospective Studies of Crime and Delinquency*. Hingham, Mass.: Kluwer-Nijhoff.

Vermeiren, R. (2003). Psychopathology and delinquency in adolescents: a descriptive and developmental perspective. *Clinical Psychology Review* 23(2): 277–318.

Viding, E., Blair, J. R., Moffitt, T. E. and Plomin, R. (2005). Evidence of substantial genetic risk for psychopathy in 7-year-olds. *Journal of Child Psychology and Psychiatry* 46(6): 592–597.

Vierikko, E., Pulkkinen, L., Kaprio, J. and Rose, R. J. (2004). Genetic and environmental influences on the relationship between aggression and hyperactivity-impulsivity as rated by teachers and parents. *Twin Research and Human Genetics* 7(3): 261–274.