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

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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)
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 1.2 GxE Interaction: differential heritability across measured environments.
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](image-url)
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 (Munafo 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


