Society’s interest in crime reduction strategies largely increased over the last few decades as incarceration rates and their associated costs skyrocketed. The expenses associated with law enforcement practices, legal fees, correctional facilities, and victim services have likely all contributed to this economic burden placed on society (Fass & Pi, 2002; Lovell & Jemelka, 1996; Miller, Cohen, & Rossman, 1993; Petersilia, 1992). Although a large proportion of government funding remains allocated towards supporting crime control and punitive practices, discussions of offender rehabilitation and crime prevention have resurfaced within the realm of the criminal justice system (Anestis & Carbonell, 2014; Davis, Sheidow, & McCart, 2015; Tripodi, 2014). This opportune shift in criminal justice practices is welcomed, given the failed crime preventive efforts and negative offender outcomes that are often associated with the punishment paradigm of the latter half of the twenty-first century.

Paralleling the movement towards reducing crime and its costs to society, but generally removed from such efforts, has been a body of research devoted to identifying the risk factors for criminality. Findings stemming from these studies indicate that biological and genetic factors are involved in the development and stability of antisocial and criminal behaviours (Barnes & Boutwell, 2012; Moffitt, 2005; Raine, 1993). Moreover, genetic factors have been shown to influence an assortment of maladaptive outcomes, including psychopathy, gang membership, weapon use, and illicit drug use (Beaver, Barnes, & Boutwell, 2014; Beaver, DeLisi, Vaughn, & Barnes, 2010; Deng et al., 2015; Fowler et al., 2009).

Despite the mounds of research revealing a strong link between biological and genetic factors and crime, limited attention has been given to the contribution that genetic research could make to preventing or reducing crime. Discussions of the potential use of genetic data in criminal trials have been faced with controversy, fear, and scepticism, in part, because of the concern that such information would be used to oppress offenders. Furthermore, fundamental misunderstandings regarding the ways in which genetic factors are involved in the aetiology of antisocial behaviours persists among scholars and the public. Findings derived from candidate-gene association studies, for instance, have likely encouraged the belief that genes work in a deterministic fashion, where the possession of one gene inevitably leads to antisocial behaviour. Despite these misunderstandings, mounting evidence reveals that human behaviour is far too complex to be attributed to a single gene, that nature and nurture are involved in the aetiology...
of antisocial behaviour, and that genetic factors interact with specific environments to produce criminal behaviours (Carey, 2003; Caspi et al., 2002; Pinker, 2002; Plomin, DeFries, Knopik, & Neiderhiser, 2013).

This chapter provides an overview of some of the key findings to emerge from behavioural genetic and molecular genetic research, as well as how these findings may be integrated into crime prevention and reduction strategies. To this end, the chapter is divided into three main sections. First, a brief overview of the empirical status on the genetics–crime association will be discussed. This section will begin by highlighting how behavioural genetic methodologies help to estimate both the genetic and environmental influences on antisocial behaviours. Additionally, we will review some of the findings derived from molecular genetic research, which has identified a number of genetic polymorphisms that are correlated with antisocial behaviours. Second, we will illustrate the ways in which environmental factors may be conditioned by one’s genotype, a phenomenon known as gene–environment interactions. The discussion on gene–environment interactions will illuminate how genetic polymorphisms interact with certain environments to produce antisocial behaviours. Third, two distinct explanations for gene–environment interactions will be introduced, which are known as the diathesis-stress model and the differential-susceptibility model. This section will conclude with a discussion on how each of these theoretical models may be integrated into crime prevention and rehabilitative practices.

Genetic influences on antisocial behaviour

Behavioural genetic research designs estimate three separate components of the variance in antisocial behaviours. These three components are defined as heritability, shared environmental, and non-shared environmental estimates. Heritability estimates represent the proportion of variance in antisocial behaviours that is due to genetic factors. The variance not accounted for by genetic influences is partitioned to the environment. Behavioural geneticists make the distinction between two types of environments: the shared environment and the non-shared environment. Shared environmental influences work to make siblings similar to one another. An example of a shared environment would be exposure to the same parenting style during childhood. Shared environments are believed to make siblings more similar to one another on the assumption that if certain environments are salient for behaviours – and are experienced equally between siblings – they should exert a similar impact on each sibling’s behaviour. In contrast, the non-shared environmental influences make siblings different from one another. One sibling may be differentially exposed to antisocial peers and become involved in delinquency during their teenage years, whereas another sibling may have prosocial friends and engage in no delinquency.

Although a variety of research designs are available to estimate both the genetic and environmental influences on antisocial behaviours, twin-based methodologies are commonly used by behavioural geneticists to obtain these estimates. Twin-based methods compare the similarity of monozygotic (MZ) and dizygotic (DZ) twin pairs on a particular measure of antisocial behaviour. This method is advantageous because it allows behavioural geneticists to estimate genetic influences more accurately. To illustrate, MZ twins share 100 per cent of their DNA, while DZ twins share approximately 50 per cent of their DNA. Because the environments experienced among MZ and DZ twins are assumed to be relatively similar, a greater similarity on a behavioural trait observed between MZ twins than between DZ twins would reveal that variance in that trait is under some level of genetic influence.

The evidence derived from behavioural genetic research designs in general, and from twin-based methodologies in particular, has revealed that genetic factors are responsible for about
50 per cent of the variance in antisocial behaviours. This overall conclusion regarding the heritability estimates for antisocial behaviours has been compiled from six meta-analyses to date (Burt, 2009a; Burt, 2009b; Ferguson, 2010; Mason & Frick, 1994; Miles & Carey, 1997; Rhee & Waldman, 2002). Additional support provided by a recent meta-analysis of more than 2,700 twin-based studies (and including more than 14,500,000 twin pairs), indicates that genetic factors account for approximately 49 per cent of the variance in human phenotypes, including antisocial traits (Polderman et al., 2015). The remaining variance in antisocial traits is due to the exposure to environmental factors. Estimations of environmental influences are further subdivided between the shared environment and the non-shared environment, which account for approximately 15–20 per cent and 30–35 per cent of the variance in antisocial behaviours, respectively (Moffitt, 2005). This body of research highlights the importance of both genetic risk factors and environmental exposure for fostering the development of antisocial behaviours.

Molecular genetics

Although behavioural genetic research designs provide researchers with an estimate of the genetic influences on an antisocial phenotype, they are unable to provide any information on the specific genes that are involved in fostering antisocial behaviour. Instead, molecular genetic research designs are needed to obtain this information. Findings stemming from molecular genetic research have identified several candidate genes that are involved in the development of criminality, aggression, and violence (DeLisi, Beaver, Vaughn, & Wright, 2009; Guo, Roettger, & Shih, 2007; Liao, Hong, Shih, & Tsai, 2004). It is important to note, however, that most of the genes that are implicated in the development of antisocial and criminal behaviour are associated with neurotransmission.

Briefly, neurotransmission is the process by which information is communicated throughout the body. Neurotransmitters are chemical messengers in the brain and are responsible for transmitting this communication from one brain cell, known as a neuron, to another. Neurotransmitters carry signals from one neuron to an adjacent neuron across a gap known as a synapse. After the neurotransmitters have delivered the signal to the post-synaptic neuron, they have to be cleared out of the synapse through one of two processes. First, neurotransmitters are cleared out of the synapse by transporter proteins that deliver the neurotransmitters back to the presynaptic neuron – referred to as re-uptake. Second, neurotransmitters that remain in the synapse are broken down by enzymes. If there are structural abnormalities to the transporter proteins or enzymes responsible for breaking down neurotransmitters, then neurotransmitter levels may be altered and the process of neurotransmission will no longer work effectively.

Genes that are involved in the transportation and breakdown of neurotransmitters, such as dopamine and serotonin, have been found to be associated with impulsivity, aggression, and criminal behaviour (Beaver, Wright, & Walsh, 2008; Retz, Retz-Junginger, Supprian, Thome, & Rösler, 2004; Faraone, Doyle, Mick, & Biederman, 2001; Raine, 1993). Some of the genes have different variants (i.e. alleles), meaning that different copies of the gene can be inherited. Genes with two or more alleles are called polymorphisms, and the alleles for some of these polymorphisms have been found to vary in their efficiency at removing or breaking down neurotransmitters in the synapse. Accordingly, certain alleles for genes involved in neurotransmission have been found to code for the production of proteins that differentially influence neurotransmitter levels, which may lead to unregulated behaviour and the risk for developing antisocial phenotypes.

Several dopaminergic polymorphisms, for instance, have been found to be related to involvement in antisocial and criminal behaviour. The 10-repeat allele of DAT1, a dopamine
transporter gene, has been linked with aggression and criminal behaviour (Guo et al., 2007; Beaver, et al., 2008). Additionally, the A1 allele of DRD2, a dopamine receptor gene, has been found to be associated with violent delinquency (Guo et al., 2007) and an increased risk of violent victimisation (Beaver, Wright, DeLisi, Daigle, Swatt, & Gibson, 2007). Certain genes of the serotonergic system have also been found to be related to antisocial behaviour. For instance, the short allele of the serotonin transporter promoter gene (5-HTTLPR) has been found to account for variation in certain forms of antisocial behaviour (Retz et al., 2004; Liao et al., 2004).

Some of the genes that code for the production of enzymes, which are responsible for breaking down these neurotransmitters, have also been found to be associated with violence and criminal behaviour (Volavka, Bildern, & Nolan, 2004; Caspi et al., 2002). Monoamine oxidase A (MAOA), for instance, is a gene that codes for an enzyme involved in the breakdown of neurotransmitters such as dopamine and serotonin. The MAOA gene is polymorphic, and some MAOA alleles code for the production of high-activity MAOA while others code for the production low-activity MAOA. The low-functioning MAOA alleles are less efficient at metabolising neurotransmitters and have been linked to an assortment of antisocial outcomes (Beaver, Wright, Boutwell, Barnes, DeLisi, & Vaughn, 2013; Guo, Ou, Roettger, & Shih, 2008). All in all, the findings gleaned from molecular genetic research have provided some insight into systems of genes that might, in some capacity, be involved in the aetiology of antisocial behaviours.

**Gene–environment interactions**

Despite the supporting evidence found for the association between a number of genetic polymorphisms and antisocial behaviour, there is also evidence suggesting that the effects of some of these genes can be even stronger when paired with certain environmental conditions. This phenomenon is referred to as a gene–environment interaction (Rutter, 2006). The main premise of gene–environment interactions is that the effect of the environment depends on genotype, and the effects of genotype depend on the environment. To illustrate, an individual with a genetic predisposition towards antisocial behaviour is more likely to engage in criminal behaviour when exposed to a criminogenic environment. An individual without the genetic predisposition towards antisocial behaviour, in contrast, is significantly less likely to engage in criminal behaviour when exposed to the same criminogenic environment. Gene–environment interactions highlight the complex relationships that exist between genetic predispositions and environmental factors and can help to explain why different people respond to the same environment in different ways.

In one of the first studies to examine gene–environment interactions, the low-functioning MAOA genotype was found to interact with childhood maltreatment to predict the development of antisocial phenotypes in males (Caspi et al., 2002). According to the findings by Caspi and colleagues, even though only 12 per cent of the sample possessed both risk factors (i.e. the low-functioning MAOA genotype and exposure to maltreatment in childhood), this group accounted for 44 per cent of the violent convictions in the sample. Furthermore, 85 per cent of the subjects in the study who were exposed to both risk factors developed antisocial behaviour. This gene–environment interaction has been replicated in several other studies (Fergusson, Boden, Hornwood, Miller, & Kennedy, 2012; Kim-Cohen et al., 2006; Foley et al., 2004) and a recent meta-analysis examining this interaction reaffirms that the low-functioning MAOA genotype and childhood maltreatment interact to increase the likelihood of developing antisocial phenotypes (Byrd & Manuck, 2014).

More recent studies have uncovered gene–environment interactions between dopaminergic polymorphisms and environmental factors that affect the likelihood of developing antisocial
phenotypes. For instance, DRD2 has been found to interact with having a criminal father to predict the development of serious delinquency, violent delinquency, and police contact (DeLisi, Beaver, Vaughn, & Wright, 2009). Additional research indicates that DRD2 and several other dopaminergic polymorphisms (DAT1, DRD4) interact with environmental factors to predict an array of behavioural outcomes including violent behaviour (Barnes & Jacobs, 2013; Beaver, Gibson, DeLisi, Vaughn, & Wright, 2012; Vaughn et al., 2009), early-onset offending (DeLisi, Beaver, Wright, & Vaughn, 2008), externalising behaviours (Bakermans-Kranenburg & van IJzendoorn, 2006), and number of police contacts (Vaughn et al., 2009).

Although gene–environment interactions have helped to shed light on some of the factors involved in the aetiology of antisocial behaviours, the underlying mechanisms which account for gene–environment interactions remain to be fully discovered. To date, scientists have advanced two models to account for gene–environment interactions: the diathesis-stress model and the differential-susceptibility model.

Until relatively recently, the diathesis-stress model provided the primary explanation for gene–environment interactions. The diathesis-stress model posits that genetic risk factors predict antisocial behaviours when coupled with the exposure to an adverse environment. Under this explanation, genetic polymorphisms partially determine how vulnerable a person is to a negative environment. A person with a genetic predisposition for antisocial behaviours, for example, will be more likely to engage in criminal behaviours when exposed to a criminogenic environment. In this light, adverse and criminogenic environments act to serve as triggers on genetic predispositions.

More recently, however, another explanation for gene–environment interactions has been developed by Belsky, which is known as the differential-susceptibility model (1997, 2005). Belsky’s model suggests that rather than being viewed as an indicator of vulnerability to the negative environment, genetic predispositions should be viewed as an indication of plasticity to both a positive and a negative environment. Under the differential-susceptibility model, no longer are gene–environment interactions viewed as the result of negative environments interacting with genetic risk to predict negative outcomes. Instead, genetic risk is recast as genetic plasticity, thereby highlighting the potential for genetic polymorphisms to work with negative and positive environments in a ‘for-better-or-for-worse’ fashion. Individuals with genetic plasticity markers will respond the most positively when exposed to positive environments, just as they will respond the most negatively in the face of adversity.

The diathesis-stress and differential-susceptibility models can be used to help guide crime-prevention and reduction practices. Both frameworks posit that the influences of certain genetic polymorphisms can be moderated by the exposure to certain environments. In the following section, we will discuss how the differential-susceptibility model may be used to inform prevention programmes that seek to thwart antisocial outcomes from surfacing early in life. Following the discussion on crime-prevention efforts, we will illustrate how the diathesis-stress and differential-susceptibility models may be used to guide rehabilitative efforts among offender populations. Under this recommendation, both frameworks could be implemented in conjunction with the principles of effective intervention (PEI) to reduce recidivism rates.

Integrating genetic information into crime-prevention and reduction practices

Intervention attempts to reduce and/or prevent antisocial and other problem behaviours have been shown to exert modest effects at best. The long-term intervention benefits for reducing youthful antisocial behaviour, moreover, have been found to be less effective than anticipated.
(Sawyer, Borduin, & Dopp, 2015). Most often, the lack of empirical support for some of these prevention and treatment programmes has been attributed to poor programme dissemination and implementation, as well as to variability in participant demographics and intervention features. A growing body of experimental-intervention research, guided by the differential-susceptibility framework, suggests that the reason for inconsistent programme efficacy could be due to ignoring participant variability in genetic susceptibilities to environmental influences (Belsky & van IJzendoorn, 2015). Thus, prevention efforts that fail to consider the importance of individual characteristics will not be as successful at identifying the individuals for whom the prevention programmes would be the most effective.

Genetically informed intervention research has already begun to examine whether genetic polymorphisms can moderate programme effectiveness among participants. Importantly, many of the genetic polymorphisms found to be associated with antisocial behaviours have also been identified as plasticity genes in randomised intervention trials. Research by Bakermans-Kranenburg and colleagues (2008), for instance, found that children with a history of externalising behaviour problems benefitted the most from family-based interventions if they also possessed the risk allele of the dopamine D4 receptor gene (DRD4). The moderating role of the DRD4 risk allele has also been revealed in substance use preventive interventions. Adolescents assigned to a family-oriented intervention programme displayed decreased substance use, especially if they were carriers of the 7 repeat allele of DRD4 (Brody et al., 2014).

The evidence gathered from genetically informed prevention science suggests that the moderating role of genotype has important implications for programme efficacy. If prevention interventions expanded their focus of participant characteristics to include genotype, then prevention scientists would be able to predict with more accuracy who would benefit the most from the intervention. Indeed, programme participants are commonly targeted for interventions based on a number of risk factors, which include sex, age, race, and a history of antisocial behaviours. With the implementation of a differential-susceptibility framework, no longer would participants be solely identified as belonging to the most at-risk population in need of intervention. Instead, participants would be targeted based on their susceptibility or plasticity for change. This ‘change’ would include change for the better. Many existing preventive interventions aimed at preventing or reducing antisocial behaviours could easily begin to include genetic data in their research designs. Again, the inclusion of data on participant genotype could explain more variation in programme effectiveness and increase the precision of programme implementation.

While the differential-susceptibility model has clear implications for increasing the effectiveness of prevention programmes, both the differential-susceptibility and diathesis-stress models have the potential to increase the effectiveness of rehabilitation/treatment programmes. The key way in which these models have application to these programmes is via their integration with the principles of effective intervention (PEI). The PEIs developed out of the need to increase rehabilitative success among offender populations (Andrews, Bonta, & Hoge, 1990; Bonta & Andrews, 2007). There are three principles of effective intervention, but only two that have direct application to genetic profiles: the risk principle and the responsivity principle. The risk principle suggests that high-risk offenders are the most amenable to change, and in turn, benefit the most from treatment programmes. The second PEI that is germane to genetic information is known as the responsivity principle and seeks to identify the most efficient modes of treatment for offender populations. The responsivity principle is further subdivided between general responsivity and specific responsivity. General responsivity posits that offenders will respond best to certain treatment models. In contrast, specific responsivity highlights the reality that offenders have individual needs and characteristics (e.g. learning styles, personalities, motivation,
and abilities), which modify treatment success. Therefore, specific responsivity suggests that offenders will benefit the most from individualised treatment programmes.

The diathesis-stress and differential-susceptibility frameworks share similarities with the risk principle and the specific responsivity principle, respectively. First, recall that the diathesis-stress model suggests that individuals with genetic risk and exposure to environmental risk factors are the most likely to develop antisocial behaviours. The risk principle aligns with the theoretical framework of the diathesis-stress model, by virtue of the explicit focus on participant risk. Indeed, both theoretical models highlight the importance of individual-level risk factors for antisocial outcomes. Incorporating genetic data into risk assessments would allow the most at-risk offenders to be identified and treated in rehabilitative programmes. By default, the offenders with the lowest genetic risk would be diverted from such programmes. Utilising genetic data would allow practitioners to provide services to the offenders who are most in need of treatment.

Second, the specific responsivity principle aligns with the differential-susceptibility model because both theoretical models focus on how individual-level characteristics can moderate programme effectiveness. To illustrate, offenders could be assigned to various treatment programmes based on their level of genetic plasticity, personality traits, and learning styles. Once combining these theoretical models, practitioners might begin to recognise how genetic factors could work with other individual-level characteristics to help moderate the effectiveness of rehabilitative treatment programmes, and in turn, increase offenders’ chances for success.

Conclusion
Incorporating genetic data into preventive and rehabilitative programmes has the potential to lead to positive outcomes for offenders. Instead of fearing the possibility that genetic data would be used to oppress offenders, practitioners and the public alike should begin to focus on the many ways genetic factors could increase crime-prevention efficacy and reduce recidivism. Incorporating the theoretical frameworks of gene–environment interactions into existing prevention practices and PEIs could ultimately lead to better programme implementation, more accurate risk assessments, and greater programme success rates.

References


