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Is There a Genetic Susceptibility to Engage in Criminal Acts?

Katherine I. Morley and Wayne D. Hall

Debates about criminality have long focussed on the relative contributions of environment and genetics as components of antisocial and destructive behaviour. Although genetic explanations for criminal behaviour have been circulated since the emergence of modern criminology in the 1700s, until recently, there has not been the scientific evidence to substantiate or refute any claims. The past decade or so has seen an increase in research on the genetics of behaviour, including antisocial behaviour. The findings of some of this research have inspired media speculation about its policy implications. Many criminologists are understandably concerned about the potential misuse of this research given the earlier historical experiences with the eugenic use made of biological explanations of crime, and of genetic explanations in particular.

This brief paper summarises this evidence. Recent twin studies show persuasive evidence that both genetic and environmental factors contribute to antisocial behaviour. However the genetic evidence indicates that there is no single gene, or even a small number of genes, that predict an increased risk of antisocial behaviour. Where there have been some effects the increase in risk associated with antisocial behaviour is modest..

A technical appendix to this paper discussing candidate genes for antisocial behaviour is available on the AIC website <<http://www.aic.gov.au/publications/tandi2/tandi263.html>>.

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Acting Director

Genetic theories of the origins of criminal behaviour have been a source of contention for over a century since Lombroso proposed quasi-biological explanations of criminal behaviour (Pick 1989; Andrews 1999). Genetic theories of criminality have been especially controversial within the field of criminology because of the eugenic policies that they inspired that were implemented during the Nazi era (Kevles 1985).

The sequencing of the human genome has created a renewed interest in the contribution of genetics to socially disapproved behaviour such as addiction, mental disorders and criminal behaviour. Both the media and the public have shown significant interest in stories relating genes to such disorders and their presumed implications for policy. Criminologists, lawyers and policy makers in the criminal justice field need to be well informed about the results of research on genetics of criminal behaviour and its limitations, a need that will only increase as genetic research on behaviour becomes more sophisticated.

There is an understandable fear among criminologists that information on increased genetic risks of engaging in criminal

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acts may adversely affect strategies used to prevent and deal with people who commit crimes. Some commentators fear that genetic information on criminal predisposition may be used by policy makers to justify reduced funding for programs directed at environmental causes of crime (Wasserman & Wachbroit 2001). More speculatively, there is a concern that the identification of genetic susceptibility to criminality may lead to proposals for genetic screening of the population for susceptibility to criminal behaviour (Rowe 2002). These programmes would aim to identify persons at increased risk of engaging in criminal activities and then intervene in some ways to reduce their risk. Such proposals understandably raise fears of a return to the type of state-sponsored intervention in reproduction, pre-emptive incarceration or medication, and scientifically sanctioned racism that earlier enthusiasms for biological explanations of crime have prompted (Comings 1996; Andrews 1999; Rowe 2002).

Before the policy implications of genetic research are addressed we believe that it is essential to critically examine the current state of research on this topic. Such an examination provides the necessary basis for evaluating the validity and ethical acceptability of speculative proposals for the preventive use of genetic information about individual risks of engaging in criminal behaviour.

In this paper we accordingly review current knowledge of genetic influences on criminal behaviour and make some tentative predictions about its future direction. This is a preliminary to a more detailed analysis. The potential preventive uses of such information by society and the criminal justice system will be the subject of a separate paper.

Defining criminal behaviour

One of the major challenges in researching the causes of criminal behaviour — whether these be genetic or environmental — is how we should define it. Criminal behaviour is defined by statute and as such is necessarily a social and legal concept rather than a biological one. In light of this fact, some researchers have argued that criminal behaviours should be examined within the wider context of antisocial behaviour (Rutter et al. 1998). This is the approach we follow in this paper.

Three ways of defining antisocial behaviour can be distinguished. The first approach equates it with criminality and delinquency. Criminality is defined as engaging in activities that result in criminal prosecution or incarceration, while delinquency is defined as engagement in unlawful activities while under the age of 18 (Rhee & Waldman 2002). Information on these types of antisocial behaviour can be collected either through police and court records of criminal offences or via anonymous self-reports of participation in activities that would be considered criminal if they had resulted in arrest and conviction (Rhee & Waldman 2002). This categorisation of criminal behaviours is problematic because it means that what constitutes criminal behaviour is defined by statute and therefore changes over time and varies between countries (Rutter et al. 1998).

The second approach that is often used in genetic studies is to use diagnostic criteria for various personality disorders that are associated with an increased risk of criminal activity, namely, Antisocial Personality Disorder (ASPD). ASPD is characterised by a persistent disregard for, and violation of, the rights of others. It can only be diagnosed in individuals over the age of 18 (First 2000). Three childhood disorders — Attention Deficit

Hyperactivity Disorder (ADHD), Conduct Disorder (CD) and Oppositional Defiant Disorder (ODD) — are also often assessed because they have been identified as risk factors for development of ASPD. ADHD is distinguished by frequent inattention and/or hyperactivity-impulsivity, while individuals with CD display behavioural characteristics that are comparable to ASPD (violation of societal norms or rules) (First 2000). ODD is similar to CD in that it involves disobedient or hostile behaviour, but if more serious forms of behaviour are present, the diagnosis of CD takes precedence (First 2000).

A third approach to antisocial behaviour has been to investigate personality traits that may be risk factors for engaging in criminal behaviour. Aggressiveness and impulsivity have been the most heavily researched traits, usually assessed by personality questionnaires (Rhee & Waldman 2002). Adult hyperactivity, often appearing as ADHD, may also be of interest because individuals who exhibit both antisocial and hyperactive behaviour are more likely to engage in criminal behaviour (Rutter et al. 1998).

These three broad approaches to measurement overlap and are interrelated. For example, a prior diagnosis of CD is part of the criteria for ASPD and approximately half of all children clinically diagnosed with ADHD also have ODD or CD (First 2000). Additionally, childhood aggression has been found to predict adult criminality, and criminality, aggressiveness and impulsivity are also part of the criteria for ASPD (Rhee & Waldman 2002).

A number of limitations should be highlighted before considering studies that use these three approaches to investigate the role of genetics in antisocial behaviour. Firstly, these studies are primarily concerned with more serious crimes against property or person. They are not thought to have any significant

influence on criminal behaviours such as fraud, embezzlement, or other “white collar” crimes (Rutter et al. 1998). Secondly, the correlation between these disorders and crime is not perfect. Not all individuals who are diagnosed with ASPD and related disorders will engage in criminal behaviour and not all convicted criminals will meet the criteria for one or more of these disorders (Rhee & Waldman 2002). Finally, most of this research does not aim to identify genetic influences on criminal behaviour per se. Rather these studies aim to find gene variants that increase the risk of developing a particular psychological disorder, which may in turn increase the risk of engaging in criminal behaviour.

Heritability and antisocial behaviours

Antisocial behaviour often clusters within families, suggesting that both inherited genetic factors and family environment are risk factors for this behaviour. Twin and adoption studies have been used to separate genetic and environmental influences and to assess the contribution that these factors make to the liability to engage in antisocial behaviour.

Adoption studies are those in which individuals with a family history of antisocial behaviour are adopted out to families without such a history. If the majority of adoptees later engage in antisocial behaviour, this suggests that genetic background has more influence on liability than family environment. Twin studies compare the occurrence of the behaviour in monozygotic (MZ) and dizygotic (DZ) twin pairs. If more MZ than DZ twin pairs both have the disorder, this indicates a genetic contribution to the development of the trait. Statistical models are used to determine the “heritability of the trait”, that is the contributions made by shared genes as well as the contributions of shared (e.g.

family) and non-shared environment.

Rhee and Waldman (2002) recently conducted a review of the majority of the twin and adoption studies on antisocial behaviour that have been carried out. They found that although genetic background has a strong influence on whether an individual will engage in antisocial behaviour, the influence of environmental factors is even stronger. These results highlight the fact that even if individuals have a strong genetic predisposition, they may never engage in any antisocial behaviours if they are not exposed to the necessary environmental factors.

Mode of inheritance

The manner in which the personality disorders and behavioural traits associated with criminal behaviour are inherited has important implications for research and the potential policy uses of the research. First, all of these behavioural characteristics are determined by many different factors. An individual’s risk of developing these disorders or displaying these traits is not determined simply by their genotype; environmental influences such as parenting style, socioeconomic status, and peer groups also play a role (Rutter et al. 1998; Gatzke & Raine 2000). Additionally, interactions between genetic and environmental factors, and between different genes, probably influence the development of these traits and disorders.

Although they have some genetic basis, ASPD and related disorders are not influenced by a single gene, and are not inherited in one of the simple patterns of inheritance identified by Mendel. The consensus view is that these traits are influenced by the additive effects of many different gene variants that are widely distributed throughout the general population rather than

confined to a small proportion of individuals. Individuals engage in antisocial behaviour when they inherit a sufficient number of variant genes and are exposed to the right (or wrong) social environment (Comings 2000).

Candidate genes

Candidate genes are specific genes that are thought to contribute to an increased risk of engaging in antisocial behaviour. They are usually selected on the basis of information about the brain-related bases of behaviour and personality traits. Association studies are usually used to investigate candidate genes. These studies examine whether one variant of a candidate gene occurs more often in individuals who display antisocial behaviour than in some comparison group.

As has been true in studies of many other personality traits, research on candidate genes for antisocial behaviour has primarily focused on genes that influence the ways in which nerve impulses are transmitted and received in the brain. Three such pathways have been investigated in relation to antisocial behaviours.

The serotonergic pathway

The serotonergic pathway is involved in brain development and dysfunction in this system is thought to increase aggressiveness and impulsivity (Reif & Lesch 2003). Associations have been found between a number of genes involved in this pathway and antisocial behaviours, namely impulsivity, aggression and ADHD (see Table 1).

The dopaminergic pathway

The dopaminergic system is involved in “reward pathways” in the brain (Reif & Lesch 2003). Genes involved in this pathway have primarily been investigated for involvement in ADHD, although one study did find an association with impulsivity and ADHD-related symptoms in violent offenders (see Table 1).

The noradrenergic pathway

The noradrenergic system functions as a central arousal system (Reif & Lesch 2003). Disruptions to the regulation of the noradrenergic pathway have been implicated in psychological disorders such as anxiety and depression. Only two genes involved in this pathway have been examined for a relationship with antisocial behaviours. They have been found to be associated with ADHD and also impulsivity and hostility (see Table 1).

Genes involved in two or more pathways

Dopa decarboxylase (DDC) is involved in both the serotonergic and dopaminergic systems. Two studies have provided evidence that suggests the involvement of this gene in ADHD.

Monoamine oxidase A (MAOA) is involved in the serotonergic, dopaminergic and noradrenergic pathways. MAOA has become the focus of much genetic research on criminal or antisocial behaviour because the study by Brunner et al. (1993) identified an association between a mutation in MAOA and impulsive aggression. Although this relationship has not been confirmed outside the family examined in the original study, MAOA has been the focus of a number of studies, some of which

suggest that the gene has some influence upon antisocial behaviours.

Multi-gene studies

The inconclusive results from studies of individual candidate genes for antisocial behaviour reflect the fact that these behaviours are likely to be influenced by the interaction of multiple genes. Each genetic variant that influences antisocial behaviour will only have only a small impact on an individual's overall predisposition to such behaviour. It is therefore unsurprising that individual studies of single candidate genes do not always produce the same result (Ioannidis et al. 2001). Some researchers have begun to address this problem by studying multiple susceptibility genes for behavioural traits and disorders that increase the risk of engaging in antisocial behaviour.

Comings et al. have simultaneously examined multiple candidate genes for their involvement in ADHD, CD and ODD. These studies suggest that some of the genes in the serotonin, dopamine and noradrenergic pathways do influence the development of these disorders (Comings 2000; Comings et al. 2000a; Comings et al. 2000b). However, some of the results of these studies conflict with the

results of some single-gene studies. The authors found that the noradrenergic genes had a stronger influence than other groups, but only single-gene studies of DBH have produced relatively consistent positive results. It remains to be seen whether this inconsistency is due to different research methods, or the fact that the noradrenergic pathway has not been as well investigated in single-gene studies.

How much can genes tell us?

Genetic research is beginning to identify genetic variants that may have some bearing on an individual's liability to develop antisocial behavioural characteristics. In keeping with the polygenic pattern of inheritance proposed for antisocial behaviours, the amount that each individual gene contributes to an individual's overall liability is likely to be small. This is evident in Table 1 which summarises the relative risks (RR) and odds ratios (OR) for the candidate genes reviewed above.

These measures of risk indicate that an individual with a susceptibility variant of one of these genes will only have ~1.5 times the risk of antisocial behaviour compared to an individual from the general population. Thus an individual will only have a significantly increased risk of engaging in antisocial behaviour if they carry a large number of variant genes. This average RR is consistent with the results of meta-analyses of associations between individual genes and risk of developing a range of disorders and diseases (Ioannidis 2003).

Implications and some tentative predictions

This review of genetic research on antisocial behaviour has summarised growing evidence for

Table 1: Relative risks, odds ratios and associated behaviours for candidate genes

Gene	Risk	Behaviour
Serotonergic system		
Tryptophan hydroxylase	Not available	Impulsivity, aggression
Serotonin receptors	RR=1.24	Impulsivity (males), ADHD
Solute carrier family 6, member 4	RR=1.29	ADHD
Dopaminergic system		
Dopamine receptor D4	RR=1.5; OR=1.4	ADHD
Dopamine receptor D5	RR=1.57-1.67	ADHD
Dopamine receptor D3	Not available	Impulsivity, ADHD
Solute carrier family 6, member 3	RR=1.2; OR=1.5	ADHD
Noradrenergic system		
Dopamine-beta-hydroxylase	RR=1.31	ADHD
Alpha adrenergic receptor 2A	Not available	Impulsivity, hostility
Other genes		
Dopa decarboxylase	RR=1.48; 1.63	ADHD
Monoamine oxidase A	OR=2.8	Impulsivity, aggression, CD, criminal conviction

a genetic contribution to antisocial behaviour but it has also indicated that it is highly unlikely that variants of single genes will be found that very substantially increase the risk of engaging in criminal behaviour. Instead, it is much more likely that a large number of genetic variants will be identified that, in the presence of the necessary environmental factors, will increase the likelihood that some individuals develop behavioural traits that will make them more likely to engage in criminal activities. This review has a number of implications for proposed uses of genetic information in crime prevention and offender rehabilitation that we will briefly sketch here and develop in more detail elsewhere.

Firstly, adoption and twin studies of antisocial behaviours suggest that there are significant environmental, as well as genetic, risk factors for these behaviours. Research such as that of Caspi et al. (2002) has also shown that genetic studies are likely to provide information about both types of risk factors. We believe that genetic research is more likely to refine social policies by better specification of environmental risk factors than to divert funds from environmental crime prevention strategies.

Secondly, susceptibility alleles for antisocial behaviours only increase risk. They are not deterministic and only poorly predict the likelihood that an individual will engage in such behaviour. Additionally, the presence or absence of environmental risk factors cannot be identified by a genetic test. Taking this information into account, proposals for population-wide genetic screening for criminality do not appear to be feasible. We believe that eugenic governmental policies such as pre-emptive incarceration are unethical. Such policies are also impractical because they require genetic tests with high predictive

value that do not exist and are unlikely to be found.

Thirdly, the majority of genetic research on antisocial behaviours has been conducted on Caucasian populations, and does not aim to identify race-specific susceptibility alleles for antisocial behaviour. The polygenic nature of antisocial behaviour also means that even if a susceptibility allele is found at a high frequency in a particular ethnic group, it is likely that a different susceptibility allele will be found at a similarly high frequency in another ethnic group. We believe it is unlikely that genetic research in this area will lead to or inspire racist crime policies, but anxieties about this issue need to be addressed by behavioural geneticists.

Genetic research on criminal behaviour may, however, have some uses in offender treatment and rehabilitation. Information from genetic studies may be used to develop new treatments for personality disorders such as ASPD, CD, ADHD and ODD that are risk factors for criminal behaviour. Genetic information could also be used to assist in diagnosing offenders who have treatable psychological disorders. Comings et al. (2000b) have suggested that their multi-gene tests could have such diagnostic applications in the future. It is less certain what the consequences of such genetic diagnostic tests may be for criminal cases in which they may be cited as empirical evidence of a defendant's diminished responsibility. Many issues need to be examined in more detail before genetic information could be used in legal settings to assess guilt and to decide upon penalties for criminal acts.

An analysis of candidate genes for antisocial behaviours and a glossary is available on the AIC website <<http://www.aic.gov.au/publications/tandi2/tandi263.html>>.

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