Chapter 9

Review of the evidence: antisocial behaviour

Review of the evidence: antisocial behaviour[1]

Background

9.1 Various biological explanations of antisocial and criminal behaviour have been offered historically but none have stood up to rigorous analysis or offered successful or acceptable solutions to the continuing problems caused by such behaviour. It is widely accepted that crime and antisocial behaviour are the results of many different influences, some of which, such as deprivation and poverty, are already the target of existing interventions. This chapter explores attempts by researchers in behavioural genetics to examine a potential genetic contribution towards such behaviour.

Trait definition and measurement

9.2 Antisocial behaviour is studied by different disciplines, each of which has its own perspective on the definition and measurement of what is antisocial. Mental health clinicians, criminologists and personality psychologists conceptualise and measure antisocial behaviour somewhat differently, but all three fields share in common the underlying assumption that antisocial behaviour is behaviour that violates the rights and safety of others.

9.3 Mental health clinicians interested in pathological behaviour conceptualise antisocial behaviour as a mental disorder. As a result, their definitions require that the behaviour is seriously harmful to others, involves a number of different types of antisocial acts, or has persisted over a long time period. The primary labels assigned to antisocial pathology are: *conduct disorder*, in young people under 18; *antisocial personality disorder*, in adults; and *psychopathy*, also in adults. These clinical definitions tend to apply to fewer than 5–10% of the population, depending on age. Such mental disorders are typically measured as diagnostic categories (either the individual meets the criteria for the disorder, or not). However, there are instruments that measure these disorders as continuous distributions, in which the number of different symptoms exhibited is counted. Examples of dimensional instruments that have been used in research in behavioural genetics with children include the Achenbach Child Behaviour Checklist for externalising behaviours and the Rutter antisocial scale. An example of a dimensional measure used in research with adults is the Minnesota Multiphasic Personality Inventory (MMPI) psychopathy scale, which was discussed briefly in paragraph 8.1 in relation to personality tests. Information about the presence or absence of symptoms is usually gathered about young children through reports from parents and teachers, about older children and adolescents through parents, teachers and self-reports, or about adults through self-reports or clinical records.

9.4 Criminologists conceptualise antisocial behaviour as behaviour that is against the law. As a result, their definitions do not require there to be serious harm, a variety of acts or persistence. Nonetheless, in practice most criminologists discriminate between the minor offender who commits a one-off offence and the more extreme alternatives, namely

[1] The material in this Chapter is taken from a paper by Professor Terrie Moffitt, Institute of Psychiatry, King's College London. The paper is available on the Council's website: www.nuffieldbioethics.org.

violent, proficient or persistent recidivistic offenders, because the latter are of greater concern in policy terms. The primary labels assigned are: *delinquency*, in juveniles, whose age is legally defined; and *crime*, in adults. These legal definitions tend to apply to between 20–30% of the population, depending on age. These constructs are sometimes measured as legal categories (either the individual has been convicted at court, or not). However, they are more commonly measured as continuous distributions, in which the number of different illegal behaviours committed and the frequency with which they have been committed are counted. Information about the presence or absence of illegal acts is usually gathered through self-reports or police records (about older children and adolescents), or through self-reports or court conviction records (about adults).

9.5 Personality psychologists conceptualise antisocial traits in terms of attitudes, beliefs, interests and preferences that indicate an inclination to take advantage of or harm others, or a willingness to break the law. As a result, their definitions do not require that any antisocial act has occurred. Personality psychologists think of the 'aggressiveness' of humans as a characteristic analogous to the 'brittleness' of glass; always there, but not necessarily expressed. Although older personality measures that were labelled 'aggression' sometimes included items asking about actual physical acts, it has been shown that such acts are not integral to the measures. The primary labels assigned are *hostility*, which refers to temper, *socialisation*, which includes such traits as conscientiousness and honesty, or *aggression*. Definitions of personality traits tend to apply to the entire population; the high end of an 'aggression' scale may indicate enthusiasm for aggression whereas the low end may indicate timidity. These personality constructs are virtually never measured as discrete categories, because a fundamental assumption about personality traits is that they are continuously distributed in the population. They are generally measured using checklists which count the number of different antisocial attitudes endorsed by the respondent. Examples of dimensional instruments that have been used in research in behavioural genetics include the Multidimensional Personality Questionnaire (MPQ) aggression scale, the Buss–Durkee hostility scales, and the California Psychological Inventory (CPI) socialisation scale. Information about antisocial personality traits is usually gathered through self-reports about older adolescents and adults.

9.6 Although the discussion above highlights differences between the three ways of conceptualising and measuring antisocial behaviour, research that has crossed disciplines has repeatedly shown that clinical, legal and personality measures are moderately to strongly related to each other. For example, aggressiveness measured in adolescence is strongly correlated with later court conviction for violent criminal offending in adulthood, as well as with psychiatric diagnosis of antisocial personality disorder.[2,3] Moreover, as we shall see, estimates of genetic and environmental influences on the three types of measure are more similar than different.

9.7 Classifying individuals into dichotomous categories based on their antisocial behaviour is fraught with difficulty and generally classification is not strongly reliable. Is an individual a psychopath, or not? Is an individual a criminal, or not? Does he or she have a diagnosis

[2] Moffitt, T. E., Krueger, R. F., Caspi, A. & Fagan, R. W. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology* **38**, 201–35.

[3] Krueger, R. F., Caspi, A., Moffitt, T. E., Silva, P. A. & McGee, R. (1996). Personality traits are differentially linked to mental disorders: A multi-trait/multi-diagnosis study of an adolescent birth cohort. *J. Abnorm. Psychol.* **105**, 299–312.

of conduct disorder, or not? Does he or she have a diagnosis of antisocial personality disorder, or not? Assigning a person to such categories is a matter of deciding that his or her behaviour has surpassed some cut-off point along a continuous measure of antisocial behaviours. The cut-off points are more arbitrary than based on evidence. Unreliable assignment arises frequently because individuals' scores fall just above or below cut-off points. Quantitative genetic theory, psychometric theory and accumulated research findings suggest that a truer, and more reliable, measure of individual differences in antisocial behaviour is obtained when the natural continuum is not arbitrarily cut into categories. For this reason, we consider antisocial behaviour in this Report as a trait that is normally distributed and therefore able to be measured as part of variation in the normal range.

9.8 The advantages of the principle of continuous dimensional measures do not apply only to research in behavioural genetics. Rather, measures of antisocial behaviour that sample a variety of behaviours are more useful in other types of research as well. For example, longitudinal research shows that the variety of different antisocial acts a child has exhibited is the single best predictor of his or her adult outcome.[4] There is therefore a growing consensus that measures of antisocial behaviour should gather data about a large number of different behaviours covering a wide range of antisocial severity, and should include a period of observation that allows enough time for research participants to exhibit the behaviours.

Current findings: quantitative genetics

Antisocial behaviour

9.9 Estimates of heritability for antisocial behaviour from recent research in quantitative genetics cluster around 0.50. The most reliable estimates come from contemporary studies in the Netherlands, Britain, Norway, Sweden, Australia and the US, because these studies examine large, representative samples using sophisticated quantitative modelling techniques. A complementary meta-analysis of 51 twin and adoption studies yielded an estimate of heritability of 0.41 for the genetic influence on antisocial behaviour.[5] Estimates of heritability below 0.20 tend to emerge from studies with unusual design features; for example, observational measures, small sample sizes, very wide age ranges, small groups of girls, or adults being asked to report childhood symptoms retrospectively. Similarly, some, but not all, studies yielding estimates above 0.70 have non-optimal designs, such as small sample sizes or adults being asked to report their childhood symptoms retrospectively.

9.10 The sizes of estimates of heritability vary somewhat across different types of measures of antisocial involvement. Overall, this variation appears to be systematic. It reflects the fact that (all other aspects of methodology being equal) higher estimates of heritability are obtained from studies using measures that sample the most different types of antisocial behaviours. Measures that sample many behaviours yield more accurate estimates because their scores are less contaminated with error, and also because they are more sensitive to

[4] Robins, L. N. (1978). Sturdy childhood predictors of antisocial behaviour: replications from longitudinal studies. *Psychol. Med.* **8**, 611–22.

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

the full range of behaviour in the population. The lowest estimates of heritability emerge from observational measures, which sample only a narrow type of behaviour such as hitting a doll or arguing with a parent, over brief spans of time, usually minutes. The next lowest estimates emerge from measures of official offending in juveniles, which also sample a narrow range of behaviour, namely illegal behaviour, which is seldom detected. Fewer than half of juveniles who offend are arrested, and of those arrested 75% are arrested only once or twice. Medium estimates of heritability tend to emerge from measures of self-reported offending, symptoms of conduct disorder and official offending in adults, all of which tend to aggregate across a moderately wide sample of behaviours and moderately long periods of ascertainment.

9.11 The largest estimates of heritability tend to emerge from studies using measures able to array individuals along a continuum from non-antisocial to severely and persistently antisocial. These are studies using other-reported delinquent or aggressive behaviours (such as the Child Behaviour Check List (CBCL) externalising scale), and self-reported personality traits (such as the MPQ aggression scale). These studies tend to include a very large number of items inquiring about a variety of antisocial attitudes and behaviours. Some of these items, such as robbery, are exhibited rarely by people, but others, such as enjoying violent films, are exhibited commonly. As a result, the instruments are sensitive to population variation in the severity of antisocial behaviour.[6] Overall, the distribution of more than 100 estimates of heritability from recent papers approximates a bell-shaped normal curve. This distribution is to be expected from a sample of more than 100 imperfect estimates of a true effect that equals 50% in nature.

9.12 As well as the possibility that genes influence antisocial behaviour, it is also possible that antisocial experience can influence how genes are distributed in the population. This is an implication of the finding that men and women mate on the basis of similarity between the partners' antisocial behaviour (this is called assortative mating), and that couples in which both people exhibit antisocial behaviour tend to have more children than the norm.[7] Assortative mating on a genetically-influenced phenotype, such as antisocial behaviour has consequences for genetic variation in the population. Because people form unions with other people like themselves, the result is that families differ more from each other on average than they would if people mated randomly. If successive generations mate assortatively, genes relevant to the phenotype will become concentrated within families. Consider height as an example. Whole families clearly differ from other families in terms of height, yet families are made up of persons who are similar in height. Part of the explanation for this phenomenon is likely to lie in the positive assortative mating that occurs for this trait.

[6] A study of 14,500 Danish adoptee families provides a good example of the importance of measures that sample different acts along a dimension to improve sensitivity to features of antisocial behaviour such as severity, frequency and persistence (Mednick, S. A., Gabrielli, W. F. & Hutchings, B. (1984). Genetic factors in criminal behaviour: evidence from an adoption cohort. *Science* **224**, 891–3). When adoptee family members were classified simply as 'not convicted' or 'convicted' (a legal status applying to nearly one quarter of Danish males), the estimate of heritability was modest. However, when the number of convictions in an individual's life-time from age 15 to 50 was considered, stronger estimates of heritability emerged for individuals who had repeatedly been convicted on many successive court dates, presumably reflecting what criminologists call a 'crime career'.

[7] See for example Farrington, D. P., Barnes, G. C. & Lambert, S. (1996). The concentration of offending in families. *Leg. Criminol. Psychol*. **1**, 47–63; Rowe, D. C. & Farrington. D. P. (1997). The familial transmission of criminal convictions. *Criminology* **35**, 177–201; Farrington, D. P., Jolliffe, D., Loeber, R., Stouthamer-Loeber, M. & Kalb, L. (2001). The concentration of offenders in families, and family criminality in the prediction of boys' delinquency. *J. Adolescence* **24**, 579–96; Kreuger, R. F., Moffitt, T. E., Caspi, A., Bleske, A. & Silva, P. A. (1998). Assortative mating for antisocial behaviour: development and methodological implications. *Behav. Genet*. **28**, 173–86.

9.13 Another important insight from research in behavioural genetics is that while genes influence tendencies towards antisocial personality and antisocial behaviour, they have relatively little influence on the probability of becoming officially designated as a delinquent at any particular court appearance. The designation of 'delinquent' is a characteristic influenced by the behaviour of co-offenders, police, parents, lawyers and judges, not merely by the behaviour of the young person.

Violence

9.14 Public debate about the implications of heritability for criminal responsibility often focuses on violent crime. Findings about genetic effects on violence are rare and inconsistent. Three studies report evidence of a value of zero for the heritability for violence,[8] whereas three studies report evidence that heritability for violence is about the same as for non-violent antisocial behaviour (0.50).[9]

9.15 Two main difficulties arise in the study of individual differences in official records of conviction for violent crime. First, even in the largest samples, official convictions for violent crime occur at very low rates. Inconsistent findings arise from such low indicators, because they cannot be reliably aggregated. Secondly, contrary to popular assumption, offenders designated 'violent' by virtue of court conviction are not necessarily the most serious, persistent criminals at the antisocial extreme. To illustrate, studies of murderers reveal that approximately half have lengthy histories of repeated assaults, rapes, robberies and other offence types, but the other half have committed a single extreme act after a lifetime free from crime. This indicates that the most serious of violent offences, homicide, as a legally constructed status, captures individuals likely to be quite heterogeneous in their genetic dispositions. Low base rates and heterogeneous participants may explain why studies using conviction data have found no heritability for violence.

9.16 The antidote to studying convictions is to use measures of violence that inquire about violent behaviours that have physical differences and cover a range of severity (fighting, hurting animals, robbery, hitting, aggravated assault using a weapon, gang-fighting, rape and domestic abuse). It is also helpful to use a reporting period long enough for research participants who are violently inclined to exhibit these relatively rare behaviours. This approach was used in two studies of self-reported violence in twins and siblings, which yielded significant estimates of heritability.[10]

9.17 Researchers in behavioural genetics usually do not single out violence for separate analysis. Researchers are dissuaded from doing so by the strong psychometric evidence that antisocial behaviour is a unified construct and therefore a separate research focus on violence is not warranted. Most studies of the structure of antisocial behaviour have

[8] Bohman, M., Cloninger, R., Sigvardsson, S. & von Knoring, A. L. (1982). Predisposition to petty criminality in Swedish adoptees. I. Genetic and environmental heterogeneity. *Arch. Gen. Psychiatr.* **39**, 1233–41; Mednick, S. A., Gabrielli, W. F. & Hutchings, B. (1984). Genetic factors in criminal behaviour: evidence from an adoption cohort. *Science* **224**, 891–3; Sigvardsson, S., Cloninger, C. R., Bohman, M. & von Korring, A. (1982). Predisposition to petty criminality in Swedish adoptees. III. Sex differences and validation of the male typology. *Arch. Gen. Psychiatr.* **39**, 1248–53.

[9] Heritability is reported as 50% in Cloninger, C. R. & Gottesman, I. I. Genetic and environmental factors in antisocial behaviour disorders. In Mednick, S. A., Moffitt, T. E. & Stack, S. A., editors. (1987). *The Causes of Crime: New Biological Approaches*. Cambridge: Cambridge University Press. pp. 92–109; 32% in Rowe, D. C., Almeida, D. M. Jacobson, K. C. (1999). School context and genetic influences on aggression in adolescence. *Psychol. Sci.* **10**, 277–80; 55% in Rushton, J. P. (1996). Self-report delinquency and violence in adult twins. *Psychiatr. Genet.* **6**, 87–9.

[10] Rowe, D. C., Almeida, D. M. & Jacobson, K. C. (1999). School context and genetic influences on aggression in adolescence. *Psychol. Sci.* **10**, 277–80; Rushton, J. P. (1996). Self-report delinquency and violence in adult twins. *Psychiatr. Genet.* **6**, 87–9.

suggested that items measuring physical aggression belong together with items assessing stealing, lying, fraud, vice, reckless irresponsibility and other forms of antisocial behaviour.[11]

9.19 Many studies in behavioural genetics have examined measures known to be strong, specific predictors of physical violence. For example, many have used the MPQ aggression scale and have reported strong estimates of heritability. The MPQ aggression scale measures attitudes, values, and beliefs that are consistent with approval of the use of physical violence.[12] Longitudinal research shows that the MPQ aggression scale empirically predicts future conviction for violent crime.[13]

9.20 Overall, the question of genetic influences for violent crime has not interested researchers in behavioural genetics as much as it has the general public. As a result, the evidence base is not sufficient to answer the question decisively. However, there is good evidence of heritability for antisocial traits and behaviours associated with risk for engaging in violent crime, and this suggests that heritable liability for violence is a reasonable hypothesis. An area overlooked by research in behavioural genetics is violence within relationships, a type of violence for which there are reliable aggregate measurement tools.

Sex differences

9.21 Differences between the sexes in heritability of antisocial behaviour may exist, but are small. It is unclear as yet whether these small differences should be interpreted as substantive, or as artefacts of sex differences in measurement. On balance, the results of model tests in large samples suggest that estimates of heritability may be slightly higher among males than females, but that sex-specific models of heritability cannot be justified.[14]

9.22 Estimates of heritability may be slightly smaller for females because measurements of antisocial behaviour among females represent less of the full range of antisocial severity, relative to measurements among males. The antisocial behaviour performed by females is less serious and less frequent than that of males, and females participate in antisocial activities for a much shorter period of the life course than males.[15] The relative rarity and brevity of females' antisocial behaviour makes it difficult to obtain strong aggregate measures of it, and this may influence estimates of heritability downwards for females.

---

[11] Moffitt, T. E., Krueger, R. F., Caspi, A. & Fagan, R. W. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology* **38**, 201–35; Blumstein, A., Cohen, J., Das, S. & Moitra, S. (1988). Specialization and seriousness during adult criminal careers. *J. Quan. Criminol.* **4**, 303–45; Farrington, D., Snyder, H. & Finnegan, T. (1988). Specialization in juvenile court careers. *Criminology* **26**, 461–85.

[12] Do not be misled by the names of measures; most scales labelled 'aggression', including the MPQ, do not measure *physical* aggression.

[13] Moffitt, T. E., Krueger, R. F., Caspi, A. & Fagan, R. W. (2000). Partner abuse and general crime: How are they the same? How are they different? *Criminology* **38**, 201–35.

[14] Gjone, H. & Stevenson, J. (1997). The association between internalizing and externalizing behaviour in childhood and early adolescence: Genetic or environmental common influences? *J. Abnorm. Child Psychol*. **25**, 277–86; Eaves, L. *et al*. (1997). Genetics and developmental psychopathology: 2. The main effects of genes and environment on behavioural problems in the Virginia study of adolescent behavioural development. *J. Child Psychol. Psychiatr*. **38**, 965–80; Taylor, J., McGue, M., Iacono, W. G. & Lykken, D. T. (2000). A behavioural genetic analysis of the relationship between the socialization scale and self-reported delinquency. *J. Pers.* **68**, 29–50; Finkle, D. & McGue, M. (1997). Sex differences and nonadditivity in the heritability on the Multidimensional Personality Questionnaire scales. *J. Pers. Soc. Psychol*. **72**, 929–38.

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

Current findings: molecular genetics

9.23 Strong estimates of heritability for a behavioural trait are generally taken to recommend further genetic research at the molecular level, to identify specific genes associated with the trait, and to ascertain their functions in relation to the brain. Antisocial behaviour is not currently a high priority for research in molecular genetics, because it is not as strongly heritable as disorders such as autism or schizophrenia. Nonetheless, molecular genetic research into antisocial behaviour will be pursued because antisocial behaviour forms part of a syndrome alongside two disorders that are currently of great interest in molecular genetics: Attention Deficit Hyperactivity Disorder and substance-dependence. Antisocial behaviour is a complex disorder, quantitatively distributed in the population, and as a result, antisocial behaviour must be influenced by many genes of small effect.

9.24 There is one study that has claimed to find an association between a genetic variant and forms of antisocial behaviour. In the 1980s, researchers became interested in a particular family in the Netherlands. Many of the male members of the family behaved in a notably violent and aggressive manner, and a considerable number had been involved in serious crime including rape and arson. Analysis of the family pedigree led researchers to look for a gene on the X chromosome that might be linked to this tendency in the men. They focused on a gene responsible for producing a protein called monoamine oxidase A (MAOA), involved in regulating the metabolism of serotonin in the brain. The male members of the family who engaged in aggressive behaviour were found to have abnormally low levels of MAOA in their bodies, and a defect in the gene was identified in these men in 1993.[16] They were also found to have lower than average IQs of around 80.

9.25 In August 2002, a study was published which investigated the link between MAOA and antisocial behaviour in a group of 500 male children.[17] The study examined the genotypes of the boys and identified a variant in the MAOA gene that was associated with high levels of MAOA activity in the brain, and another that was associated with low levels. The researchers found that children with the genotype conferring low levels of MAOA activity were significantly more likely to grow up to exhibit antisocial behaviour than those with high levels, but only if they were also maltreated and abused as children. In other words, it was the interaction between the genetic variant and the environment to which the children were exposed that was important. Children with low levels of MAOA activity who were not maltreated did not display antisocial behaviour. Nor did children with high levels of MAOA activity who were maltreated. The researchers stated that their findings 'may partly explain why some victims of maltreatment grow up to victimise others'. This research is particularly interesting because it demonstrates the connection between genetic and environmental influences on behaviour. We discuss its implications further in paragraphs 14.34 – 14.44 in the context of predicting future criminal and antisocial behaviour.

Current findings: research involving animals

9.26 The relevance of animal models of aggression for human antisocial behaviour has not yet been fully established. However, rodents offer the clear advantage of experimental manipulation to test effects of specific genes on aggression. Genetic research into

[16] Brunner, H. G., Nelen, M., Breakefield, X. O., Ropers, H. H. & van Oost, B. A. (1993). Abnormal behavior associated with a point mutation in the structural gene for monoamine oxidase A. *Science* **262**, 578–80.

[17] Caspi, A. *et al*. (2002). Role of genotype in the cycle of violence in maltreated children. *Science* **297**, 851-4.

aggression in animals (such as selective breeding of highly aggressive mouse strains, or studies of enhanced aggression in mice with 'knockout' manipulations of the genome) is proceeding rapidly, which should lead to findings in molecular genetics in the related area of human antisocial behaviour.[18]

9.27 One example of such a study was reported in May 2002, which claimed to have identified a genetic mutation that caused violent behaviour in mice.[19] The mutation, nicknamed 'fierce', has a range of effects in mice including extremely violent behaviour towards other mice, significant brain defects and physical differences such as decreased size, body fat and eye abnormalities.[20] A counterpart of the gene does exist in humans, but its precise function is not known.

Future directions for research

9.28 Quantitative research techniques are useful for revealing the contribution of environmental factors to antisocial outcomes in humans. This is a priority for future research because it may indicate the possibility of strategies for prevention. Researchers in behavioural genetics are beginning to include in their research, measures of the environmental factors that are thought to contribute to antisocial behaviour, such as the maltreatment of children, poverty and inconsistent discipline. Studies will hope to ascertain how the environments of young people interact with their genetic vulnerabilities, to exacerbate or protect against their risk for antisocial behaviour. Longitudinal research will follow samples of twins and adoptees as they age, to explore the changing balance between genetic and environmental factors that influence antisocial behaviour over the course of an individual's life. Because 'crime' itself is not inherited, researchers are working to investigate which features of personality and cognitive function may be associated with antisocial behaviour. With regard to molecular research techniques, research into MAOA-related genotypes is likely to continue (see paragraphs 9.24–9.25), along with research into other genes identified in research involving animals, and genes known to have functional significance in the brain. Importantly, quantitative and molecular work is converging on the possibility that genes act to augment the resistance of young people to environmental factors that would otherwise increase the likelihood of antisocial behaviour.

[18] For a review see Maxson, S. C. Genetic influences on aggressive behavior. In Pfaff, D. W., Berrettini, W. H., Toh. H. J. & Maxson, S. C., editors. (2000). *Genetic Influences on Neural Behavioural Functions*. New York: CRC Press. pp. 405–15.

[19] Young, K. A. *et al*. (2002). Fierce: a new mouse deletion of Nr2e1; violent behaviour and ocular abnormalities are background-dependent. *Behav. Brain Res.* **132**, 145–58.

[20] An interesting observation about this study is that the researchers did not set out to identify genes that influenced violent behaviour – they were studying reproduction. This illustrates the point made in Chapters 3 and 4 that findings related to behavioural traits are likely to emerge as a result of genetic research into disorders, making it difficult to prevent this kind of knowledge from being discovered.