

# 1 Advancing knowledge about the early prevention of adult antisocial behaviour

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The main aims of this book are to review what is known about the causes and prevention of adult antisocial behaviour. The book aims to specify what we know, what we do not know, and what we need to know, recommending priority research that would address key questions and fill key gaps in knowledge. The main aim of this introductory chapter is to set the scene for the more detailed chapters that follow by outlining some of the key topics, issues and questions arising in the early prevention of adult antisocial behaviour. This chapter defines the territory by briefly reviewing epidemiology, development, risk and protective factors, and prevention programmes.

Four types of prevention can be distinguished (Tonry and Farrington, 1995). Criminal justice prevention refers to traditional deterrence, incapacitation and rehabilitation strategies operated by law enforcement and criminal justice agencies. Situational prevention refers to interventions designed to reduce the opportunities for antisocial behaviour and to increase the risk and difficulty of committing antisocial acts. Community prevention refers to interventions designed to change the social conditions and social institutions (e.g. community norms and organisations) that influence antisocial behaviour in communities. Developmental prevention refers to interventions designed to inhibit the development of antisocial behaviour in individuals, by targeting risk and protective factors that influence human development (see Farrington, 2000a).

This book concentrates on early developmental prevention programmes, including those implemented in pregnancy and infancy, parenting programmes, preschool programmes, individual skills training, and school programmes. Many of these involve primary prevention, targeting unselected individuals in the whole community, but secondary prevention programmes targeting children at risk are also reviewed. The focus of the book is on risk factors and early prevention in childhood and adolescence; for reviews of risk factors and early interventions for conduct disorder and delinquency, see Farrington (1999) and Rutter, Giller and Hagell (1998).

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## **Definition and measurement**

### *Definition of antisocial behaviour*

There is clearly a syndrome of adult antisocial behaviour defined by a cluster of antisocial symptoms. This syndrome is given different names in different countries and different classification systems: antisocial personality disorder in DSM-IV (American Psychiatric Association, 1994), dissocial personality disorder in ICD-10 (World Health Organisation, 1992) and psychopathic disorder in the English Mental Health Act 1983, for example.

Both types of behaviour and features of personality are included in the antisocial behaviour syndrome. Types of behaviour include property crimes such as burglary, violent crimes, drug use, heavy drinking, drunk or reckless driving, sexual promiscuity or risky sex behaviour, divorce/separation or unstable sexual relationships, spouse or partner abuse, child abuse or neglect, unemployment or an unstable employment history, debts, dependence on welfare benefits, heavy gambling, heavy smoking, and repeated lying and conning. Personality features include impulsiveness and lack of planning, selfishness and egocentricity, callousness and lack of empathy, lack of remorse or guilt feelings, low frustration tolerance and high aggressiveness.

An important question is the relative importance of behavioural and personality symptoms in defining antisocial personality disorder. Hare and his colleagues (e.g. Hare, Hart and Harpur, 1991) have consistently criticised the DSM criteria for antisocial personality as too behavioural and insufficiently concerned with personality features. Hare's Psychopathy Checklist (PCL-R) distinguishes two factors. Factor 1 consists of personality features such as egocentricity, lack of remorse, and callousness, while factor 2 describes an impulsive, antisocial, and unstable lifestyle. The problem is that some features of an antisocial lifestyle (e.g. unemployment and dependence on welfare benefits) may either reflect an antisocial personality or may be caused by circumstances outside the person's own control. Because of this, it is desirable to include both behavioural and personality features in the definition of antisocial personality.

Another important question is whether individuals differ qualitatively (in kind) or quantitatively (in degree) in antisocial personality (Clark, Livesley and Morey, 1997). People can be scored according to their number of symptoms. For example, Robins and her colleagues (e.g. Robins and Price, 1991) have consistently argued that the number of childhood conduct disorder symptoms predicts the number of adult antisocial behaviour symptoms, rather than any specific childhood behaviour

predicting a specific adult behaviour. The key problem is where to set the boundary between normal and pathological, or between health and illness. Existing boundaries depend largely on clinical judgement. For example, according to DSM-IV, 'only when antisocial personality traits are inflexible, maladaptive and persistent and cause significant functional impairment or subjective distress do they constitute Antisocial Personality Disorder' (American Psychiatric Association, 1994, p. 649). Far more is known about the early prevention of particular types of antisocial behaviour than about the early prevention of antisocial personality disorder or psychopathy.

#### *Measurement of antisocial behaviour*

Antisocial behaviour can be measured in a variety of ways. Interviews by psychiatrists are necessary to yield psychiatric diagnoses in Great Britain, where explicit diagnostic criteria are not as widely used as in North America. However, psychiatrist interviews are not very practical for large-scale epidemiological studies. One possible strategy is to use a two-stage procedure in which the population is initially screened using brief symptom questionnaires (e.g. Bebbington *et al.*, 1981). Then, more intensive clinical interviews can be given to all those with high symptom scores and to a representative sample of those with low scores.

Another possible method is to use an interview designed for non-clinicians, such as the NIMH Diagnostic Interview Schedule used in the Epidemiological Catchment Area project (Robins and Regier, 1991). Ratings or checklists completed by informants such as institutional staff can also be used, based on interviews and records, as in the case of the PCL-R (Hare, 1991). Alternatively, semi-structured interviews with informants such as relatives or close friends can be used, as with the Standardized Assessment of Personality (Pilgrim and Mann, 1990), or psychological tests and self-completion questionnaires can be used (e.g. Blackburn, 1975).

It is important with all measurement techniques to assess validity and reliability. However, one problem in assessing validity is that the external criterion for antisocial personality disorder or psychopathy is often based on psychiatric diagnoses, which may have low reliability (Malgady, Rogler and Tryon, 1992). It is especially important to measure the predictive validity of instruments given at a relatively early age or stage of development.

In this chapter, I will refer to results obtained in the Cambridge Study in Delinquent Development, which is a prospective longitudinal survey of 411 South London males from age 8 to age 46 (Farrington, 1995,

2002c). At age 32, a measure of antisocial personality was devised, based on the following twelve items: convicted in the last five years, self-reported offender, involved in fights, drug-taker, heavy drinker, poor relationship with parents, poor relationship with wife/cohabitee, divorced/child living elsewhere, frequent unemployment, anti-establishment attitude, tattooed, and impulsive (Farrington, 1991). These were measured in a structured social interview. The reliability of this scale was 0.71, and the worst quarter of the males had four or more adverse features out of twelve.

#### *Inter-relationships between behaviours*

In general, all the behavioural and personality symptoms listed above tend to be intercorrelated, since people who show one of them have an increased risk of also showing any other. For example, the two factor scores of the PCL-R are highly intercorrelated (over 0.5: Hare *et al.*, 1991), and the total PCL-R score is highly correlated with the diagnosis of antisocial personality disorder (0.67 in Hare, 1985). Comorbidity is a common finding, and it is assumed that all of the symptoms reflect the same underlying theoretical construct. However, it is important to quantify the degree of versatility in antisocial behaviour, and to assess whether it is more reasonable to assume two or more underlying constructs rather than only one. Another important question is whether conclusions are different with continuous as opposed to dichotomous measures of symptoms.

To the extent that intercorrelated clusters of symptoms are identified within the general category of antisocial behaviour, it may be useful to distinguish typologies of individuals. For example, Moffitt (1993) distinguished between 'life-course-persistent' individuals, who began their antisocial behaviour at an early age and persisted for a long time, and 'adolescence-limited' ones who began later and desisted earlier. However, it is unclear how far these categories differ in degree rather than in kind.

### **Epidemiology and development**

#### *Epidemiology*

It is important to establish the prevalence of antisocial symptoms, and of antisocial personality disorder, at different ages. It is useful to determine the peak ages of different types of antisocial behaviour, and the peak ages for acceleration and deceleration in prevalence. Information is also

needed about the frequency and seriousness of behaviours at different ages. Other important questions centre on how prevalence, frequency and seriousness vary with gender, ethnicity, and geographical area, and over time. Perhaps the most extensive data on the epidemiology of antisocial behaviour was provided by the Epidemiological Catchment Area Project (Robins, Tipp and Przybeck, 1991). For example, the estimated life-time prevalence of antisocial personality disorder in the USA was 7.3 per cent of males and 1.0 per cent of females. Similarly, Bland, Orn and Newman (1988) estimated that the life-time prevalence was 7 per cent of males in Edmonton, Canada. However, in Great Britain, the current prevalence of antisocial personality disorder was 1 per cent of males and 0.2 per cent of females in a national survey (Singleton *et al.*, 2002). The epidemiology of antisocial personality disorder has been most extensively reviewed by Moran (1999). In chapter 2, Jeremy Coid reviews epidemiological data and its implications for early prevention.

Another important epidemiological question concerns how far antisocial behaviour is concentrated among a small segment of the population. For example, in the Cambridge Study in Delinquent Development, about 6 per cent of the cohort males accounted for half of all the convictions up to age 32 (Farrington and West, 1993). These 'chronic offenders' were particularly likely to show other symptoms of antisocial personality, such as an unstable employment record, spouse assault, involvement in fights, drug-taking, heavy drinking, and anti-establishment attitudes. It is useful to quantify the degree of concentration of antisocial behaviour using the Lorenz curve and the Gini coefficient (Wikström, 1991, p.29).

### *Development*

It is important not only to establish the prevalence of antisocial behaviour but also key features of antisocial careers such as the age of onset, the probability of persistence after onset, the duration of antisocial behaviour, and the age of desistance. According to Robins (1978), most boys who eventually developed antisocial personality disorder showed signs of conduct disorder (truancy, stealing and classroom disciplinary problems) as soon as they began attending school. This suggests that the antisocial syndrome has a very early age of onset, and conversely that early prevention is useful. Many other features of antisocial careers could be measured, such as acceleration and deceleration in the frequency of committing antisocial behaviour, escalation and de-escalation in seriousness, diversification, switching, and stabilisation (Loeber and LeBlanc, 1990). It may be difficult to distinguish between true desistance and intermittency or periods of remission.

More is known about criminal careers than about more general antisocial careers. For example, in the Cambridge Study up to age 40, the average age of the first conviction was 18.6, the average age of the last conviction was 25.8, the average length of the criminal career was 7.2 years, and the average number of offences leading to conviction was 4.6 (Farrington, Lambert and West, 1998). The males first convicted at the earliest ages (10–13) tended to become the most persistent offenders, committing an average 8.8 offences leading to convictions in an average criminal career spanning 9.9 years. It is generally true that an early onset of antisocial behaviour predicts a long and serious antisocial career.

It is important to study developmental sequences in antisocial careers, where one type of behaviour tends to be followed by another. Three types of sequences can be distinguished (Farrington, Loeber, Elliott *et al.*, 1990). First of all, different acts following each other may be different behavioural manifestations of the same underlying construct (e.g. antisocial personality) at different ages. Second, different acts may be different behavioural manifestations of the same or similar underlying constructs at different ages and also part of a developmental sequence, where one act is a stepping stone towards or facilitates another (e.g. where smoking cigarettes leads to marijuana use). Third, different acts may be indicators of different constructs and part of a causal sequence, where changes in an indicator of one construct cause changes in an indicator of a different construct (e.g. where low attainment leads to truancy). A further problem is that the same behaviour at different ages may reflect different underlying constructs (e.g. compare sexual intercourse at age 12, which is deviant, with sexual intercourse at age 25, which is normal).

#### *Intragenerational continuity*

It is important to assess the degree of continuity and stability in antisocial behaviour over time. Several researchers have reported that childhood conduct disorder tends to predict adult antisocial personality disorder. For example, in an Inner London study Zoccolillo *et al.* (1992) found that almost half of the males with three or more symptoms of conduct disorder at age 9–12 showed persistent antisocial behaviour after age 18 and fulfilled the criteria for adult antisocial personality disorder (see also Offord and Bennett, 1994; Rey *et al.*, 1995; Rutter *et al.*, 1994; Storm-Mathisen and Vaglum, 1994). In the Cambridge Study, the antisocial personality score at age 8–10 correlated 0.38 with the score at age 18, and the score at age 18 correlated 0.55 with the score at age 32 (Farrington, 1991).

These correlations help to quantify the degree of stability in the relative ordering of individuals as opposed to the degree of change. They do not indicate absolute stability in antisocial behaviour. For example, in the Cambridge Study the prevalence of marijuana use decreased significantly between ages 18 and 32, but there was a significant tendency for the users at age 18 also to be users at age 32 (Farrington, 1990). Conversely, binge drinking increased significantly between ages 18 and 32, and there was again significant consistency over time. Hence, relative stability often coincided with absolute change. It may be that stability varies according to the initial level of antisocial behaviour; for example, the most antisocial people may be the most stable.

Continuity refers to relationships between different behavioural manifestations over time. For example, hyperactivity at age 2 may predict cruelty to animals at age 6, which in turn predicts conduct disorder at age 10, which in turn predicts burglary at age 14, violence at age 18, partner abuse in the 20s and child abuse in the 30s. The major problem is how to establish that one behaviour leads to another in some way, since any behaviour A tends to be followed by many other behaviours (B, C, D...) with varying probabilities after varying time intervals.

#### *Intergenerational continuity*

Antisocial parents tend to have antisocial children. For example, in the Cambridge Study, 63 per cent of boys with convicted fathers were themselves convicted (odds ratio = 3.9), as were 61 per cent of boys with convicted mothers (odds ratio = 2.8). Convictions were highly concentrated in families; about 6 per cent of the cohort families accounted for about half of all the convictions of all family members (Farrington, Barnes and Lambert, 1996). Having a convicted parent at age 10 was the best single predictor of antisocial personality at age 32 (Farrington, 2000b).

It is unclear how far there is specific transmission of types of antisocial behaviour as opposed to general transmission of antisocial tendencies. For example, it is not clear that violent parents tend specifically to have violent children, or that drug-using parents tend specifically to have drug-using children, over and above the general tendency for antisocial parents to have antisocial children. Nor is it clear how far this transmission is attributable to genetic as opposed to environmental factors; behaviour-genetic designs (e.g. twin or adoption studies) are needed to disentangle these factors. Chapter 4 by Terrie Moffitt and Avshalom Caspi discusses intergenerational continuity in more detail, with special reference to partner violence.

### **Risk and protective factors**

Risk factors are prior factors that predict an increased probability of antisocial behaviour. Longitudinal data are required to establish the relative ordering of risk factors and antisocial outcomes. Few longitudinal studies have explicitly investigated risk factors for antisocial personality; the most relevant available information usually concerns risk factors for offending. Apart from the seminal work of Robins (1979), 'we have relatively few studies that have measured the effects of these [child and family] risks, prospectively measured, on adult personality disorder symptoms' (Cohen, 1996, p.126). However, in the Cambridge Study, risk factors for antisocial personality at age 32 (Farrington, 2000b) and for chronic offending (Farrington and West, 1993) were investigated.

Few studies have conducted research on risk factors for career features such as onset, persistence, escalation, and desistance as opposed to risk factors for antisocial behaviour in general. It is sometimes difficult to disentangle risk factors from antisocial outcomes. For example, impulsiveness may be regarded as a cause of antisocial behaviour or as an element of the antisocial personality syndrome. Because of the overall emphasis on prevention in this book, this chapter will concentrate on potentially changeable risk factors that could have causal effects on antisocial behaviour. It is important to study the independent, interactive, and sequential effects of risk factors on antisocial behaviour, but these factors will be briefly reviewed one by one in this chapter. Only a brief review of risk factors can be presented here; chapter 3 by Rolf Loeber, Stephanie Green and Ben Lahey provides a more extensive review of risk factors for antisocial personality.

#### *Biological and individual risk factors*

A number of biological risk factors for antisocial behaviour have been identified (Raine, 1993). How far these are changeable is not always clear. For example, there may be some genetic contribution. In the Minnesota study of identical twins brought up apart, the heritability of adult antisocial personality disorder was estimated to be 0.28 (Grove *et al.*, 1990). Neurochemical factors (e.g. testosterone), neurotransmitters (e.g. serotonin), psychophysiological factors (e.g. a low heart rate), and neuropsychological deficits (e.g. in executive functions) have all been linked to antisocial behaviour (Raine *et al.*, 1997). Other relevant factors include head injuries, pregnancy and birth complications, low birth weight of the child, and substance use in pregnancy by the mother (e.g. Kolvin *et al.*, 1990; Raine, Brennan and Mednick, 1994).



A major cluster of individual risk factors includes hyperactivity, impulsivity, attention problems, clumsiness, daring or risk-taking, and other elements of Attention Deficit Hyperactivity Disorder (ADHD). These factors are often closely linked to childhood conduct disorder, but hyperactivity-impulsivity-attention deficit and conduct problems at age 8–10 were independent predictors of later convictions in the Cambridge Study (Farrington, Loeber and van Kammen, 1990). Lynam (1996) argued that children who had both hyperactivity-impulsivity-attention deficit and conduct problems were especially at risk of becoming psychopaths. Also in the Cambridge Study, daring and poor concentration were among the best independent predictors of chronic offenders (Farrington and West, 1993).

The most extensive research on different measures of impulsiveness was carried out in another longitudinal study of males (the Pittsburgh Youth Study) by White *et al.* (1994). The measures that were most strongly related to self-reported delinquency at ages 10 and 13 were teacher-rated impulsiveness (e.g. ‘acts without thinking’), self-reported impulsiveness, self-reported under-control (e.g. ‘unable to delay gratification’), motor restlessness (from videotaped observations), and psychomotor impulsiveness (on the Trail Making Test). Generally, the verbal behaviour rating tests produced stronger relationships with offending than the psychomotor performance tests, suggesting that cognitive impulsiveness (based on thinking processes) was more relevant than behavioural impulsiveness (based on test performance).

Other important individual risk factors for antisocial behaviour include low intelligence, low attainment, low empathy, low guilt, unpopularity, and poor interpersonal skills (Blackburn, 1993). For example, in the Cambridge Study, low non-verbal IQ and low junior school attainment were strong childhood predictors of antisocial personality at age 32 (Farrington, 2000b). Similar results have been obtained in other projects (Lynam, Moffitt and Stouthamer-Loeber, 1993; Wilson and Herrnstein, 1985). Delinquents often do better on non-verbal performance tests, such as object assembly and block design, than on verbal tests (Walsh, Petee and Beyer, 1987), suggesting that they find it easier to deal with concrete objects than with abstract concepts.

#### *Family interaction and socio-economic risk factors*

Numerous family factors predict a child’s later antisocial behaviour. Having criminal or antisocial parents has already been mentioned. Important family interaction factors include inconsistent, harsh or abusive parenting, cold or rejecting parental attitude, poor parental supervision or

monitoring, low parental involvement with the child, separation/divorce and parental conflict (Farrington, 2002b; Smith and Stern, 1997). For example, in the Cambridge Study, poor parental supervision was an important childhood predictor of both chronic offending and antisocial personality at age 32. However, poor child-rearing (harsh or erratic attitude or discipline) predicted chronic offending but not antisocial personality, and separation from a parent (usually the father) predicted antisocial personality but not chronic offending (Farrington, 2000b; Farrington and West, 1993).

Numerous socio-economic factors predict a child's later antisocial behaviour, including low family income, large family size (which is also a family interaction factor), poor housing, a teenage mother, dependence on welfare benefits, and unemployed parents. For example, in the Cambridge Study, low family income, large family size (four or more biological siblings) and low socio-economic status (but not poor housing) were important childhood predictors of chronic offending and antisocial personality at age 32 (Farrington, 2000b; Farrington and West, 1993).

#### *Peer, school and community risk factors*

It is well established that having delinquent friends is an important correlate of offending; in the Cambridge Study, 75 per cent of chronic offenders had highly delinquent friends at age 14, compared with 33 per cent of non-chronic offenders and 16 per cent of non-offenders (Farrington and West, 1993). What is less clear is how far antisocial peers encourage and facilitate antisocial behaviour, or whether it is merely that "birds of a feather flock together". Delinquents may have delinquent friends because of co-offending, which is particularly common under age 21 (Reiss and Farrington, 1991). Interestingly, withdrawal from the delinquent peer group seemed to be an important influence on desistance in the Cambridge Study (West and Farrington, 1977).

It is also well established that delinquents disproportionately attend high delinquency rate schools, which have high levels of distrust between teachers and students, low commitment to the school by students, and unclear and inconsistently enforced rules (Graham, 1988). In the Cambridge Study, attending a high delinquency-rate school at age 11 significantly predicted both chronic offending and antisocial personality at age 32 (Farrington, 2000b; Farrington and West, 1993). However, what is less clear is how far the schools themselves influence antisocial behaviour by their organisation, climate and practices, and how far the concentration of offenders in certain schools is mainly a function of their intakes.