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The epidemiology of antisocial personality disorder

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Abstract This paper reviews the current state of knowledge about the frequency, natural history, risk factors and associations of antisocial personality disorder. Important recent developments are discussed, and where possible, findings have been tabulated. Epidemiological surveys have shown that antisocial personality disorder is a common disorder, with a prevalence rate of between 2 and 3% among community samples, rising to 60% among male prisoners. Antisocial personality disorder is a chronic condition, and is associated with a multitude of medical and social problems. These include substance abuse, deliberate self harm and crime. Genetic and environmental factors have been implicated in the aetiology of the disorder. However, despite the large amount of research into antisocial personality disorder, longitudinal data are missing and the validity of the diagnosis, therefore, remains questionable. The paper concludes with recommendations for future research.

Introduction

Psychiatry has a long tradition of ambivalence towards the personality disorders. Whilst their importance as a source of impairment is acknowledged (Merinkangas and Weissman 1986; Nakao et al. 1992), they have been criticised for being pejorative terms (Lewis and Appleby 1988) that do not accurately reflect the nature of abnormal personality and that instil therapeutic nihilism in clinicians (Livesley et al. 1994). Nevertheless, the concept has a long history, reflecting the medical profes-

sion's preference for categories over dimensions. Of all the personality disorders, arguably the most controversial is antisocial personality disorder (Frances 1980). Currently, as a result of a highly publicised case of murder perpetrated by an individual with an antisocial personality disorder, the medical response to the condition has become the subject of a national political debate in the UK (Eastman 1999). Doubts about whether psychiatrists should be responsible for patients with the disorder are compounded by the fact that evidence for the treatability of this condition is lacking (Dolan and Coid 1993).

The term 'antisocial personality disorder' (ASPD) was introduced by the American Psychiatric Association in 1980, and represented an attempt to operationalise the older much-berated term 'psychopathy'. The category has been criticised for over-emphasising criminal acts at the expense of traditional 'Cleckleyan' concepts of psychopathy (Widiger and Corbitt 1995), but it is the only personality disorder diagnosis that has been shown to be used with adequate levels of inter-rater reliability in routine clinical practice (Mellsop et al. 1982). In addition, the DSM criteria for antisocial personality disorder are the only ones to have been derived from empirical research (Robins 1966).

Over the past decade, interest in the epidemiology of antisocial personality disorder has flourished and a large literature has accumulated. The present paper provides an overview of this literature and considers research in the following areas: descriptive epidemiology, natural history, associated conditions, risk factors and associated burden.

Descriptive epidemiology

Community

Antisocial personality disorder has been the most consistently studied personality disorder in community psychiatric surveys. The lifetime prevalence rates for

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antisocial personality disorder, obtained from 'third generation' surveys (Dohrenwend and Dohrenwend 1982) are listed in Table 1. Before comparing rates, it should be noted that studies have employed a variety of methods of data collection and diagnostic criteria. Nevertheless, the findings are remarkably consistent, with the majority of studies reporting prevalence rates of between 2 and 3% of the sampled population. The sociodemographic correlates of antisocial personality disorder that emerge from these studies are of higher prevalence rates among:

- 1. Males (estimates range from 6:1 to 8:1, male:female)
- 2. Younger age groups (25–44 years in the Epidemiological Catchment Area study)
- 3. The poorly educated (in the National Comorbidity Survey, the odds of developing antisocial personality disorder for those leaving formal education at 11 years was almost five times that of those remaining in education until 15 years)

Lifetime prevalence rates decline with increasing age, and this has been cited as evidence that the prevalence of the disorder is increasing (Paris 1997). However, the disorder carries an increased risk of early unnatural death, and therefore rates in older persons are likely to be reduced by mortality more than rates in younger persons.

Primary care

Although there is a large literature on the epidemiology of mental disorders in primary care (Sartorius et al. 1993), few studies have examined the prevalence of antisocial personality disorder in this setting (Table 2). The prevalence rates vary from 5 to 11%, although only Sato and Takeichi's study (1993) used a randomly selected sample. They were unable to identify any cases of antisocial personality disorder, and attributed this finding to culture-specific factors, and the existence of fewer sociodemographic risk factors for the development of the disorder in Japanese society.

Psychiatric settings

Studies of personality disorder in psychiatric settings proliferate (de Girolamo and Reich 1993). However, owing to differences in the methodology employed by these studies, prevalence rates vary markedly. Studies that have examined the prevalence of antisocial personality disorder in selected samples of psychiatric patients, for example drug users, have given rise to inflated estimates of the disorder in secondary care. Similarly, studies of patients from specialist treatment settings, for example therapeutic communities, not un-

Table 1 Lifetime prevalence of antisocial personality disorder (ASPD; DSM III) in community surveys^a

Author	Location	Sample	Procedure	Lifetime prevalence of ASPD
Weissman & Myers (1980)	USA	511	SADS-L	0.2%
Baron et al. (1985)	USA	376	SIB SADS	0.5%
Bland et al. (1988a)	Canada	3258	DIS	3.7%
Hwu et al. (1989)	Taiwan	11004	DIS	0.14%
Reich et al. (1989)	USA	235	PDQ	0.4%
Wells et al. (1989)	New Zealand	1498	DIS	3.1%
Lee et al. (1990)	Korea	3134 (urban) 1966 (rural)	DIS	2.1% (urban) 0.9% (rural)
Zimmerman & Coryell (1990)	USA	697	PDQ SIDP	0.9% 3.0%
Robins & Regier (1991)	USA (ECA)	18571	DIS	2.4%
Maier et al. (1992)	Germany	447	SCID-II	0.2%
Chen et al. (1993)	Hong Kong	7229	DIS	2.78% (m) 0.53% (f)
Levav et al. (1993)	Israel	2741	SADS-I	0.7%
Kessler et al. (1994)	USA (NCS)	8098	CIDI	3.5%
Samuels et al. (1994)	USA	810	DSM III rating scale	1.5%
Swanson et al. (1994)	Canada	3258	DIS	3.7%

^a From 'Antisocial personality disorder: an epidemiological perspective' (Moran 1999). Reproduced with permission from Gaskell publications

SADS Schedule for Affective Disorders and Schizophrenia (SADS-L, Lifetime version; SADS-I, Israeli version), SIB Schedule for Interviewing Borderlines, DIS Diagnostic Interview Schedule, PDQ Personality Disorders Questionnaire, SIDP Structured Interview for DSM-III Personality Disorders, SCID-II Structured Clinical Interview for DSM-III-R Personality Disorders, CIDI Composite International Diagnostic Interview, NCS National Comorbidity Survey, ECA Epidemiological Catchment Area Study

Table 2 Studies of antisocial personality disorder in primary care

Author	Location	Sample	Procedure	Findings
Schulberg et al. (1985)	Pittsburgh, USA	294 primary medical care patients	DIS	Prevalence rate of ASPD: 5.8%
Smith et al. (1991)	Arkansas, USA	118 primary care patients with somatisation disorder	DIS SCID-II	Prevalence rate of ASPD (DIS): 11%; 3:1, M:F
Sato & Takeichi (1993)	Saga City, Japan	172 randomly selected primary care attenders	DIS-JM	No cases of ASPD detected
Hueston et al. (1996)	Wisconsin, USA	93 primary care patients	SCID-II	70% were at 'high risk' for personality disorder 22% at 'high risk' from ASPD
Barry et al. (1997)	Wisconsin, USA	1898 primary care patients	DSM-III-R criteria	Prevalence rate of ASPD: M: 8%; F: 3%

DIS Diagnostic Interview Schedule, (DIS-JM = Japanese modified), SCID-II Structured Clinical Interview for DSM-III-R Personality Disorders

expectedly report high prevalence rates of all personality disorders (Dolan, Evans and Norton 1995). Studies of unselected admissions to psychiatric hospitals provide a more balanced picture of the distribution of a condition, and some of the major studies of this kind are listed in Table 3. These studies show that antisocial personality disorder is a rare category of personality disorder in general psychiatric settings, with the majority of listed studies quoting prevalence rates of between 1 and 3%. UK hospital admission data confirm this impression and show that, over the period 1989–1995, sociopathic personality disorder (the ICD-9 equivalent of ASPD) constituted a small but constant proportion of total finished consultant episodes (about 0.2–0.3%) (Moran 1999).

Prison settings

The estimation of the prevalence of antisocial personality disorder in prisons is fraught with potential problems. The emphasis that the definition of the disorder places on antisocial acts means that antisocial personality disorder overlaps greatly with criminality. In addition, several factors significantly affect the prevalence rate of any mental disorder in prison, making comparison of estimates extremely difficult. These include: the location of the study, the type of prison (sentenced or remand), the type of prisoner studied, and other factors that determine the actual numbers of mentally disordered offenders entering the criminal justice system at any point in time. Many of the prison surveys conducted

Table 3 Surveys of the prevalence of antisocial personality disorder in psychiatric patients^a

Author	Country	Sample	Method of assessment	Findings
Kass et al. (1985)	USA	609 new consecutive outpatients	DSM-III rating scale	51% had a personality disorder Prevalence of ASPD = 2%
Koenigsberg et al. (1985)	USA	2,462 patients from outpatient clinic, inpatient unit, 'walk-in' clinic and consultation-liaison service	Clinical diagnosis	36% had a personality disorder Prevalence of ASPD = 2%
Cutting et al. (1986)	UK	100 consecutive admissions with major affective disorder or psychosis	SAP	44% had abnormal personality disorder 3% had sociopathic traits
Dahl (1986)	Norway	103 consecutive admissions to hospital	SADS	45% had a personality disorder Prevalence of ASPD = 18.2%
Alnaes & Torgesen (1988)	Norway	298 consecutive outpatients	SIDP & SCID	81% had a personality disorder No cases of ASPD found
Pilgrim & Mann (1990)	UK	120 consecutive new admissions to psychiatric hospital	SAP	36% of patients had a personality disorder Prevalence of dissocial PD = 1.6%
Jackson et al. (1991)	Australia	112 inpatients	SIDP	46% had a personality disorder. ASPD occurred in 20% of schizophrenic and 15% of manic patients
Oldham et al. (1995)	USA	200 consecutive applicants for inpatient care or psychotherapy	SCID-II PDE	3.5% ASPD

^a From 'Antisocial personality disorder: an epidemiological perspective' (Moran 1999). Reproduced with permission from Gaskell publications

SADS Schedule for Affective Disorders and Schizophrenia,

SIDP Structured Interview for DSM-III Personality Disorders, SCID-II Structured Clinical Interview for DSM-III-R Personality Disorders, SAP Standardised Assessment of Personality, PDE Personality Disorder Examination

prior to 1980 were methodologically flawed, making it hard to draw any conclusions about the distribution of personality disorder among prisoners (Coid 1984). Over the past decade, however, there have been a number of well-conducted surveys, which have used randomly selected samples and standardised assessment procedures (see Table 4). These studies show that antisocial personality disorder is extremely common in prisons with prevalence rates as high as 40-60% among the male sentenced population. As with community samples, the disorder is more common among young men and there is high co-morbidity with substance misuse. The recently completed ONS survey of psychiatric morbidity among prisoners in England and Wales (Singleton et al. 1998) is an important recent addition to the forensic literature. Among those who had a clinical interview, the prevalence of any personality disorder was 78% for male remand, 64% for male sentenced and 50% for female prisoners. Antisocial personality disorder had the highest prevalence of any category of personality disorder, with 63% of male remand prisoners, 49% of sentenced prisoners and 31% of female prisoners. Such high prevalence estimates raise important questions about the validity of the diagnosis and the medicalisation of criminality. However, in defence of the diagnosis, Rob-

ins (1991) has shown that on the basis of Epidemiological Catchment Area (ECA) data, the occurrence of a significant arrest record is not predictive of an antisocial personality disorder diagnosis. In fact, in the ECA study, the most common symptoms of the disorder were job troubles, violence and marital difficulties, and not criminality.

Natural history

Assumptions about the natural history of personality disorders are inherent in their description. ICD-10 diagnostic guidelines refer to abnormal behaviour patterns that are 'enduring' and that appear during childhood or adolescence and continue into adulthood (World Health Organisation 1992). DSM-IV postulates that personality disorders have an onset that can be traced back to early adulthood and that they are disorders which lead to impairment in social and occupational functioning (American Psychiatric Association 1994). There is, however, a dearth of empirical data regarding the longitudinal course of personality disorders (Drake and Vaillant 1988; Perry 1993; Stone 1993). This may be due to difficulties in achieving stable case identification, high

Table 4 Prison surveys (sentenced and remand) of antisocial personality disorder

Author	Location	Sample	Procedure	Findings
Hare (1983)	2 Canadian prison populations: a) federal medium secure unit b) provincial prison	246 white male inmates	PCL Clinical interview using DSM-III criteria	Point prevalence rate ASPD for sample: a) 33.3% b) 41.5% Overall prevalence of ASPD: 39%
Robertson (1987)	Winnipeg remand centre, Canada	100 consecutive female arrestees	Clinical interview with a psychologist	Point prevalence DSM-III ASPD: 60%
Côté & Hodgins (1990)	Sample of Quebec penitentiaries	Random sample of 650 male inmates	DÍS	Lifetime prevalence rate of DSM-III ASPD 61.5%
Bland et al. (1990)	2 correctional centres in Edmonton, Canada	Random sample of 180 male inmates and 1006 age-matched controls	DIS	Lifetime prevalence rate of DSM-III ASPD: 57% (standardised prev. ratio 7 times that of gen pop)
Côté & Hodgins (1992)	Sample of Quebec penitentiaries	87 homicide convictions selected from 1990 study	DIS	No significant difference in freq. of ASPD
Andersen et al. (1996)	Western prison of Copenhagen	2 random samples of newly admitted Danish-born prisoners (males & females)	PCL-R Case notes	Lifetime prevalence ICD-10 dissocial personality disorder 17%
Jordan et al. (1996)	Correctional institute for women, Raleigh, NC	Unselected 'census' of all women entering prison over $14/12$; $n = 805$	DIS CIDI DIPD	Lifetime prevalence rate DSM-III-R ASPD 11.9%
Teplin et al. (1996)	Cook Co. Dep. of corrections, Chicago	Stratified random sample of 1272 female arrestees awaiting trial	DIS	Lifetime prevalence DSM- III-R ASPD 13.8%
Singleton et al. (1998)	All prisons in England and Wales	505 randomly selected sentenced and remand prisoners	SCID-II	ASPD had highest prevalence of any category of PD: 63% male remands 49% male sentenced 31% females

attrition rates, and also the costs of running large prospective studies.

Most of what is known about the natural history of antisocial personality disorder is derived from Robins' longitudinal study of children referred to a child guidance clinic (Robins 1966). From this study, the criteria for sociopathic personality disorder (the forerunner to ASPD) were devised. Robins judged that at 30 years (when subjects were in their fourth or fifth decade), 12% were in remission, 27% showed a greatly reduced range in the severity of antisocial behaviour, although, 60% still showed little improvement. The following additional conclusions have been drawn from Robins' research:

- 1. Adult antisocial behaviour virtually requires childhood antisocial behaviour. ECA data showed that 95% of males with four or more adult symptoms had at least one childhood symptom (Robins 1991).
- 2. Most antisocial children do not become antisocial adults. (In Robins' original study, only 27% of males showing three or more childhood symptoms also showed four or more adult symptoms).
- 3. The variety of antisocial behaviour is a better predictor of adult antisocial behaviour than is any particular behaviour.
- 4. Adult antisocial behaviour is better predicted by childhood behaviour than by family background or social class of rearing.

The best prospective data on antisocial personality disorder come from Black et al.'s (1995a) follow-up of 71 psychiatric inpatients with DSM-III antisocial personality disorder. The study is notable for its relatively low attrition rate (96% of the original sample were traced and 48% were interviewed), the length of follow-up (29 years), the inclusion of control groups (depressed and surgical controls), and the examination of broader psychosocial outcome, by using the Global Assessment Scale (GAS). At follow-up, 42% of subjects were judged to be unimproved, 31% improved but not remitted, 27% remitted and 22.5% had attempted suicide. By comparison with depressed and surgical controls, the subjects had poorer GAS ratings in all areas, with the exception of residential status (Black et al. 1995b).

Personality disorders are associated with suicidal behaviour, although the magnitude of risk varies considerably between specific categories of disorder (Dirks 1998). Antisocial personality disorder is associated with both attempted suicide and completed suicide, although recent research has shown that the risks are perhaps not as high as were once thought (Woodruff et al. 1971). Using the psychological autopsy method and a casecontrol design, Lesage et al. (1994) were able to examine the association of specific mental disorders with suicide among a group of young men from Canada. At least one axis I disorder was identified in 88% of suicide subjects (odds ratio, OR: 12.3; 95% confidence interval, CI: 5.3– 12.5) and at least one axis II disorder was identified in 57% of suicide subjects (OR: 4; 95% CI: 2-7.9). The most common axis II disorders were borderline (OR: 9.3; 95% CI: 2.6–33) and antisocial personality disorders

(OR: 4.1; 95% CI: 1–15.4). Using a biographical reconstructive interview conducted for consecutive suicides from three ethnic groups in East Taiwan, Cheng et al. (1997) found that between 47 and 77% of suicides suffered from ICD-10 personality disorder. In all groups, the most prevalent category among suicides was emotionally unstable personality disorder, occurring in 41% of suicides (OR: 9.9; 95% CI: 4.6–21.1). Dissocial personality disorder was identified in only 3.5% of suicides.

Dyck et al. (1988) examined the lifetime histories of attempted suicide and psychiatric disorders in a random sample of 3,258 household residents of Edmonton, Canada. By comparing the prevalence of psychiatric disorder in attempters and non-attempters, they were able to calculate the relative risk of attempted suicide for each disorder. Approximately 80% of those with a history of suicide attempts had a lifetime psychiatric disorder. The greatest relative risks were associated with schizophrenia (23.1) and mania (21.0). The relative risk for ASPD was 4.0.

Beautrais et al. (1996) adopted a case-control design in order to test the association between mental disorders and attempted suicide. Three hundred and two consecutive subjects who made serious suicide attempts were compared with 1,028 randomly selected controls. Each subject was interviewed with a modified version of the Structured Clinical Interview for DSM-III-R disorders. Of those who attempted suicide, 90.1% had a mental disorder at the time of the attempt. Correlations between different mental disorders were controlled for in the analysis with logistic regression, and this showed that the odds of a serious suicide attempt were significantly higher for individuals with a diagnosis of mood disorder (OR: 33.4; 95% CI: 21.9–51.2); substance use disorder (OR: 2.6; 95% CI: 1.6–4.3); or antisocial personality disorder (OR: 3.7; 95% CI: 2.1-6.5). However, an estimation of the population-attributable risk showed that only elimination of mood disorders would result in a substantial reduction in the frequency of attempted

Prospective research has also shown that a diagnosis of antisocial personality disorder is highly predictive of death from unnatural causes. In a 6- to 12-year follow-up of 500 psychiatric outpatients, Martin et al. (1985) found that the Standardised Mortality Ratio for patients with antisocial personality disorder was nearly four times that found in the reference population. In addition, retrospective data confirm that antisocial personality disorder is a risk factor for both sudden violent death (Rydelius 1988) and accidental injury (McDonald and Davey 1996).

Associated conditions

Axis I conditions

One of the most important reasons for diagnosing personality disorders is that they are linked to higher rates

of mental disorder. They also significantly affect the course and outcome of mental illness (Reich and Vasile 1993). However, the assessment of the relationship between personality disorders and mental illness is complicated by the shortcomings of the current categorical classification system, the poor specificity of some of the personality assessment instruments used, and a number of confounding factors, the most important of which are state effects. In addition, studies of axis I–II associations often neglect to account for the presence of multiply cooccurring axis I disorders, and therefore are at risk of finding spurious associations (Coid 1996).

By far the most heavily studied associated conditions have been substance use disorders. Research at all epidemiological levels confirms a strong association between substance abuse and antisocial personality disorder, and highlights the implications in terms of poorer treatment outcome (Woody et al. 1985; Rounsaville et al. 1991). The nature of the relationship is, however, complex, and questions have been raised about the validity of the distinction between substance use disorders and antisocial personality disorder. Robins (1998) has suggested three possible explanations for the association. First, that the symptoms of the disorders overlap; for example, the criteria for a DSM-IV diagnosis of substance use disorder include 'recurrent substance-related legal problems.' Clearly, such diagnostic overlap may artificially increase rates of antisocial personality disorder among substance abusers. Second, that the relationship is accounted for by shared risk factors, although genetic studies show that at least for alcoholism and antisocial personality disorder, the aetiological factors are different (Cadoret et al. 1995). Finally, Robins suggested that there may be a causal connection between the two disorders, although the absence of incidence and prospective data on these disorders makes this hard to establish.

At a community level, the ECA study (Regier et al. 1990), found that antisocial personality disorder was the most commonly co-occurring disorder among those with a drug disorder. In keeping with this, the National Comorbidity Survey (Kessler et al. 1996) found that almost 80% of all respondents with ASPD also had a lifetime addiction. Studies of selected substance misusers show that opiate dependency has the highest level of cooccurrence with ASPD (Gill et al. 1992; Brooner et al. 1993; Darke et al. 1994; Kokkevi and Stefanis 1995; Brooner et al. 1997). The prevalence rate varies considerably between studies (25–70%) and is dependent on which diagnostic criteria and instrumentation were used. Consistent with the impulsive nature of individuals with antisocial personality disorder, is the finding that the disorder is associated with HIV risk-taking behaviour (Dinwiddie et al. 1992; Gill et al. 1992; Brooner et al. 1993). High prevalence rates of ASPD have also been found in samples of cocaine addicts (Weiss et al. 1993; Kranzler et al. 1994; Barber et al. 1996), alcoholics (Ross 1995; Tomasson and Vaglum 1995) and solvent abusers (Dinwiddie et al. 1990).

The National Comorbidity Survey (Kessler et al. 1994) showed that at least in the USA, psychiatric morbidity is unevenly distributed, with the majority of lifetime disorders occurring in one-sixth of the population with a lifetime history of three or more disorders. Antisocial personality disorder behaves like other conditions in this respect, and has been shown to covary with most axis I conditions, although the degree of covariation is variable and less than that found with substance use disorders. Significant covariation with ASPD has been reported in samples of subjects with the following disorders: depression (Sanderson et al. 1992; Pepper et al. 1995), phobic disorders (Renneberg et al. 1992), panic and anxiety disorders (Hoffart et al. 1994), eating disorders (Braun et al. 1994), schizophrenia (Bland et al. 1987), obsessive compulsive disorder (Kolada et al. 1994) and pathological gambling (Crockford and el-Guebaly 1998).

Axis II conditions

Research in psychiatric settings suggests that many individuals fulfil criteria for more than one of the personality disorder categories (Morey 1988; Widiger et al. 1991), and at least 50% of personality-disordered subjects can be expected to have two or more co-existing personality disorders (Pfohl et al. 1986; Loranger et al. 1987; Oldham 1992). Specifically, antisocial personality disorder has been shown to consistently demonstrate a high level of covariation with histrionic, narcissistic, impulsive and borderline personality disorders – the so-called 'Cluster B' personality disorders (Zimmerman and Coryell 1989; Widiger et al. 1991; Moldin et al. 1994; Coid 1996).

Risk factors

Genetic studies

Antisocial personality disorder has been shown to demonstrate familial aggregation (Cloninger et al. 1978), although stronger evidence for the heritability of the disorder comes from twin and adoption studies. Until comparatively recently, the majority of twin studies had only examined criminality (Christiansen 1974; Dalgaard and Kringlen 1976). McGuffin and Gottesman (1984) pooled the results of seven studies of adult criminality and five studies of juvenile delinquency and concluded that whilst there was a definite genetic contribution to adult criminality (monozygotic:dizygotic concordance rates, 51%:22%), the genetic contribution to juvenile delinquency was more modest (MZ:DZ concordance rates, 87%:72%). Grove et al. (1990) found that in a study of 32 sets of monozygotic twins reared apart, both childhood and adult antisocial symptoms showed significant heritability (childhood symptoms, heritability: 0.4; adulthood symptoms, heritability: 0.28). In contrast, Thapar and McGuffin (1996) found that in a sample of 198 same-sex twin pairs, transmission of antisocial symptoms could be explained entirely by shared environmental factors. Adoption studies focusing on antisocial behaviour have found substantial gene-environment interaction in the genesis of such behaviour (Cloninger et al. 1982; Sigvardsson et al. 1982; Mednick et al. 1984; Baker 1986; Moffitt 1987). Recent behaviour-genetic research, looking at the nature of this interaction, has shown that the behavioural correlates of antisocial personality are associated with high levels of assortative mating (Krueger et al. 1998). Clearly the transmission of antisocial behaviour and personality does not fit a 'simple model' (Carey 1994), and in line with other psychiatric epidemiological research, there is a growing awareness of the need to incorporate measures of both genes and environment into study designs (Mann 1997).

Childhood antecedents

Conduct disorder

The majority of adults with antisocial personality disorder have been conduct-disordered children (Robins 1966, 1991; Robins and Ratcliff 1980). However, recent research has shown that the diagnosis of antisocial personality disorder may not provide adequate coverage of the range of disabilities in adult life that are the sequelae of childhood conduct disorder (Zoccolillo et al. 1992). In addition, conduct disorder acts as a non-specific risk factor for a number of other adult psychiatric conditions, including borderline personality disorder (Soloff and Millward 1983), substance abuse (Darke et al. 1994) and generalised anxiety disorder (Storm-Mathiesen and Vaglum 1994). Additional risk factors must therefore be operating.

Childhood hyperactivity

Research that has attempted to link childhood hyperactivity with antisocial personality disorder has been hampered by several methodological problems. These include diagnostic heterogeneity at both child and adult levels, and the failure to account for the overlap between conduct disorder and hyperactivity (Lilienfeld and Waldman 1990). Prospective and retrospective research has shown that children diagnosed as hyperactive in childhood show high rates of antisocial behaviour in adolescence and are at high risk of receiving a diagnosis of antisocial personality disorder in adulthood (Morrison 1980; Gittelman et al. 1985; Weiss and Hechtman 1986; Mannuzza et al. 1998). Studies that have controlled for the confounding effects of conduct disorder have found that children with mixed symptoms of hyperactivity and conduct disorder have the worse outcome, in terms of antisocial behaviour (Farrington et al. 1990; Loeber et al. 1990). In addition, genetic studies have indicated that the morbid risk of antisocial personality disorder is higher in relatives of children with this mixed symptom profile compared to the relatives of children with pure conduct disorder (Lahey et al. 1987; Faraone et al. 1991). Finally, relative to conduct-disordered children, children with mixed disorders have been found to have an earlier onset of antisocial behaviour, and to exhibit more frequent and more severe antisocial behaviour across more settings (Walker et al. 1987; Loeber et al. 1990; Moffitt 1990).

Delinquency

The Cambridge Delinquency Survey demonstrated the continuity between childhood and adult antisocial behaviour (West 1969, 1982; West and Farrington 1973, 1977; Farrington 1995). Using regression analysis and a series of scales designed to measure 'antisocial personality', Farrington (1990) demonstrated that the most important independent predictors of future convictions fell into six categories: socio-economic deprivation, poor child-rearing, antisocial family members, school failure, impulsivity and antisocial child behaviour. On the basis of the results of the Cambridge Survey, Farrington (1986, 1992, 1993) has proposed a theory to explain the development of offending, which might be used to explain the development of antisocial behaviour.

Childhood temperament

A large literature has established that aggression is one of the most stable forms of behaviour over time, particularly in boys (Olweus 1979; Roff and Wirt 1984). In addition, aggression appears to be stable across generations (Huesmann et al. 1984). More recent research has confirmed that temperamental deviance in childhood is a risk factor for adult psychiatric disorder and specifically, antisocial personality disorder. As part of the Dunedin Multidisciplinary Health and Development Study, Caspi et al. (1996) classified a sample of 1,037 3-year-old children into one of five temperamental groups: 'undercontrolled' (irritable and impulsive), 'inhibited' (socially reticent) 'well-adjusted' (within normal limits for age) 'confident' (eager to explore) and 'reserved' (timid and uncomfortable in the testing situation). At 21 years, over 90% of the sample were interviewed with the Diagnostic Interview Schedule to obtain diagnoses of mental disorder. Overall, undercontrolled and inhibited children were the most likely to be diagnosed with psychiatric disorder. More specifically, undercontrolled children were 2.9 times more likely to be diagnosed with antisocial personality disorder (P < 0.05; 95% CI: 1.1– 8.1), 2.2 times more likely to be recidivistic offenders (P < 0.05; 95% CI: 1.1-4.7), and 4.5 times more likely

Table 5 Studies of the burden of ASPD^a

Study	Sample	Measures	Findings
Bland & Orn (1986)	1200 randomly selected adult household residents	DIS	2.6% admitted to abusing a child. 49% of those who were violent had ASPD, recurrent depression and/or alcoholism. ASPD + alcohol: 48 times more likely to commit family violence
Bland et al. (1988b)	3258 randomly selected adult household residents	DIS	Lifetime prevalence of all psychiatric disorder higher in unemployed group. Odds ratio of unemployment given ASPD: 6.2
Koegel et al. (1988)	379 homeless adults	DIS	Lifetime prevalence ASPD: 21% ASPD 4 times more likely in homeless population
Dinwiddie & Bucholz (1993)	1869 St Louis ECA sample	DIS	4% admitted to child abuse. Abusers more likely to receive diagnosis of ASPD, alcohol abuse or depression
Dinwiddie (1992)	a) 61 wife batterers b) 319 controls	HELPER	Prevalence of ASPD (Feighner criteria): a) 46% b) 31% Odds ratio: 1.9 (95% CI: 1.0–3.6)
Else et al. (1993)	21 wife batterers, 21 controls	MMPI	Batterers scored higher than controls on measures of antisocial and borderline PD
Hart et al. (1993)	85 wife assaulters	MCMI-II, PDE	MCMI-II: 90% had a PD PDE: 29% ASPD

^a From 'Antisocial personality disorder: an epidemiological perspective' (Moran 1999). Reproduced with permission from Gaskell publications

DIS Diagnostic Interview Schedule, PDE Personality Disorder

Examination, *HELPER* Home Environment & Lifetime Psychiatric Evaluation Record, *MMPI* Minnesota Multiphasic Personaltiy Inventory, *MCMI-II* Millon Clinical Multiaxial Inventory-II

to be convicted for a violent offence (P < 0.01; 95% CI: 1.8–10.9).

Childhood experiential antecedents

Patients with personality disorders frequently report traumatic childhood experiences. However, the normal process of child development is susceptible to interference by a multitude of adverse environmental factors, many of which act non-specifically in the pathogenesis of a variety of psychiatric disorders. In addition, much of the literature on specific associations between early adversity and axis II categories has been based on weak methodology characterised by unsystematic observation, overdependence on self-report and retrospective data, and infrequent use of control groups. The betterquality research has shown that, by comparison with controls, abused/neglected children are more likely to be arrested as juveniles and as adults (Widom 1989), and as adults, are more likely to meet criteria for ASPD (Luntz and Widom 1994).

Burden

The assessment of the burden associated with personality disorders is complicated by the fact that the disorders are defined in terms of their associated handicap. This leads to an inherent circularity in their definition, which is exemplified by antisocial personality disorder, as its definition specifically emphasises antisocial be-

haviour. A possible solution to this problem would be to adopt a definition of personality disorder that limits itself to impairment (i.e. personality traits) and to consider associated disability and handicap quite separately.

Studies of selected populations indicate that antisocial personality disorder is especially prevalent among the unemployed, homeless, wife-batterers, and child abusers and the findings of studies of these selected groups are summarised in Table 5. Studies that have examined the social disturbance associated with a wide range of disorders have found that the odds of being divorced, unemployed or committing a felony are higher for antisocial personality disorder than for any other axis I or axis II disorder (Thompson and Bland 1995). Unsurprisingly, antisocial personality disorder has also been found to be strongly associated with crime (Hodgins et al. 1996), and in particular, violent crime (Eronen et al. 1996).

Studies of health service utilisation patterns indicate that individuals with personality disorders are frequent users of health services (Saarento et al. 1997), although the literature on antisocial personality disorder is sparse in this respect. In a naturalistic follow-up study of 91 subjects, Perry et al. (1985) found that subjects with borderline and antisocial personality disorders and bipolar II affective disorders all used psychiatric care frequently, and that individuals with borderline personality disorder used the highest levels of care over time. Leaf et al. (1985) found that having a diagnosis of antisocial personality disorder increased the odds of having a mental health visit in the previous 6 months by a factor of almost six. ECA data on mental health service usage

by persons with mental disorder indicate that whilst persons whose primary complaint is antisocial personality disorder constitute only a small proportion of the total number of those presenting (3%), the 1-year visit rate of these individuals is amongst the highest, with a rate of 26.4 visits per person (Narrow et al. 1993).

Conclusions

Epidemiological research has shown that antisocial personality disorder is a common disorder, which is particularly prevalent among prisoners. It has major public health implications in terms of its association with drug abuse, early unnatural death, violent crime, unemployment, homelessness, and family violence. A number of environmental and genetic risk factors have been elucidated, and there is growing evidence for substantial interaction of these factors in the aetiology of the disorder. However, the diagnosis of antisocial personality disorder is problematic and has questionable validity. In particular, the criteria overlap greatly with those for criminality and substance abuse, leading to possible overdiagnosis of the disorder in prisoners and drug addicts. In addition, crucial longitudinal data, which would help validate the diagnosis, are lacking (Robins and Guze 1970).

The most important priority for future epidemiological research into antisocial personality disorder (and indeed all personality disorders) should therefore be the accurate determination of natural history. This would facilitate the delineation of risk factors and protective factors, and provide a more representative picture of axis I and axis II associations. Longitudinal data would also have the benefit of indicating preventative strategies. More importantly, such data would test the validity of the diagnosis, by discovering its power to predict the outcome of associated psychiatric and physical morbidity and patterns of health service use. Future longitudinal studies should attempt to correct for the deficiencies of previous research and use repeated follow-ups and psychosocial outcome variables in preference to conviction data. Finally, for those currently debating the medical response to this disorder, surely the question of diagnostic validity needs to be properly addressed before we can answer the issue of responsibility for treatment?

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