

tise. Investment needs to cover individuals and infrastructure as well as institutions. Doctors have benefited from decades of investment in research and development. Nurses and allied health professions are showing that they deserve to do so too.

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The impact of antisocial lifestyle on health

Family, school, and police interventions can reduce health risks

An antisocial lifestyle comprises a range of related behaviours that include violent and non-violent offending, substance misuse, truancy, reckless driving, and sexual promiscuity, some of which constitute self evident health risks.¹ Overall, onset peaks at 8-14 years, prevalence peaks at 15-19, and desistance peaks at 20-29 years of age. Early onset predicts a long antisocial career. Since antisocial behaviour and risk taking is more prevalent in men, explanations may be biological as well as social. Antisocial individuals tend to be versatile in their behaviours, although early adulthood is characterised by a switch from group offending to lone offending. Overall, diversification in antisocial behaviours is seen up to the age of about 20, followed by gradual specialisation in particular types of antisocial behaviours, such as illicit use of drugs.²

Independent precursors of an antisocial lifestyle include antisocial child behaviour, impulsivity, school failure, an antisocial family, poor parenting, and economic deprivation.² Turning points away from an antisocial lifestyle include getting a job, getting married, moving to a better area, and joining the army.³ Weak bonds to society and individuals, self centredness, low empathy, and lack of religious belief are all associated with substance misuse and an antisocial lifestyle.^{4,5}

The impact of an antisocial lifestyle on health is increasingly well understood. For example, early contact with the police, truancy, school misconduct, and divorce are significant predictors of premature death.⁶ Higher death rates among offenders have been attributed largely to concurrent alcohol and illicit use of drugs. Impulsivity, aggression, alienation, and a tendency to experience anger and irritability in response to daily life hassles characterise those taking single health risks: rejection of social norms, danger seeking, impulsivity, and little need or capacity for relationships with other people have been found to characterise those taking multiple health risks.⁷

Longitudinal research has found particular links between an antisocial lifestyle and injury, especially injury sustained in assaults at age 16-18 and on the roads or at work at age 27-32.^{1,8} Injuries due to assault have been found to predict future convictions.

Attempts to explain the observed association of criminal behaviour, involvement in crashes, and injuries have focused on control theory, which explains behaviour in terms of the way children are socialised, particularly through parental care and control.⁹

DATES syndrome, comprising drug abuse, injury sustained in assaults and accidental trauma, and elective surgery, has been attributed to an antisocial lifestyle.¹⁰ This range of disorders and treatment was significantly more frequent in young adults injured in assaults than in other ways.

Injury is related to elements of an antisocial lifestyle up to the age of 32 including heavy drinking, low job status, and convictions for motoring offences.⁸ Although antisocial men aged 16-18 seem to be less ill than their peers, links between psychiatric illness and convictions and between smoking and illness are established by age 32. A picture emerges of fit, well, but vulnerable risk takers from poor family backgrounds at 18 beginning to reap the consequences of unhealthy lifestyles by age 32. In turn, this fits with the concept that risk factors for adult disease accumulate differentially throughout life.

Given the roots of antisocial behaviour in childhood, families, and risk taking it is perhaps not surprising that prevention targeted at young families, in schools and through criminal justice efforts to deter have been shown to be effective across a range of behaviours.^{11,12} For example, preschool education and early family support have, in randomised trials, been shown to have positive health outcomes in terms of reduced child abuse, neglect and injury, drug misuse, and teenage pregnancy.^{11,12} The High/Scope Perry Preschool programme saved \$49 044 (£30 429; €44 603) in costs of crime alone for every \$12 356 spent on each child.¹² Home visiting and education of parents in day care settings, training in cognitive-behavioural child skills, and management training for parents have been shown to reduce a range of antisocial behaviours including offending and alcohol or other drug misuse. No programmes targeting community risk factors have yet been found to be effective.¹¹

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Effective police interventions include patrols targeted at known hotspots of violence and arrest of serious repeat offenders, drunk drivers, and employed suspects of domestic violence. In terms of rehabilitation programmes, intensive targeting of specific offender problems, prison based community treatment of offender drug misuse, cognitive behavioural therapy, and sex offender treatment outside prisons have all been found to be effective.^{11 13}

Nowhere are the impacts of antisocial lifestyle on health more apparent than in prisons. Although a captive population provides unique opportunities for treatment, problems related to prisoner health are often established and intransigent. The recent transfer of responsibility for prison health services in England

and Wales from the Home Office to the Department of Health, however, is logical, and a prompt both to acknowledge relationships between crime, injury, and illness and to develop integrated prevention and treatment. While links between deprivation and health have been widely studied, links between antisocial lifestyle and health have been neglected.

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Treatment of pulmonary hypertension

Several options exist, but they are expensive and necessitate specialist care

For understandable reasons, the pulmonary circulation remains an enigma to most doctors. This is because the cardinal symptom, dyspnoea, is shared with many more common diseases, and the signs of pulmonary hypertension are difficult to elicit for the non-specialist. Consequently, the delay between onset of symptoms and diagnosis is two years, and the mean survival from the time of diagnosis is only another two years in untreated patients with severe hypertension of the pulmonary artery. In the past, pulmonary hypertension was not treatable, but now several treatments are available.

Severe pulmonary hypertension, with a total prevalence of about 30-50/million, can be primary or associated with apparently disparate conditions including connective tissue disease, congenital heart disease, chronic pulmonary thromboembolism, HIV infection, use of an appetite suppressant, and liver disease. Surprisingly, nearly all these conditions have a similar histological picture of vascular remodelling. The pathobiology of pulmonary artery hypertension is now better understood. A gene for familial pulmonary arterial hypertension, which codes for BMPR2 a receptor in the transforming growth factor β (TGF- β) family, has been discovered.^{1 2} This gene is also found in up to 26% of patients with so called sporadic pulmonary hypertension. The genetic abnormality perhaps must be accompanied by some additional environmental factor to cause pulmonary artery hypertension ("the double hit hypothesis"), and the remodelling occurs because that factor (or factors) acts in concert with dis-

turbed BMPR signalling to cause an increase in production of cytokines and other factors.

In the past pulmonary artery hypertension was considered untreatable unless the underlying cause could be treated (for example, HIV infection), and patients could be offered only oxygen and transplantation. It was later realised that oral anticoagulants alone improved survival, which implies a thrombotic component to the cause or progression of the disease. Furthermore, in patients with an acute response to vasodilators, a high dose of calcium channel blockers resulted in a five year survival of more than 90%.³ Unfortunately only 10-15% of patients fall into this "responder" category, and therefore a need for other forms of treatment existed.

The breakthrough came in the early 1980s when it was shown that patients could be maintained on continuous intravenous epoprostenol, which improves both exercise capacity and haemodynamics.⁴ Subsequently it was also shown that long term treatment with intravenous epoprostenol improved survival.⁵ Unfortunately, because of its short half life, epoprostenol must be given continuously intravenously, with the attendant risks of air embolism and sepsis. This difficulty and the need for treatments working through alternative mechanisms prompted several trials. No placebo controlled trials were conducted before 1999, but since 2000 over 1000 patients worldwide have been enrolled in trials, all of which have examined similar end points and studied similar patients. Most of the trials studied patients in WHO functional classes 2-4 and lasted