‘Screen and intervene’: governing risky brains

NIKOLAS ROSE

ABSTRACT

This article argues that a new diagram is emerging in the criminal justice system as it encounters developments in the neurosciences. This does not take the form that concerns many ‘neuroethicists’ – it does not entail a challenge to doctrines of free will and the notion of the autonomous legal subject – but is developing around the themes of susceptibility, risk, pre-emption and precaution. I term this diagram ‘screen and intervene’ and in this article I attempt to trace out this new configuration and consider some of the consequences.

Key words criminal justice, law, neuroscience, risk, screening

In March 2008 Gary Pugh, the director of forensic science and the DNA spokesman for the UK’s Association of Chief Police Officers, attracted much publicity when he called for a debate on the measures required to identify future offenders. He suggested that badly behaved primary school children – aged between 5 and 11 years – might have their DNA ‘fingerprints’ stored in the UK’s National DNA Database. He said: ‘If we have a primary means of identifying people before they offend, then in the long term the benefits of targeting younger people are extremely large. . . . We have to find who are possibly going to be the biggest threat to society.’ Of course, the DNA profile that would be stored on the database would be non-coding – just for identification purposes – but the sample itself is retained, and, although Pugh
did not raise this possibility, it is entirely possible that, at some point, this could have been analysed for anomalies in coding regions related to brain functions; say, for neurotransmitters or receptors, that might be linked to the child’s behavioural problems. Pugh was not a lone voice arguing for early identification – many claim that those who will become prolific criminals start offending young, and some research claims that by the time they reach 28, those who have had childhood conduct problems cost society up to 10 times more than those without (Romeo, Knapp and Scott, 2006; Scott, Knapp, Henderson et al., 2001) and that adults with ‘personality disorder’ are exceptionally costly to society (National Institute for Mental Health in England, 2003). While those who think this way differ in their accounts of causation, and their suggestions as to the measures that are required, all seem agreed that early identification and intervention is the way to go (Harrington and Bailey, 2003; Kim-Cohen, Moffitt, Taylor et al., 2005; Margo and Stevens, 2008).

These arguments bring together two closely related senses of risk. The first, is the desire to identify risky individuals – that is to say, those who will present a future risk to others – before the actual harm is committed. The second is the hope that one might be able to identify individuals at risk – those whose particular combination of biology and life history makes them susceptible to some future condition – here personality disorder, impulsivity, aggressivity or whatever, but more generally susceptibility for any psychiatric disorder. They bring these two senses of risk – risk to others and risk to the self – together in a socio-political and cultural context with a number of salient features. First, in domains from education to psychiatry we have seen the rise of the belief that advances in neurobiology are central to understanding individual conduct, both normal and pathological. Second, while there are many non-biological screening techniques in use, a whole range of new technologies, notably those from behavioural genetics and brain scanning, claim that they can identify the precursors, signs or markers of future riskiness in advance, presymptomatically or asymptptomatically – that they can identify a ‘susceptible individual’. Third, we inhabit a culture of precaution, prevention and pre-emption, where the logic of many practices for the conduct of conduct, and the obligation of those who must govern conduct, is to act early, to seek to prevent future undesirable events materializing, even if one is acting only on the possibility that they might occur (Ericson and Doyle, 2003; Ewald, 2001; Sunstein, 2005). Whether it is early intervention for children at risk of developing psychiatric disorders, early intervention for persons convicted of minor offences who may go on to more serious crimes, or early diagnosis and intervention in cases of Mild Cognitive Impairment that may lead on to Alzheimer’s, many of those working in these areas share the view that, as the introduction to a recent collection of research papers put it, ‘earlier is almost always better’ (Lebowitz, 2004: 349). And while predictions are always probabilistic, decisions and actions are determinate, and tend
to assume the worst outcome and act to mitigate or prevent it – this is the obligation of risk management (Power, 2004).

This is the context – the conjuncture – that I want to reflect upon today.

**NEUROLAW – NEUROETHICAL RUMINATIONS**

Over the last two decades there has been a revived interest in the biology of criminality. But this does not search for the biological roots of crime in general, as did the earlier biological criminology (Rafter, 1997). It focuses specifically on the biological basis of particular types of undesirable behaviour – those that involve impulsive or aggressive conduct and an apparent weakening of the conventional workings of guilt and remorse (Denno, 1990; Cauffman, Steinberg and Piquero, 2005; Fishbein and Henry, 2002). We can identify a number of research pathways here, all of which converge on the brain. There are attempts to identify neurochemical anomalies that correlate with low impulse control or aggression (Brunner, 1996; Crusio, 1996; Miczek, de Almeida, Kravitz *et al.*, 2007). There is the use of brain-scanning techniques to see if there are characteristic patterns of brain activity in those who commit violent or aggressive acts, or those who lack remorse or a sense of guilt (Buffkin and Luttrel, 2005; Krämer, Jansma, Tempelmann *et al.*, 2007; Raine, Buchsbaum and LaCasse, 1997; Raine, Buchsbaum, Stanley *et al.*, 1994). There is research on localization of functions, seeking to identify brain regions or neural circuits activated in aggression or impulsivity (Chen, Porjesz, Rangaswamy *et al.*, 2007; Hollander and Evers, 2001). There is genetic research, which today does not seek ‘genes for crime’ but tries to identify the specific variations at the level of single nucleotides, or SNPs, that may be linked to low impulse control in specific biographical and environmental contexts (Brunner, 1996; Brunner, Nelen, Breakefield *et al.*, 1993; Caspi, McCly, Moffitt *et al.*, 2002; Caspi and Moffitt, 2006; Wasserman and Wachbroit, 2001). And, in a more directly forensic context, there is research that tries to use brain-scanning techniques to identify deception, for lie detection purposes in the interrogation of suspects or witnesses (Haynes and Rees, 2006; Kozel, Padgett and George, 2004; Kozel, Revel, Lorberbaum *et al.*, 2004; Langleben, Schroeder, Maldjian *et al.*, 2002).

These developments have generated much speculation: predictions of the imminent convergence of behavioural genetics, neuroscience and law (Garland and Frankel, 2006); florid claims-making on the part of some neurobiologists (‘brain overclaim syndrome’) (Kirchmeier, 2004; Mobbs, Lau, Jones *et al.*, 2007; Redding, 2006; Zeki and Goodenough, 2004); and much worrying on the part of neuroethicists (Glannon, 2007; Illes, 2005; Levy, 2007; Marcus, 2002; Parens, Chapman and Press, 2006; Wolpe, Foster and Langleben, 2005). Will these developments do away with the idea of free will, upon which our
legal systems depend? Will defence lawyers claim that the responsibility of their client for a crime was mitigated – that it was the client’s genes, or brain, that made him or her commit the act? Should we think of crime as a disease, and with what consequences? Would the use of ‘brain-scanning’ in the legal system violate ‘neural privacy’? And much more in the same vein. Is there any evidence for such speculations? Let us begin with the courtroom, with criminal trials.

First, how about the ‘genetic defence’ – parodied as ‘my genes made me do it’? The case that has been most widely discussed is that of Stephen Mobley, accused of murdering the manager of a Domino’s pizza store in 1991. His lawyers sought to introduce genetic evidence – not to support a defence of not guilty but in mitigation of sentence. This was based on a family history which was claimed to show several generations of violence, aggression and behaviour disorder in uncles, aunts and grandparents. The lawyers argued that this was relevant because of Han Brunner’s study which showed a link between a particular syndrome – borderline mental retardation accompanied by violence and aggression – and a point mutation in a gene regulating the production of an enzyme – monoamine oxidase A – linked to changes in levels of various neurotransmitters (Brunner, Nelen, Breakefield et al., 1993). The Brunner study has become something of an exemplar to all future attempts to find a genetic basis and a neurochemical mechanism for impulsive or violent crime, seeming to show a clear causal pathway between a base substitution, the alteration in the structure and function of an enzyme, a determinate effect on a neurotransmitter linked to conduct and hence to that pathological conduct itself. But the court in the Mobley case refused to admit this evidence. The grounds were similar to those used in earlier ‘biological’ defences – for example, the XYY cases in the 1970s (Saulitis, 1979), and the pre-menstrual syndrome cases in the 1980s (Allen, 1984). What the courts demand is a reasonably certain causal connection between the biological or genetic condition in question and the specific act of criminal conduct. The Brunner study was more complicated than popular reports implied. And Brunner himself explicitly rejected this interpretation of his research, arguing that

the notion of an ‘aggression gene’ does not make sense, because it belies the fact that behaviour should and does arise at the highest level of cortical organization, where individual genes are only distantly reflected in the anatomical structure, as well as in the various neurophysiological and biochemical functions of the brain . . . although a multitude of genes must be involved in shaping brain function, none of these genes by itself encodes behaviour. (Brunner, 1996: 16)

Nonetheless his study has led to a host of follow-up research, some of which I will discuss presently. But the point remains stubbornly true: there is a
considerable distance between the probabilistic world of genetic research and the deterministic thinking of the courts.

Although genetic arguments have been used by the defence in other cases, in order to mitigate responsibility – such as the Kip Kinkel and Cary Stayner cases which I discuss below – I know of no case where a genetic defence has succeeded. Thus in Stayner’s, the defence called Dr Allison McInnes, assistant professor of psychiatry and human genetics at the Mount Sinai School of Medicine in New York, who showed the jury a genealogical table of Stayner’s family history, with putative sufferers from mental disorders marked in different colors. Fresno Bee reporter Cyndee Fontana described the testimony thus:

The story of Cary Stayner’s family tree rose in bursts of bright color from a white horizontal chart. . . . Yellow for psychosis. Green for obsessive-compulsive disorder. Red for substance abuse. Purple for pedophilia. Even more colors for more mental diseases ranging through four generations down to Stayner himself – the fruit of a family gene pool marked by psychiatric disorders. ‘So many different mental disorders’, said Dr McInnes as she led a jury through the branches of men and women, the flashes of color, that yielded the man convicted last week of murdering three tourists in February 1999.

The jury was not convinced. 6

Second, what of evidence from brain scans? Some – for instance the US Law and Neuroscience Project – suggest this will have a major impact in the courts. 7 Scans have been used in personal injury cases in the courtroom where issues of brain injury are involved. Positron Emission Tomography (PET) and Magnetic Resonance Imaging (MRI) evidence of brain tumours has, on at least one occasion, been used successfully to support a claim of insanity. In the famous case of John Hinckley, who was found ‘not guilty by reason of insanity’ for his attempt to assassinate Ronald Reagan the defence claimed that computerized axial tomography (CAT) scans of the brain provided organic evidence that Hinckley was schizophrenic. 8 Hinckley’s acquittal on the grounds of ‘not guilty by reason of insanity’ (NGRI) gave added impetus to the US campaign to reform the insanity and diminished capacity defences, which were severely limited in some 39 states, transformed into ‘guilty but mentally ill’ in 8 others (a verdict that allows any sentence up to and including death), and abolished entirely in Illinois and Idaho (Moran, 1991). 9 Nonetheless, evidence from the new technologies for visualizing the brain found its way into the American courts in the 1980s (Anderson, 1992). 10 But, as far as I am aware, later attempts by defence lawyers in the USA to use scans to demonstrate ‘functional’ abnormalities – that is to say, where there is no lesion or injury – have not passed the test of reliability required for novel scientific evidence to be accepted by the courts in criminal cases.
Thus in the case of Kip Kinkel in Oregon, who murdered both his parents and then returned to his school and shot a number of schoolchildren, the defence called a pediatric neurologist who showed images from a single photon emission computed tomography (SPECT) scan apparently showing areas of decreased blood flow in the prefrontal cortex, the temporal occipital and parietal lobes, and suggested that this was consistent with research showing a correlation between deficiencies in grey matter in these regions and the onset of childhood schizophrenia. The judge, however, referred to a 1996 change to the Oregon State constitution which shifted the focus of criminal punishment from ‘the principle of reformation’ to ‘the protection of society’, and argued that the protection of society should be given most importance in his ruling in this case. He sentenced Kinkel to 25 years for the murders, and a further 40 months for each attempted murder, making a total of more than 111 years in prison without parole. The subsequent appeal failed, the judge stating that the ‘protection of society’ consideration held greater weight than any other sentencing guidelines. And, in the 2002 trial of Cary Stayner for the horrific sexual assault and murder of three tourists in Yosemite, defence lawyers supported their plea of ‘not guilty by reason of insanity’ with evidence from a host of psychiatrists. Experts sharply disagreed over the significance of Stayner’s brain scans, Dr Joseph Wu, for the defence, seeing abnormalities in the images that could account for the defendant’s violent tendencies, while Dr Alan Waxman, called by the prosecution, saw nothing of the sort: on 26 August 2005, the jury took less than 5 hours to find Stayner guilty of three counts of first-degree murder.

If such arguments from functional brain imaging were to gather strength, however, what would be the implications? Not, I suspect, to eliminate the legal fiction of freedom of will. When the judiciary defend the non-genetic, non-psychiatric fictions of free will, autonomy of choice and personal responsibility, this is not because legal reasoning considers this to be a scientific account of the determinants of human conduct. Rather, the criminal justice system deems it necessary to act on the basis of this image of the human being for reasons to do with prevailing notions of moral and political order. Indeed, the trend of contemporary legal thought, especially in the USA, is to operate on the premise of the inescapability of moral responsibility and culpability. On this basis, no appeal to biology, biography or society should be allowed to weaken moral responsibility for the act, let alone to diminish the requirement that the offender be liable to control and/or punishment. In this context, the argument from biology is likely to have its most significant impact, not in diminishing the emphasis on free will necessary to a finding of guilt, but in the determination of the sentence. This is unlikely to be in the direction of mitigation. For if antisocial conduct is indelibly inscribed in the body of the offender, reform appears more difficult, and mitigation of punishment inappropriate. More likely are arguments for the long-term pacification
of the biologically irredeemable individual in the name of public protection. This view seems to be gaining hold, even if it means the rejection of many ‘rule of law’ considerations, such as those concerning the proportionality of crime and punishment.

But there is, however, another area where I think that neurobiology will have a more significant impact. Would it be possible to identify individuals whose propensity to such conduct arises from genetic or neurochemical anomalies or is, in some other way, inscribed in their biology? Perhaps to identify them before any offending had taken place? For those who follow this line of reasoning, early diagnosis coupled with preventive intervention may enable individuals so afflicted to be diverted from their path to criminality (Farrington and Coid, 2003). This returns us to the question of risk with which I began. Before turning to neurobiology, let us consider the way in which the problem of risk is posed at the points where the mental health system and legal systems intersect.

RISK-THINKING IN MENTAL HEALTH

Over the last 20 years, mental health legislation and procedures in almost all western countries have become pervaded by risk-thinking (Alberg, Hatfield and Huxley, 1996; Coid, 1996; Crichton, 1995; Moon, 2000; Rose, 1998a; Rose, 1996; Steadman, 2000). The question of risk to others was a central concern in recent reform of the UK mental health legislation. The Foreword to the 2002 Consultation Document on this bill recognized that only ‘a very few’ people pose a risk to others ‘because of their illness’. But, nonetheless, the focus of the proposed legislation was on risk assessment, risk management, risk communication between professionals and agencies, psychiatric treatment of mentally disordered offenders to reduce the risk of reoffending, and, of course, the use of the mental hospital as a site for the detention of those thought to pose a risk to ‘the public’ irrespective of whether they are ‘treatable’ and for as long as this risk of serious harm to others is judged to persist.

It is well known that there are many technical problems in predicting a rare event, especially when the base rate of such events within the population is extremely low – as it is with violent acts committed by those of us who have received inpatient treatment for mental health problems. The historian of statistics Gerd Gigerenzer, in his Reckoning with Risk, quotes the estimate made by John Monahan for the US Supreme Court in 1980: the ‘best estimate is that two out of three predictions of long-term future violence made by psychiatrists are wrong’ (Gigerenzer, 2002). Monahan was drawing on his own research at the start of the 1980s which showed that in only 1 out of 3 cases in which psychiatrists predicted violence did violence occur, despite the fact that the subjects were institutionalized populations with a history of
violence and a mental illness diagnosis. Monahan estimated that the reverse error – to wrongly predict there will be no violent act – is much less frequent, and seems to occur in about 1 out of 10 cases. His influential 1993 paper with Steadman and others, entitled ‘From Dangerousness to Risk Assessment’, argued for a shift away from a binary and fixed distinction – dangerous/not dangerous – in favour of a continuous, day-to-day risk assessment involving locating the potentially risky person at the appropriate point on a continuum. As his colleagues put it:

If an actuarially valid array of risk markers for violence could be reliably identified, clinicians could be trained to incorporate these factors into their routine practice, and the accuracy of clinical predictions of violence among the mentally disordered would be commensurately increased. (Steadman, Monahan, Robbins et al., 1993: 13)

In their early work, Monahan and Steadman were concerned to reduce the threat to civil liberties resulting from the overdiagnosis of psychiatric patients as dangerous. It is ironic, then, that the current political demand for risk assessment arises not from the fear of too much detention, but from the fear of too little.

The whole point of the shift from dangerousness to risk was the recognition that behaviour is a product of multiple dynamic factors in a complex situation. Robert Castel, in an insightful paper entitled ‘From Dangerousness to Risk’ published a couple of years before Monahan and Steadman’s argument, identified this trend very well – a move away from seeing dangerousness as an inherent part of an individual’s makeup towards a calculation of risk based on multiple factors (Castel, 1991). Within such a risk algorithm, the mental health status of the specific individual dissolves into this complex of factors – housing, employment, marital status, substance misuse and the like. The point about such factors is that they are not inscribed within the person. They are not diagnoses based on symptoms of illness. And they vary across time and space. This conception of the situational genesis of violence may be accurate. But to actualize it in practice would require a quite impractical continuous monitoring of the everyday life of the ‘community mental patient’. So the reality is inescapable: the risk estimate is attached, not to the situation, but to the individual.

How can we account for the prevalence of risk-thinking in psychiatry? Some researchers claim to find clear evidence of a strong relation between severe mental illness, notably schizophrenia, and violent offending (Brennan, Mednick and Hodgins, 2000; Raine, Brennan and Farrington, 1997). But the number of homicides committed by those with a mental health diagnosis is stable, and the proportion of all murders is falling. Each year in the UK, there are about 3,000 deaths from transport accidents, 2,000 from falls in the home, perhaps as many as 4,500 from suicide, around 700 from homicide, of which
around 70 – or 10 per cent – have been committed by people with a mental illness diagnosis – a figure which has stayed more or less constant over the last 20 years (Taylor and Gunn, 1999). So what is the threat that generates this fear of such violent or predatory monsters, and this demand for risk management, out of all proportion to their actual contribution to violence or harm? I suspect this is something to do with that apparently harmless category of ‘the public’. In our ‘advanced liberal’ societies, there is a fundamental division between ‘we, the public’ who can, in our imagination, conduct ourselves responsibly according to the norms of civility, and those others that threaten us (Rose, 2000b). On the one hand there are fantasies of security, imagined communities where normal individuals and families can live an untroubled life of freedom. And, as its inescapable reciprocal, there is a constant fear of predatory monsters. A host of measures respond to this perception – gated communities, closed circuit television cameras, the use of architectural devices – designed to ward off those who are thought to threaten this security. The demand for risk management of those who have a psychiatric diagnosis is one more way of seeking to manage the insecurities that the fantasy of security itself generates and intensifies. Risk assessment, or the demand for it, has a significance which is more symbolic than instrumental – it answers not to the reality of dangers but to the politics of insecurity.

Mary Douglas and Aaron Wildavsky famously argued that ‘each form of social life has its own typical risk portfolio’ (Douglas and Wildavsky, 1982: 6). A risk portfolio is a way of selecting, out of all possible, real or imagined threats and harms, those that shall be the focus of individual or collective attention. This selection is, inescapably, done in relation to moral evaluations shaped by cultural norms. Risk, in relation to mental disorder, is certainly high in the risk portfolio for many – the perception that mental illness carries the risk of violence towards others, and alternatively that violence is, at root, a matter of mental pathology. This perception, rather than any increase in actual harms, underlies the promulgation of laws and other measures to focus risk assessment and risk management on psychiatric patients. But what places the risks of those in this heterogeneous category so high in the risk portfolio of public, media and politicians?

Perhaps this can be understood, in part, by the ways in which risk-thinking, in psychiatry, brings together two rather different senses of risk. In the first, as we have seen, there is a continuum of risk. In principle we can all be placed on this continuum, for – given certain circumstances – each of us might commit violent acts, and those who are young, are male and consume alcohol might find ourselves rather high on such a scale. However, the arbitrary categories of persons placed high on our contemporary ‘risk portfolio’ emerge from another, older sense. This is not a continuum but a binary opposition between the normal and the abnormal. In this opposition, some people are fundamentally different. They are ‘monstrous individuals’. A monstrous
individual is an anomaly, an exception. This is not merely one who diverges from a norm, but one who is of a radically different nature, implacably pathological, evil. These are the ‘predators’ of popular imagination – sex offenders, paedophiles, serial killers and, as the newspapers would put it, deranged mental patients freed to kill again. And, of course, these are the people who are dangerous because of their ‘personality disorders’ who caused so much concern in the UK in the early years of this century, because it seemed that they escaped both mental health and criminal legislation: hence a change in the law to enable them to be detained so long as they posed ‘a grave risk to the public’.

I call this ‘governing through madness’. By this I mean the ways in which contemporary politics of mental health has come to be shaped in response to a more general demand for a politics of community protection and public defence: anxiety about the risks posed by those with mental health problems thus becomes one element in the justification of a more general shift in the logics of regulation. Psychiatry itself has been reoriented within these strategies of control formulated in terms of risk. To satisfy the public and political demand for the identification of the potentially monstrous, psychiatric risk-thinking has come to connect the routine management of those with a history of psychiatric troubles and the problem of the identification of the exception along a single dimension of risk assessment. Risk assessment in the name of the prevention of relapse has become entwined with strategies for pre-emptive intervention in the name of community safety; with the dream – or nightmare – that it is possible to identify and exclude those who are incorrigibly risky and potentially monstrous – incarceration without reform. Historical precedents would suggest that such strategies are unlikely to reduce the overall frequency of the very rare incidents they seek to prevent. But they are likely to result in threshold-lowering and net-widening, and the detention of many individuals who are capable of leading lives that might sometimes be troublingly different but would pose no dangers to others (Cohen, 1985).

DETAINING THE DANGEROUS

The shift from dangerousness to risk has not displaced an earlier attempt – to read danger in the makeup of the individuals themselves. Consider two recent developments in the UK. The first goes under the name of ‘Dangerous and Severe Personality Disorder’ or DSPD. The second goes under the name of the Indeterminate Public Protection Sentence. Let me begin with DSPD. The concern that led to the formulation of this category arose from a peculiar paradox in mental health legislation – under the Acts passed since the 1950s, an individual could only be detained against his or her will in a psychiatric
hospital if their condition was treatable. But there were some characters, prone to commit unpleasant acts ranging from urinating in the street to violence, whose disorders were not considered to be psychiatric illnesses – although they were diagnosed by psychiatrists – but disorders of personality: not states of illness but traits of the person. Traits, for psychiatrists, were enduring features of personality that might be managed but they could not be ‘treated’. So those individuals, often termed psychopaths, could not be detained under the mental health legislation. But nor could they be detained under the criminal law except by means of a specific sentence after a finding of guilt for a particular act that they had already committed. In the classical retrospective gaze of the criminal law, individuals could not be detained simply because authorities or experts thought it likely that they would commit certain acts in the future. Hence, the problem for those who would govern risk, for their aim is indeed to govern the future.

Of course, to some extent concern with such persons is coextensive with psychiatry itself. In 1835 Pritchard identified a condition that he termed ‘moral insanity’:

... a morbid perversion of the natural feelings, affections, inclinations, temper, habits, moral dispositions, and natural impulses without any remarkable disorder or defect of the intellect or knowing or reasoning faculties and in particular without any insane delusion or hallucination. ... The moral and active principles of the mind are strongly perverted or depraved; the power of self government is lost or greatly impaired, and the individual is found to be incapable not of talking or reasoning upon any subject proposed to him, but of conducting himself with decency and propriety in the business of life. (Pritchard, 1835: 6)

Forty years later, Maudsley wrote: ‘[It is] a form of mental alienation which has so much the look of vice or crime that many people regard it as an un-founded medical invention’ (Maudsley, 1874). And it was this condition that became the psychopath: the Oxford English Dictionary tells us that the Pall Mall Gazette first used this term in 1885: ‘We give M. Balinsky’s explanation of the new malady. “The psychopath ... is a type which has only recently come under the notice of medical science. ... Beside his own person and his own interests, nothing is sacred to the psychopath.”’ In the UK’s Mental Deficiency Act 1914, these people were referred to as ‘moral defective’ (UK MDA 1914) and in the legislation of 1927, as ‘moral imbeciles’ (UK MDA 1927). The UK’s Mental Health Act 1959 introduced the term ‘Psychopathic Disorder’ and over the 1970s, psychiatrists began to explore the world of ‘Personality Disorder’ and ‘Anti-Social Personality Disorder’ (McNeil, 1970). This is not the place to go into this history in any depth, just to locate the most recent expression of this problematization: termed in the UK ‘Dangerous and Severe Personality Disorder’.
This term was introduced in 1999 in a report entitled ‘Managing Dangerous People with Severe Personality Disorder’ although it was neither a psychiatric diagnosis nor a legal category (Home Office and Department of Health, 1999). It seems to have been understood as an extension of the diagnosis of antisocial personality disorder (ASPD) – which appears to be most closely associated with the notion of psychopathic disorder, the legal term used in UK mental health legislation to refer to people who have ‘a persistent disorder or disability of mind . . . which results in abnormally aggressive or seriously irresponsible conduct’. The Diagnostic and Statistical Manual of Mental Disorders (DSM) definition of this condition is

... a pervasive pattern of disregard for and violation of the rights of others occurring since age 15, as indicated by three (or more) of the following: failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing acts that are grounds for arrest; deceitfulness, as indicated by repeated lying, use of aliases, or conning others for personal profit or pleasure; impulsivity or failure to plan ahead; irritability and aggressiveness, as indicated by repeated physical fights or assaults; reckless disregard for safety of self or others; consistent irresponsibility, as indicated by repeated failure to sustain steady work or honor financial obligations; lack of remorse, as indicated by being indifferent to or rationalizing having hurt, mistreated, or stolen from another. (American Psychiatric Association, 2000: 649–50)

The initial move in the UK was to assert that ASPD should ‘no longer be a diagnosis of exclusion’ – that is to say, that those with this diagnosis should and could be treated, although not all would associate ‘inclusion’ with the extension of the powers of the Mental Health Act to compulsorily detain and treat persons so diagnosed. But pretty soon, the strategy for ASPD merged with that for DSPD. Over £100 million has been spent on setting up a DSPD programme which declares itself dedicated to ‘Ensuring the public is protected from some of the most dangerous people in society’. In other words, including those with DSPD by excluding them – by confining them in secure facilities in the name of public protection.

At about the same time as the DSPD programme was being established, the UK government instituted some other ‘public protections measures’ in the Criminal Justice Act 2003 including something known as an ‘indeterminate sentence for public protection’. This enables judges, not only to set a minimum tariff for a prison sentence, but to require the defendant to satisfy the authorities that he or she is fit for release at the end of the allotted stretch, and does not pose any threat to the community. Individuals considered to be a continuing threat can be detained for indefinite periods, on the grounds of an expert psychiatric judgement of their dangerousness, even if they have been
convicted of a minor offence such as setting a fire. An Indeterminate Public Protection Order is in effect a life sentence for individuals judged to be risky, or from whom the public is deemed in need of protection: as with a life sentence, even if released, the individual is on a lifetime licence and can be recalled to prison at any time. Initially designed as a measure to detain a small number of exceptionally dangerous individuals, by 2007 almost 3,000 people were being detained in prison under these provisions, with the number expected to rise to over 12,000 by 2012.22

I think I have said enough to indicate that, in the UK at least, the hunt is on for measures to identify those who are at risk of risk. The same is true in the United States, as we will see presently. It is in this context that we can locate current concerns about the neurobiology of antisocial conduct. Let me turn directly to this issue.

**RISK PREDICTION IN A BIOLOGICAL AGE**

As in the UK, in the United States in the 1990s, there was a belief that there was something of an epidemic of crimes of aggression, impulsivity, lack of self-control. Whereas in the past, some had argued that criminality itself was a disease, now the argument was slightly different: violence was a public health problem. In the early 1990s, the US National Institutes of Mental Health launched the National Violence Initiative. Psychiatrists would seek to identify children likely to develop criminal behaviour and would employ intervention strategies with them. The official report from this initiative issued in 1993 and 1994 in four volumes called for more research on biological and genetic factors in violent crime (Reiss and Roth, 1993; Reiss, Roth and Miczek, 1994). It also called for research into new pharmaceuticals that might reduce violent behaviour. By 1992, the US federal government, in partnership with the Macarthur Foundation, was sponsoring a large-scale initiative entitled the ‘Program on Human Development and Criminal Behavior’ to the tune of some $12 million per year.23 The project aimed at screening children for biological, psychological and social factors that may play a role in criminal behavior, and proposed to follow subjects over an 8-year period, with a view to ultimately identifying biological and biochemical markers for predicting criminality. While this umbrella program was withdrawn as a result of controversy surrounding the violence initiative (Wasserman, 1995), resulting in part from some injudicious remarks by the head of the programme, Dr Frederick Goodwin, individual projects continued to be sponsored by the federal government.

While critics saw this as a dangerous programme of social control, proponents saw it somewhat differently. Diane Fishbein of the US Department of Justice argued that
Once prevalence rates are known for genetically influenced forms of psychopathology in relevant populations, we can better determine how substantially a prevention strategy that incorporates genetic findings may influence the problem of antisocial conduct... [At a minimum the evidence] suggests the need for early identification and intervention. (Fishbein, 1996: 91–4)

Indeed, as Daniel Wasserman has pointed out, biological criminologists did not believe that their work would discover ‘causes of crime’ but hoped that it might identify markers and genes associated with that behaviour (Wasserman, 1996).

I have already mentioned the Han Brunner study, linking aggressive behaviour and lack of impulse control to variations in the gene sequence regulating the monoamine oxidase A. This study, and some others by the same team, has been cited hundreds of times in the subsequent 15 years. It has become a model for a certain style of reasoning which tries to trace a more or less direct line from a single nucleotide polymorphism in a coding sequence, to a variation in the structure and function of the protein – the enzyme – for which it coded, to an increased susceptibility to pathological conduct. Reasoning in these terms, a cascade of papers were published in the closing years of the 20th century and the early years of the 21st claiming to have discovered susceptibility loci in sequences coding for aspects of the neurotransmitter systems in the brain, relating to depression, anxiety disorders, and the disorders of children such as Attention Deficit Hyperactivity Disorder (ADHD) and conduct disorder (Manuck, Flory, Ferrell et al., 2000).

Much of this work was done with animal models from fruit flies to rodents (Hendricks, Fyodorov, Wegman et al., 2003). Thus, for example, in 2003, Evan Deneris and his colleagues at Case Western University, working with mice, reported the discovery of the Pet-1 gene – only active in serotonin neurones – which when knocked out produced elevated aggression and anxiety in adults compared to wild type controls. The university press release pointed out that serotonin is a chemical that acts as a messenger or neurotransmitter allowing neurons to communicate with one another in the brain and spinal cord. It is important for ensuring an appropriate level of anxiety and aggression. Defective serotonin neurons have been linked to excessive anxiety, impulsive violence, and depression in humans.... Anti-depressant drugs such as Prozac and Zoloft work by increasing serotonin activity and are highly effective at treating many of these disorders. (Press release, ‘Researchers discover Anxiety and Aggression Gene in Mice; opens New Door to Study of Mood Disorders in Humans’, 2003)

And Deneris himself comments: ‘The behavior of Pet-1 knockout mice is strikingly reminiscent of some human psychiatric disorders that are characterized by heightened anxiety and violence.’24
In similar vein, Miczek et al., in their 2007 paper on the ‘Neurobiology of Escalated Aggression and Violence’, move seamlessly from fruit flies to rodents and humans: they write:

Psychopathological violence in criminals and intense aggression in fruit flies and rodents are studied with novel behavioral, neurobiological, and genetic approaches that characterize the escalation from adaptive aggression to violence. . . . One goal is to delineate the type of aggressive behavior and its escalation with greater precision; second, the prefrontal cortex (PFC) and brainstem structures emerge as pivotal nodes in the limbic circuitry mediating escalated aggressive behavior. . . . By manipulating either the fruitless or transformer genes in the brains of male or female flies, patterns of aggression can be switched with males using female patterns and vice versa. . . . New data from feral rats point to the regulatory influences on mesocortical serotonin circuits in highly aggressive animals via feedback to autoreceptors and via GABAergic and glutamatergic inputs. Imaging data lead to the hypothesis that antisocial, violent, and psychopathic behavior may in part be attributable to impairments in some of the brain structures (dorsal and ventral PFC, amygdala, and angular gyrus) subserving moral cognition and emotion. (Miczek, De Almeida, Kravitz et al., 2007: 11803)

Considering the implications of this kind of research on behavioral genomics for violence prevention in 2003, Morley and Hall, of the Australian Institute of Criminology, listed candidate gene variants that have been nominated as having a potential bearing on an ‘individual’s liability to develop antisocial behavioural characteristics’ – variants in the genes for elements of the serotonergic system linked to impulsivity, those for elements in the dopaminergic system linked to ADHD, those for elements in the noradrenergic system linked to ADHD, impulsivity and hostility, and those linked to the activity of enzymes involved in the metabolism of neurotransmitters linked to ADHD, impulsivity, aggression, conduct disorder and criminal conviction – nonetheless stressing that ‘an individual will only have a significantly increased risk of engaging in antisocial behavior if they carry a large number of variant genes’ (Morley and Hall, 2003: 4). 25 And they conclude that while ‘Genetic research is beginning to identify genetic variants that may have some bearing on an individual’s liability to develop antisocial behavioural characteristics’ this was not a matter of single genes – instead, the issue was relocated in the contemporary style of thought about ‘susceptibilities’. Indeed their report was entitled ‘Is there a Genetic Susceptibility to engage in Criminal Acts’, and argued that it was likely that ‘a large number of genetic variants will be identified that, in the presence of the necessary environmental factors, will increase the likelihood that some individuals will develop behavioural traits that will make them more likely to engage in criminal activities’ (Morley and Hall, 2003: 4–5).
This then is the style of thought that is taking shape, not only in relation to antisocial behaviour but also in relation to other problems – polygenic susceptibilities that increase or decrease susceptibility to environmental factors, and hence in appropriate (or inappropriate) circumstances will lead to an undesired outcome. The reference paper for humans themselves is usually that by Caspi and his colleagues (2002) which has been cited hundreds of times in the years that have followed. This was based on research with a large cohort study, and followed Brunner in focusing on the link between the gene for monoamine oxidase A (MAOA) and violent conduct. But its novelty lay in the claim to have discovered ‘gene-environment interactions’. The paper claimed that ‘a functional polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA)’ moderated the effect of familial environment, in this case the effect of maltreatment. ‘Maltreated children with a genotype conferring high levels of MAOA expression were less likely to develop antisocial problems. These findings may partly explain why not all victims of maltreatment grow up to victimize others, and they provide epidemiological evidence that genotypes can moderate children’s sensitivity to environmental insults’ (Caspi, McClay, Moffitt et al., 2002: 851). The authors concluded that although individuals with the combination of low activity MAOA and violence were only a small proportion of the cohort, they accounted for 44 per cent of those with convictions for violence and hence ‘these findings might inform the development of future pharmacological treatments’ (ibid.: 853). This was the first of a slew of studies by this team that were considered by many to change the way in which the relation between genes and environment was conceived in psychiatric disorders: genetic variations at the SNP level were now thought of as producing susceptibilities that were triggered in response to environmental insults. And because they were identifiable sequences that could in principle be identified in childhood by gene-sequencing, the susceptibility to maltreatment might be identified early and intervened on in the name of prevention.

What might be the implications of this line of research? Perhaps the most likely is the development of programs of screening to detect individuals carrying these markers. Neurobiological expertise could thus provide the basis for risk prevention strategies by a variety of agencies of social control, leading to pre-emptive intervention, perhaps by pharmaceuticals, perhaps by other measures. We are already seeing the rolling-out of programmes such as TMAP (the Texas Medication Algorithm Project) for screening adults in relation to major psychiatric conditions, whose version for screening children (TCMAP) proved particularly controversial due to claims that it was acting as a powerful marketing tool for pharmaceutical companies. Other US screening programmes for children include Teenscreen for screening teenagers, together with programmes for identifying young children with ADHD. As far as I am aware, none of these yet uses neurobiological or
genetic tests. But it is certainly possible to envisage the development of school-based strategies, with pre-emptive treatment a condition of continuing schooling. Indeed, now that ADHD is no longer considered a transient disorder of childhood, but potentially a lifetime condition, and indeed a biomarker of later psychopathy, we see yet another instance of the category expansion that might result from the large-scale adoption of screen-and-intervene strategies for problematic conduct.

In another field, given the rise of measures for public protection such as those I have described in the UK, one might imagine post-conviction screening of petty criminals, with compliance with treatment made a condition of probation or parole. Or one can envisage scenarios in which screening and therapy are offered to disruptive or delinquent employees as an alternative to termination of employment. There are suggestive precedents here in the ways in which psychiatric medications such as Antabuse for alcoholics and lithium for manic depression, were introduced in the USA. And biological expertise might be called upon to screen for genetic markers and neurochemical abnormalities, in order to evaluate the levels of risk posed by offenders, or non-offenders with a mental illness diagnosis prior to discharge from prison or hospital. Release would be dependent on compliance with a drug regime. But to conclude, let me give you one example from the UK to illustrate the way in which these relations between genes, brains and behaviour are now being conceptualized.

Consider, for example, the programme of research funded by the Medical Research Council, the Department of Health and the Home Office, and carried out at the Institute of Psychiatry, King’s College London (Viding, Blair, Moffitt et al., 2005; Viding, Jones, Frick et al., 2008; Viding, Larsson and Jones, 2008). This illustrates quite clearly the line of reasoning – perhaps the style of thought – that is taking shape here (Odgers, Moffitt, Poulton et al., 2008). The research has argued as follows. First, that one particular sub-type of antisocial behaviour, that with ‘callous and unemotional traits’ – AS-CU – is a precursor of adult psychopathy. Second, that the characteristics of childhood AS-CU and adult psychopathy map onto one another – in both children and adults we see lack of empathy, lack of guilt and remorse, shallow affect and manipulative conduct, and in such children we can also see precursors of adult violence, such as cruelty to animals and enjoyment of aggression. Third, if we look at studies of psychopaths, psychological tests show them to be poor at empathizing with others and bad at recognizing their fear and sadness, and brain scans show them to have a disruption of one particular part of the brain, the amygdala, which is, so it seems, implicated in these ‘affective’ defects. Fourth, if we take twin pairs aged around 7 years, some monozygotic and some dizygotic, who have been assessed for AS-CU and carry out functional Magnetic Resonance Imaging (fMRI) scans on them, we find the same deficits in the brains’ ‘affect circuitry’ in the AS-CU subjects.
as we see in adult psychopaths. Finally, if we use classical twin study methods, taking environmental factors into account, we find a much higher correlation of these brain patterns in the genetically identical twins than in non-identical twins – i.e. the trait is highly heritable, and suggests a strong genetic vulnerability. Hence, it is argued, we should search for the precise Single Nucleotide Polymorphism (SNP) level variations that underlie this vulnerability. But note, for this line of reasoning, what we have here is a vulnerability, a susceptibility. It is not surprising, then, that those working on this study are closely linked to the Caspi and Moffitt research group, who aim to identify the gene environment interactions which provoke vulnerability into frank psychopathy (Caspi, McClay, Moffitt et al., 2002; Kim-Cohen, Moffitt, Taylor et al., 2005; Odgers, Moffitt, Poulton et al., 2008). The message is not fatalism but early identification and early intervention into the home and family, using behaviour therapy, cognitive therapy and psychopharmaceuticals – for this can reshape brain mechanisms in order to nip those ‘budding psychopaths’ in the bud.

CONCLUSION

I think that, in many departments of life, we are seeing the emergence of a new ‘human kind’: the susceptible individual. In the developments I have discussed here, this takes the form of the person with an elevated neuro-biological risk of being the perpetrator of aggression or violence. This is not merely the modern version of an older idea of the dangerous individual, for biology here is not destiny. As in other areas of contemporary genomics, the relation between biology and criminality is being posed in terms of ‘susceptibility’, and the shaping, exacerbation or mitigation of that susceptibility over a life-course as a consequence of biography, experience and environment. This way of thinking, therefore, is so powerful because it is imbued with hope as well as anxiety. The hope, on the part of many researchers, practitioners and clinicians, is that these susceptibilities can be identified and hence open a pathway to preventive intervention which would steer the susceptible individual onto a more favourable path, one less damaging to herself or himself and less costly to others. Yet the anxiety on the part of policy-makers, and the priority given to the precautionary principle, have the potential to lead to a less optimistic future, in which widespread screening for biomarkers of future psychopathology or undesirable conduct, notably those made possible by developments in genetic profiling and brain-scanning, would lead to a significant increase in preventive interventions in the name of public protection. This is more likely in an environment in which psychiatric professionals have already been given the obligation of governing, and being governed, in the name of risk. And it will find a favourable ecological milieu in a political
and public culture suffused by insecurity and imbued with a fear of all those who appear unable to adopt the forms of responsible and prudent self-management that are obligatory in our current societies of freedom. In any event, perhaps we need to pause, and to ask ourselves what are the benefits, and what are the dangers, of this emerging logic for the conduct of conduct: not so much ‘discipline and punish’, but ‘screen and intervene’.

NOTES

The material presented in this article draws extensively on the text of Chapter 8 of my book *The Politics of Life Itself* (Princeton, NJ: Princeton University Press, 2006), although it is reframed in the current argument. Versions of the paper were given at a workshop organized by the European Network of Neuroscience and Society, Harvard, May 2008 and a symposium at the Centre Koyré, Paris, May 2008, and the Neurocultures Workshop organized by the Brain, Self and Society project at the BIOS Centre of the LSE and the Brainhood project of the Max Planck Institute, Berlin in February 2009. I thank the organizers and participants of these events for all their comments. As my aim was to propose a general argument on the basis of an overview of a wide range of materials, I have chosen to retain the spoken form in this article.

1 For a discussion of the loose controls on research undertaken on the UK’s National Forensic DNA database, see Nuffield Council on Bioethics (2009).

2 See, for a UK example, the programme on ‘Understanding Individual Behaviour’ launched by the ESRC as one of their ‘grand challenges’ in their Strategic Plan for 2005–10:

*Understanding Individual Behaviour and its Relationship to Biological and Social Determinants*: Solving many of the challenges facing UK society depends on improving the effectiveness with which individuals can take control of their own lives. This involves the relationship between individual behaviour and a range of biological, technological and social influences. . . . By 2010 we will have: Collaborated with the Medical Research Council to support social neuroscience research aimed at linking our growing knowledge of brain mechanisms to human and social behavior . . . available at: http://www.esrcsocietytoday.ac.uk/ESRCInfoCentre/Images/Strategic_Plan_2005–10_tcm6–12995.pdf

On neuroscience as a key to educational policy, see the mission statement of the University of London’s recently established Centre for Educational Neuroscience at: http://www.educationalneuroscience.org.uk/mission.html

Or, on the crucial importance of neurobiology in psychiatry, see Lee, Ng *et al.* (2008); Sachdev (2002); and Yudofsky and Hales (2002).

3 An ISI search shows a gradual increase in articles on susceptibility in psychiatry over the 1990s and 2000s: some 891 articles on susceptibility in psychiatry were published in 2008, 829 in 2007 and 752 in 2006; 420 in 1998, 411 in 1997 and 322 in 1996.
4 I will not discuss an earlier phase of speculation on crime as a disease, e.g. Mark and Ervin (1970), or the more extreme claims of Adrian Raine (Raine, Brennan et al., 1994); Farrington (1997); Scarpa and Raine (1997) and Raine and Liu (1998).

5 Brunner (1996) discusses the implications of his research in rather different terms.


7 Dr McInnes describes her current research area as neurobehavioral genetics.

8 http://www.lawandneuroscienceproject.org/

9 The Hinckley case and subsequent cases using brain scans are discussed in Denno (1988).

10 In fact, the implications of the Hinckley case are complicated because, under US federal law, the prosecution had to prove beyond reasonable doubt that the defendant was sane at the time of the offence; in most individual states and other jurisdictions the defence must prove by the preponderance of evidence that the defendant is insane – a standard which would probably have led to a conviction for Hinckley.

11 By 1992, for the first time in the USA, a court allowed an expert to draw upon evidence from a PET scan in determining the defendant's sanity, although in the end the matter was resolved by lowering the charge from murder to manslaughter and avoiding a trial: see People v. Weinstein, 591 NYS.2d 715 (Sup. Ct. 1992). The court concluded that expert evidence and consideration of the results of a PET scan and other physiological tests – to indicate a cyst and metabolic imbalances in the defendant’s brain – was not unreasonable in making a diagnosis of insanity, but agreed to negotiate a reduced charge from murder to manslaughter, rather than going to trial. In the trial of Michael Person, in New Haven, Connecticut, in early 1998, prosecutors contested the attempts of defence lawyers to present the jury with PET scans showing brain abnormalities, and to introduce the findings of Adrian Raine on the increased prevalence of abnormal brain scans in convicted murderers in seeking to reduce the charge from murder to manslaughter. Another early case involving a request to introduce brain scans was that of Jack Dempsey Ferrell, who was convicted in 1995 of the first-degree murder of his girlfriend in 1992. At his appeal hearing before the Supreme Court of Florida in 2005, Ferrell contended that his counsel at his first trial should have supported arguments that he had suffered neurological impairment with a brain scan. At hearings between the original trial and the final appeal, the state had objected to the request for SPECT scans to be conducted, not only because there was already a diagnosis of frontal lobe brain damage which had been taken into account as a mitigating factor, but because no scan could show how the physical brain affected Ferrell’s capacity to function. The Supreme Court of Florida, in its ruling of 16 June 2005 (No. SC03–218), was of the view that ‘a particularized showing of necessity is the polestar for whether any diagnostic test should be authorized’ and that this had not been demonstrated in Ferrell's case.

12 More recently some have suggested that Kinkel was suffering from adverse effects of Prozac and Ritalin that he had allegedly been taking at the time of the murders and extended this claim to the young people responsible for a number of other school shootings in the USA. See, for example, Dan Edwards, accessed 29 August 2005, at: http://www.geocities.com/StNektarios/BIOPSYCH.html
The fullest account of this case that I have been able to find is that provided in 2005 by Court TV and accessed 30 August 2005 at: http://www.crimelibrary.com/serial_killers/predators/stayner/ I have drawn on that report here.

Raine’s work, cited above, also has come to focus on the issue of risk and prediction (additionally, see Raine, 2002).

Of course, concern with troubling and troublesome individuals who appear to fall between the jurisdiction of the two great apparatuses of confinement – the criminal justice system and the psychiatric system – is long-standing. However, as I and others have argued, the shift from dangerousness to risk thinking, which can probably be dated to the 1980s, and is firmly in place in most countries by the 1990s, marks a significant mutation. The work of Monahan and Steadman, which I discuss below, is exemplary here. See Duggan (1997), Rose (1996) and Rose (1998a, 1998b).

In the UK, the Consultation Document on the Draft Mental Health Bill of June 2002 makes an interesting distinction between danger and risk. ‘Some people . . .’, the ministers say in their Foreword, ‘because of their illness, can be a danger to themselves, whilst a very few can, at times, pose a risk to others.’ This distinction of danger and risk is not sustained in the arguments around the Bill (Department of Health, 2002).

The original comment can be found in Monahan (1981).

Homicide figures vary from around 600 to 750 depending on the method of calculation. See Office of National Statistics (2003).

On the basis of their examination of public inquiries into homicides by people with mental illness, Munro and Rumgay argue that improved risk assessment has only a limited role in reducing homicides and that more deaths could be prevented by improved mental health care irrespective of the risk of violence (Munro and Rumgay, 2000).

A special supplement of the British Journal of Psychiatry, in 2007, was devoted to articles debating the nature and efficacy of the DSPD programme – see British Journal of Psychiatry 190(suppl. 49) (2007) (doi: 10.1192/bjp.190.5.s1). The figure of £100 million does not include the cost of building the four DSPD units at Special Hospitals. The cost of confining an individual in a high-security hospital is over £200,000 per patient per year (Maden, 2007).

The sentences are set out by the National Offender Management Service of the Ministry of Justice at: http://noms.justice.gov.uk/managing-offenders/sentences/punishment/Life-sentences/ For a discussion and rather critical review of the Indeterminate Sentence for Public Protection by Her Majesty’s Inspectors for Prison and for Probation, see: http://inspectorates.homeoffice.gov.uk/hmiprisons/thematicreports1/IPP_thematic_(2008).pdf

The 2007 figures are from the Ministry of Justice, Sentencing Statistics 2007, England and Wales (2009: 36). These figures include both the sentence of IPP and the related sentence of Extended Sentence for Public Protection. The extrapolation, credited to a Home Office official, is given in a discussion of the sentences by David Rose (no relation) in the New Statesman in 2007, and can be found at: http://www.newstatesman.com/politics/2007/03/risk-prison-sex-act-life

Accessed 24 February 2009 at: http://www.dshs.state.tx.us/mhprograms/tmapover.shtm


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BIOGRAPHICAL NOTE

NIKOLAS ROSE is the James Martin White Professor of Sociology and the director of the BIOS Centre at the London School of Economics and Political Science.

Address: London School of Economics and Political Science – BIOS Centre, Houghton Street, London, WC2A 2AE, UK. [email: n.rose@lse.ac.uk]