The Neurochemical Self and its Anomalies

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October 2001

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Published as
What are the implications of recent advances in the sciences of life for the government of human conduct? In this paper, I want to consider this question in relation to one specific area - the contemporary government of disorders of desire. Not disordered desire in the sexual sense – which, despite the colonial ambitions of psychoanalysis, was never more than one small corner of the empire of desire.\(^1\) I want to consider those other forms of disordered desire known as cravings and addictions - disorders that Mariana Valverde (1999) has termed ‘diseases of the will’ I am interested in the mutations that may be occurring in our ways of thinking about and acting upon these pathologies in the context of recent developments in the life sciences and biomedicine - the decoding of the human genome, the new neuroscientific knowledges of the brain and its mechanisms, and the linked developments in the technologies of treatment, notably psychopharmacology. In this shift, these 'diseases of the will' have become, at least in part, diseases of the brain. One index of this is the emergence of a number of new kinds of drugs that are being used in the treatment of addictions – or as psychiatrists now like to call them, ‘dependencies.’

The best example here is probably a drug called naltrexone. Naltrexone is used in the treatment of addiction to opiates and of alcoholism – or what is termed in the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-IV) (1994) alcohol dependency. Now the interesting thing about this class of drugs is that they treat addictions not by making the satisfaction of desire unpleasant, as in antabuse, and not by creating a substitute but more acceptable addiction, as in substituting methadone for heroin, but, apparently, by removing or reducing the desire for that which was previously desirable. They act upon desire. Take this drug, and the desire, or craving of the alcoholic for drink, of the junkie for drugs, will diminish.
Whilst this is the story reported in the popular press, the arguments in the scientific or professional literature are more complex and nuanced (see chapter 15). But my aim here is to use these endeavours to reshape pathological desire by pharmaceutical means as a way of trying to understand some more general shifts in the government of pathological conduct. On the one hand, I will argue, we can see a shift in the kinds of persons upon which such government will act – the birth, that is to say, of genetic and neurochemical selfhood. On the other hand, we can see the re-birth of an old governmental aspiration that Mariana Valverde terms ‘targeted governance’. Targeted government – the direction of regulation towards those specific sites and persons thought most problematic - may seem at odds with the important argument that control, today, is not individual and reformatory but collective and probabilistic, aimed at risk reduction rather than therapy or reformation. But there is no real contradiction here. In post-social strategies of government, much control is indeed ‘designed in’ as suggested by terms such as actuarial justice, societies of control and the like (Feeley and Simon 1992; Deleuze 1995). That is to say, all manner of everyday locales such as amusement parks, and practices such as shopping, have been explicitly shaped to secure specific types of conduct. But such attempts to 'design in control' are coupled with strategies that seek to target specific ‘high risk’ or ‘high need’ persons or sites which are thought to require particular attention (Rose 2000a).

My specific focus is the pharmaco-therapeutics of desire. This is not quite what Peter Kramer (1994) was thinking of when he coined the term ‘cosmetic psychopharmacology’, though he did talk about the way in which Prozac – the cosmetic he was discussing - diminished the compulsion that one of his patients had to make use of pornography And Prozac was an SSRI, a serotonin selective reuptake
inhibitor, but naltrexone is something different, as we shall see. But despite these difference, the argument here partakes of something of the same way of thinking. And this way of thinking is exemplary of a broader shift in what I have termed, after Ludwik Fleck and Ian Hacking, the ‘style of thought’ of biological psychiatry (Rose 2000b; see also Fleck 1979; Hacking 2001). In this style of thought, the old oppositions between mind and brain are starting to be re-posed. The psychological space between organs and conduct that opened up in the twentieth century is beginning to be flattened out. Individuals are becoming somatic – in this case genetic and neurochemical – selves. Yet, contrary to may predictions of the critics, this is not a rebirth of essentialism, reductionism, geneticism and the like. As Carlos Novas and I have argued, there is plenty of scope here for autonomy, choice, individuality, responsibility and the like (Novas and Rose 2000).

**Molecularisation**

The Fourth Edition of the *Diagnostic and Statistical Manual* of the American Psychiatric Association (1994: 194-204) identifies 15 kinds of Alcohol Related Disorders divided into two types – Alcohol Use Disorders which includes Alcohol Dependency and Alcohol Abuse - and 13 varieties of Alcohol-Induced Disorders from Alcohol Intoxication (!) to Alcohol-Induced Sexual Dysfunction. Each has its own code, and its own diagnostic criteria. These are just a few of the approximately 350 categories of psychiatric illness which DSM-IV defines. Attempts at the systematic classification of types of mental illness in the United States began as a statistical, not a clinical project: the 1840 census records the frequency of “idiocy/insanity”; the 1880 census used seven categories – mania, melancholia, monomania, paresis, dementia, dipsomania and epilepsy; and the first official nosology, adopted in 1918, was an
attempt to classify the considerable population of individuals then confined in mental hospitals.

Over the twentieth century, the governmental and the clinical aspirations fused, and classification systems show the rise and fall of different conceptual models of mental illness. The first DSM, produced in 1952 in the wake of psychiatry’s wartime experience, was constructed in terms of Adolf Meyer’s notion of reaction types and conceived of mental disorders as reactions of the personality to psychological, social and biological factors. DSM II, published in 1968, had 180 categories framed in the interpretative language of psychoanalysis. DSM III, published in 1980, is often seen as a response to the crisis in legitimacy of psychiatry over the 1970s, a response that indicated a reshaping of the psychiatric gaze. Where DSM II was 134 pages long, DSM III ran to almost 500 pages, and the revised version of 1987 had 292 categories. This was a shift to a new way of seeing mental disorders as discrete illnesses, each marked by a number of supposedly objective criteria, and each, in principle at least, having a specific aetiology and prognosis, and being amenable to a specific kind of treatment. So the contemporary proliferation of categories may be more than bureaucratic inflation. Despite DSM-IVs recognition, at the outset, that individuals within any diagnostic group are heterogeneous, and that its categories only serve as aids to clinical judgement, this refinement and proliferation of categories of pathology is indicative of something more – which we might term 'the molecularisation' of psychiatry.

For the thought style of contemporary psychiatry has become inextricably bound up with developments in neuroscience that posit a specific anomaly in the brain - most frequently in one of the neurotransmitter systems - as the ground of each specific anomaly in mood, cognition, affect or conduct. Thus, for example, a
particular type of depression might now be linked to an anomaly in just one of the many subtypes of one of the seven sub-families of receptors for the neurotransmitter serotonin. And, as a further stage in this molecularisation, the new genomics seeks the precise polymorphism in a particular sequence of bases in a locus in a particular gene that is correlated with a precise type of disorder of thought, emotion or conduct. For example, attempts are made to link a particular sub-category of DSM-IV schizophrenia to a substitution in a single base in the sequence of DNA of a particular gene localised to a precise place on a particular chromosome, leading to a substitution of one amino-acid for another in an enzyme involved in neurotransmission. In this style of thought, the broad categories of the start of the twentieth century – depression, schizophrenia, neurosis – have given way to a wish to dissect pathologies of mood, cognition, will or affect at a different scale. The psychiatric gaze is no longer molar but molecular, and here this phrase should be taken literally.

The same ‘molecular’ dissection of pathology can be seen in psychopharmacology – the fabrication, trialling, marketing and prescribing of psychiatric drugs. Prozac, for example, became an iconic drug not because it was more effective than previous antidepressants, but because it purported to be the first drug whose molecule had been deliberately fabricated to disrupt one, and only one, aspect of a single neurotransmitter system. The claim was that this drug, fluoxetine hydrochloride, increased the level of available serotonin in the synapses by disrupting the reuptake of serotonin, and that it did this by locking into the precise sites on the neuronal membrane, the receptors, through which reuptake would otherwise have occurred. Hence all the hype about Prozac being a smart drug, a clean drug, which didn’t produce all those unwanted effects – shuffling gait, dry mouth, tremors and the like – that were produced by the earlier clinically derived ‘dirty’ anti-depressants.
which hit a number of sites in a number of systems at the same time. The dream, that is, is of drugs that don’t act upon the person as a whole, but are targeted precisely to correct a specific anomaly now thought to underlie a specific undesirable variation of mood, emotion, conduct or will.

These new ways of thinking have consequences for conceptions of risk and strategies for its management. I have argued elsewhere that the political vocation and professional practices of psychiatry have been restructured in terms of the management of risk. One might say that these individuals with inherited molecular anomalies, which presumably they have had since birth, are persons ‘at risk’ and that interventions directed at them are yet more examples of ‘risk government’. This would not be too misleading. But I think we loose something in intelligibility by re-coding these endeavours in the language of risk. Risk implies probabilities and calculabilities. What we have here, to use the terms usually employed in the specialist literature, are predispositions, vulnerabilities or susceptibilities. And these susceptibilities are now somatic and molecular. We are seeing the emergence of an idea of the susceptible somatic individual. And I will argue here that this idea of susceptibility has very significant consequences for what I will call, for want of a better term, control strategies.

These new ways of thinking also have implications for the ways in which mental pathologies are coloured with moral significance. One of the prevailing themes in sociological critiques of psychiatry from the 1960 onwards, in the work of Thomas Scheff and Erving Goffman, was that a psychiatric diagnoses acted as a ‘master status.’ It over-rode and effaced all other categories of selfhood: the individual so diagnosed was not ‘suffering from’ schizophrenia but was ‘a schizophrenic’. And we know too that, from the mid-nineteenth century onwards, the catalogue of such
master-statues collected together an array of problematic figures – the criminal, the juvenile delinquent, the alcoholic, the prostitute, the degenerate, the homosexual. These did not merely refer to those who engaged in particular forms of undesirable behaviour, but designated a particular type of person. Their individuality was suffused by, exhausted by their particular pathology, from the shape of their ears to their handwriting, their posture and gestures, from the tone of their voice to their intelligence and capacity for moral judgement. These were ‘the abnormal,’ those whose violation of norms was not a mere matter of an irregularity of conduct but of deviant personhood. But I will argue here that we are seeing a shift in the logic of the norm that underpinned such notions of ‘the abnormal’. I will try to show that, at the least, we are moving from dividing practices based on the binary of normality and abnormality to practices based on the idea that all individuals vary, and that most, if not all, suffer from molecular errors that are potentially correctible. In this new logic of correction, I shall suggest, what is required is a kind of constant adjustment of biology and circumstances.

**Biological Psychiatry as a Style of Thought**

When I say biological psychiatry is a style of thought (Rose 2000b), I mean to imply that it is a way of seeing, a way of explaining, a way in which reasoning is embedded within certain practical and intellectual techniques, conventions about experimentation, about instruments and inscriptions, measurements and model systems. These are conventions about sense making activities themselves and about the terms under which they may be criticised, corrected and revised. A style of thought is not just a type of explanation, it is about what counts as an explanation, and it is also about what there is to explain. That is to say, it is about the very object of
explanation, the set of problems, issues, phenomena that an explanation is attempting
to account for. A new style of thought brings, or tries to bring, a new object into
existence, or so modifies an old object that it appears in a new way, with new
properties, and new relations and distinctions with other objects. Let me examine this
particular style of thought in more detail.

In the nineteenth century, clinical medicine as we know it began when the
gaze of the doctor plunged into the interior of the body, linking the symptoms that
could be discerned on the surface of the body with the organic locations and lesions
within. From that moment onwards, to diagnose an illness was to interpret symptoms
in terms of the internal organic malfunctions that were their cause. The new
profession of psychiatry inevitably dealt with people who came to their notice, first of
all, because of a breach of the conventions of order, conscience, civility and
personhood. But despite their conviction that they were dealing with a disease of the
brain, and despite dissecting innumerable brains of deceased asylum inmates,
nineteenth century psychiatrists failed to ground the symptoms of madness in lesions
or abnormalities within this deep corporeal interior. Instead, nineteenth century
doctors of the mad used their educated vision to make their diagnoses. Drawing and
later photographs of the varieties of madness filled the textbooks from Esquirol's
*Atlas* of 1838, through Bucknill and Tuke's *Manual* of 1874 to Charcot's *Iconographie*
which was published from 1877 to 1918 (Gilman 1982; Rose 1998). This psychiatric
gaze focussed upon the surface of the body – posture, gaze, the colour of the skin of
the melancholic, the gestures of the maniac, the movements of the hysterical. In the
twentieth century, the eye gave way to the ear – whether in Freud or in Kraepelin, the
eye lost its diagnostic power. For Kraepelin it was the distinctive biography, the
aetiological pattern, diagnosed by the case history, that was the diagnostic material.
For Freud, as we know, it was the voice of the patient, what was said, that was to be interpreted, and that provided the royal road to a diagnosis. In each case, an interior psychological space opened up between the organs and conduct. Biography, environment and the like deposited their traces in here. Motives, desires, aspirations flowed from here. Understanding of pathology must locate its explanations here. The psyche became the ‘obligatory passage point’ and mental ill health now seemed undeniably to be a psychological matter.

But today, an augmented, prosthetic gaze seems to have opened up the corporeal basis of mental pathology to the gaze of psychiatry, and, in so doing, to have supplied it with an new objectivity. The previously inaccessible bodily seat of the living mind now seem to be opened out and spread before the psychiatric gaze. Brain imaging shows the differences in the regions that light up in the ‘normal’ and ‘schizophrenic’ brain, or fail to light up in the brain of a depressed person. Molecular neuroscience anatomises the neurones, the receptors, the neurotransmitters, the cell membranes and ion channels, and allocate a specific pattern of functioning to each normal or abnormal mental state. Genetics seeks the gene sequences that correlate with each diagnostic category - to identify the precise functions of the variations they discover – a deficiency in the metabolism of a neurotransmitter, in the receptors, in a particular channel. Pharmaceutical companies fund most of this research: they compete to engineer and manufacture chemical correctives to these molecular errors. In this process, psychiatry claims to have overcome, at last, the Cartesian dualism of body and soul. The deep psychological space that opened in the twentieth century has flattened out. In its new ‘neurochemical’ account of personhood, psychiatry no longer distinguishes between organic and functional disorders, with only the former being thought of as somatic. It no longer concerns itself with the mind or the psyche. Mind
is simply what the body, what the brain, does. And mental pathology is simply the
behavioural consequence of an identifiable, and potentially correctable, error or
anomaly in some aspect of the brain, in its neurotransmitters, receptors and the like.

What is this new self? First and foremost, it is neurochemical. The
neurochemical model of the brain started to take shape in the 1970s. It was now
believed that fundamental processes of the brain had to do with neurotransmission –
the communication of signals between neurones – across the synapses or points of
contact between nerves. Initially it had been thought that although nerves themselves
transmitted signals by chemical means, transmission across the synapse was electrical.
By the 1960s, largely as a result of work on the new psychiatric drugs – first the
antipsychotics such as chlorpromazine, then the antidepressants such as imipramine
and iproniazid - not forgetting the experiments on lysergic acid diethylamine - it had
been accepted that neurotransmission was carried out by chemicals. The first
neurotransmitters identified were the monoamines (dopamine, norepinephrine,
epinephrine, acetylcholine and serotonin), later some amino acids were also found to
be neurotransmitters (notably gamma aminobutyric acid or GABA) and by the start of
the twenty-first century, the number of chemicals believed to be involved has grown
into the hundreds.

All these discoveries were implicated in a rather radical reshaping of the
technologies of psychiatric truth. This had a number of components. A whole
experimental system was brought into being for the development of drugs and the
testing of hypotheses, involving animal models, notably rats, in-vitro systems, and
complex new types of apparatus, idealised objects on which scientific thought would
now work. New entities were brought into existence – receptor sites, membrane
potentials, ion channels, synaptic vesicles and their migration, docking and discharge,
receptor regulation, receptor blockade, receptor binding - first as hypothetical, then as demonstrated in the lab, then as experimental entities in their own right, finally as realities than can be rendered visible by PET scans or other visualisation techniques. New truth technologies were invented, notably the randomised controlled trial. New forces became involved, not just teams of research scientists, a whole host of new specialist journals and the like, but also big grant giving agencies, and, of course, the pharmaceutical companies.

These processes were not merely processes of discovery, but of intervention – the neurochemical brain becomes known in the very same process that invents interventions to manipulate its functioning – that is to say, therapeutic drugs. Psychiatric truth now became intrinsically linked up with the maximisation of what Catherine Waldby (2000: 19) has termed 'biovalue.' The imperative to produce a kind of surplus value from vitality and its problems drives the new symbiotic relation between knowledge, profit and products. We have a new capitalisation of the treatment capacities of medicine, as pharmaceutical companies are driven by the aim of maximising shareholder value through the marketing of drugs with a mass worldwide sale. We have a capitalisation of truth itself, as vast sums of money, expensive equipment and computing power and huge research teams are required to produce biomedical truth. And we have a capitalisation of the organs, tissues and cells of the body, as the human genome has become the territory for a new goldrush for venture capitalists and patent lawyers. Those aspects of life that were previously devalued as pathology, whose humane treatment and welfare was a drain upon a national economy, are now vital opportunities for the creation of private profit and national economic growth. Paul Rabinow’s (1996) assessment of the new life sciences is especially apt here.
The quest for truth is no longer sufficient to mobilise the production of psychiatric knowledge. Health, or rather the profit to be made from promising health, has become the prime motive force in generating what counts for our knowledge of mental disorders.

These developments were each to have their implications for the explanation and treatment of addiction. In the 1970s it began to be argued that one might treat addiction with the new psychotropic drugs that were now thought to act on these neurotransmitter systems – affecting the secretion of neurotransmitters, their uptake, or the receptor sites on the cell membranes that they bound to. Drugs such as lithium that had been used to treat depression were now being tried out for alcoholics, though the rationale was rather confused – perhaps lithium had a direct effect, but perhaps its effect might be mediated by the fact that alcoholics were actually depressed. Pharmacological treatments were, in any event, not seen as the primary therapeutic strategy. Rather, they were 'primarily pharmacological adjuncts and should be used only in conjunction with behavioural and psychosocial therapies' (Peachey and Naranjo 1984). Drugs like disulfram (Antabuse) might produce aversive responses, thus leading to deconditioning. Or the benzodiazepines might help, because they reduced the anxiety associated with withdrawal. Some speculated that the use of the addictive drugs was itself a kind of self-medication for a psychiatric disorder, in which case one should first treat the underlying depression or whatever and the alcohol use would diminish.

Ten years later, however, a new style of thought had taken shape, in which the brain and its mechanisms become the primary target for explanations and treatments of addiction or dependence. Leonard (1992: 189) stated the position baldly, 'Most of the theories of dependence envisage a change in one or more neurotransmitter systems
upon which the drug acts. Thus sedatives would be expected to facilitate GABAergic transmission and it may be hypothesised that these receptors are desensitised following chronic administration of these drugs. The withdrawal effects would then be postulated to result from a rebound hypersensitivity in receptor function. Actually alcohol remained a difficult case. It was in relation to other drugs of abuse that the neurochemical model of dependence first firmed up.

Opiate addiction - and opiate dependence were now allocated to the mu, delta and kappa opiate receptors. Benzodiazepines and sedatives were allocated to the GABA-A receptor, where it was claimed that they mimicked the action of indigenous substances on this receptor, boosted chloride conductance through a chloride channel, and thus enhanced inhibitory neurotransmission (turning down the action of the neurones in question) and had anxiolytic effects. Tolerance was thought to arise because, after awhile, the number of these receptors in the relevant brain sites decreases and/or the receptors become less sensitive. Either way, they become less effective in modulating neurotransmission in response to the drug. This not only leads to a need for more drug to produce the desired effect, but also the reduction in the numbers or functional sensitivity of GABA-A receptors generates panic disorders when the drug is discontinued. Along similar lines, cocaine was thought to inhibit the dopamine reuptake system, hence leading to more active dopamine in the synapse. Chronic use was thought to inhibit the dopamine transporter even more, leading to an excess of dopamine in the brain, a constant dopamine high, paranoia and so forth. The same was true, in a milder form, for amphetamines. PCP was thought to be an allosteric modulator of the N-methyle-D-aspartate (NMDA) subtype of the glutamate receptor (glutamate being another neurotransmitter), blocking the receptor and decreasing the flow of calcium into neurone, which is thought to produce sedation,
decrease anxiety and so forth. In this style of thought, nicotine works on the cholinergic receptors, first stimulating them to produce dopamine, then blocking them, preventing the normal stimulation of the nerves in the mesolimbic area that release dopamine – hence the mini-rush, slow down, mini-rush at next puff and so forth! But after awhile, the receptors respond to being turned off by becoming more sensitive, hence making it even more difficult to give up. Nicorette patches work, it seems, because they release a small controlled constant dose of nicotine, hence modifying this up and down mechanism, and helping the smoker give up.

Our drug, naltrexone, was first developed to treat addiction to the opiates. It is here that we see its effect as a specific antidote to pleasure. It does this by binding to the specific opiate receptors, because it is the same shape as the molecules that normally stimulate them. One needs to think here in terms of keys and locks. Binding of the 'therapeutic' drug to these receptors blocks them, hence preventing the 'drug of abuse' from binding to them. And since the drug of abuse can no longer bind to the receptors, it can no longer cause pleasure. Of course, it takes awhile for this message to get through to the user whose learned habits endure for awhile despite the absence of the neuronal rewards that had underpinned the original learning of them. This is why, in the treatment regime, the user must not be forced or persuaded or even allowed to come off the drug at this point. He or she must keep using the illegal drug whilst taking the anti-pleasure drug. In this way, the links between the use of heroin or whatever and the effects of pleasure will gradually weaken. The learned connection will be extinguished and, hopefully, the addict will no longer seek pleasure in this way.

What, then, of alcohol? The books tell us that the pharmacology of alcohol is ill understood, and up until recently the argument was that its effects, though
undoubtedly mediated through the neurotransmitters, were relatively non specific. But, it is no surprise that, despite this, some postulate the existence of an 'alcohol receptor'. In this case, the receptor of choice is the GABA-A receptor. Alcohol, it seems, may enhance inhibitory neurotransmission at the GABA-A receptor, by reducing transmission of chlorides through the little ion channel into the cell that the GABA-A receptor controls. This would enhance inhibition and reduce excitation of these receptors. Others think that alcohol may also work on dopamine receptors. Others think the serotonin system may be involved. But, in any event, molecular precision is required in explanations that are to be candidates for truth: they must work in terms of anomalies in receptors understood in terms of their specific shapes, or in terms of the production of particular enzymes, or in terms of ion channels and the like (DeChaderevian and Kamminga 1998). Take this press release from the National Institute on Alcohol Abuse and Alcoholism. It reports the identification of: a new cell membrane channel where ethanol, the alcohol found in intoxicating beverages, may act. Neurobiologists from the Waggoner Center for Alcohol and Addiction Research and Section on Neurobiology, and the Department of Pharmacology and Toxicology, College of Pharmacy, University of Texas (UT) at Austin discovered actions of alcohol while studying a subtype of potassium channels, a diverse family of ion channels that perform many central nervous system functions. Identification of the alcohol-sensitive channel has significant implications because of its key role in brain function. Molecular analysis of this cell membrane channel ultimately will increase our knowledge of how alcohol affects the brain and, thereby, the way a person functions, said Enoch Gordis, M.D., Director, National Institute on Alcohol Abuse and Alcoholism (NIAAA), a component of the National Institutes of Health.
Our drug, naltrexone, seems to reduce the pleasure in drinking in the same way as it reduces the pleasure in opiates. It is known to bind to opiate receptors. Hence, it seems plausible to suggest that alcohol may also cause the release of endogenous opioids, and that naltrexone may block effect of these. This would explain how it works for drink as well as for opiates. And, as in the treatment for opiate addiction, it will only work if the patient keeps drinking whilst taking naltrexone. For it is necessary for the subject themselves to find that the alcohol no longer produces pleasure or satisfies craving. Only then will the learned response linking booze to pleasure be extinguished (see chapter 15).

Once more, what is involved here is a nice combination of neurochemical and behavioural selves. But this behavioural self is not inhabited by the deep psyche of the psychodynamic era. It is the flattened out psyche of thought and learning that is in play here. A two-dimensional web of learned ways of thinking and acting now mediates between neurochemistry and conduct. And, in this web, aberrant conduct is learned through mechanisms that are, themselves, not aberrant - undesirable habits and thoughts, here, are the outcome of processes that may be pathological in their outcomes but are not pathological in themselves. And what is learned may be unlearned, or may be replaced by a new and less damaging set of learned ways of thinking and acting, an array of competences and skills of life management that are more desirable to others, and indeed to the dependent person themselves.

From Normalisation to Correction

Why do I suggest that this new conception of pathology, in this case of alcohol dependence, represents a shift from away the idea of ‘the alcoholic’ as a deviant personality – from all those arguments about master statuses, degradation ceremonies,
moral careers and the like? For the initial diagnosis seems normative enough. The criteria for a diagnosis of alcohol dependence, as given in DSM-IV for example, only appear as such in relation to conventional cultural standards of personhood, conduct and public order and reproduce all the well known problems of such diagnoses. Alcohol dependence is located within the general class of substance abuse, whose criteria include failure to fulfil role expectations at home or at work, neglect of conventional obligations, neglect of conventional criteria of safety and danger, and continued use despite the cause or exacerbation of recurrent social or interpersonal problems (American Psychiatric Association 1994). And, as in the conventional criticisms of biological reductionism, we seem to have the familiar reading from the social to the vital, as these 'maladaptive behaviours' are interpreted as the specific consequences of malfunctioning at the biological level, in this case mediated through excessive consumption of the substance in question. But despite Dr. Gordis’ reference to 'the way a person functions,' what we appear to have, at this biological level, seems to have, itself, no moral weight. It is merely an error, a mistake in a bit of the machinery of the brain and its neurochemistry. And this is an error that is potentially correctable, for if it is merely a question of a chemical malfunction, we can, potentially, fabricate an artificial alternative, a drug. And once the neurochemical error has been corrected, the erroneous conduct can itself be remodelled in a desirable direction. It too can be seen as a correctable error of thought and learning.

That is to say, I think we are seeing, at least, a mutation in 'the logic of the norm'. What do I mean by ‘norm’ here? It is clear that the terms normal and abnormal are used in this new discourse, and I have used them many times in my discussion so far. So I am not suggesting an abolition of the terms as such. But I am suggesting a change which relates to the specific sense in which they began to be used in the
sciences and technologies of life in the middle of the nineteenth century. As is well
know, the French historian and philosopher of the life sciences, Georges Canguilhem,
argued that this nineteenth century sense of the term was problematic. He posed this
problem in terms of a fundamental distinction between vital norms and social norms
(Canguilhem 1978). He asserted that life was itself a vital and normative process.
Vital norms arose from the very nature of living beings and the constant work of
adaptation that they try to do in order to resist death. The vital norms of the living
being, as they are used in medicine and biology, are not merely human judgements or
statistical averages across a population: they arose from the specific character of their
object, of living beings. He writes 'It is life itself, and not medical judgement that
makes the biological normal a concept of value and not a concept of statistical reality'
(ibid.: 43). Health, for the living organism, is not just normality but normativity, the
vital force that resists disease. Pathology was a reduction of this vital normativity;
ilness was fixity, inability to adapt, stasis. Life was organic, systematic, self
regulating, hence the importance of disease for knowledge: 'Disease reveals normal
functions to us at the precise moment when it deprives us of their existence… Health
is organic innocence. It must be lost, like all innocence, so that knowledge may be
possible… the truly vital wonder is the anguish caused by disease' (ibid.: 52).

Canguilhem thought that social norms were very different. Social norms
manifest only adaptation to a particular artificial order of society. They arise from the
requirements of those in power for normativity, docility, productivity, harmony and
the like. In a widely quoted passage, he remarks that 'Between 1759, when the word
"normal" appeared, and 1834 when the word "normalised" appeared, a normative
class had won the power to identify - a beautiful example of ideological illusion - the
function of social norms, whose content it determined, with the use that that class
made of them' (ibid.: 151). The norms of the economy, in the school, in the legal system and so forth thus arose from judgements of desirability made by groups with particular interests. And the most intense problems arise where one set of norms are read in terms of the other, when social norms were treated as if they were vital, and arose from the nature of the human being, rather than from a contingent social judgement of personhood.

I think we can see what Canguilhem was trying to get at. But I don’t think we can accept his distinction of vital and social. It is true that a new notion of the norm took shape within programmes for the government of individual and collective existence in nineteenth century Europe. These programmes gridded the space and time of existence with standards of conduct – of dress, manners, punctuality, conduct, performance. These were the standards that underpinned the norms that enabled judgements of deviation to be made in the schools, factories, army, hospital, reformatory, and processes of normalisation to be set in motion. And these norms were linked up with those of medicine. Violations of norms of conduct were brought into relation with the idea of abnormalities in the healthy functioning of bodily processes.

So far I seem to be agreeing with Canguilhem. Where I differ is in relation to the norms he terms vital. For these could only arise within specific apparatuses for the government of health. One such apparatus was the enclosed institutional space of the hospital. Large numbers of sick people were observed, their details were recorded, as they were citizens after all. Their symptoms were observed and written down in case notes. Their prognosis was followed. And after they were dead, their corpses were opened up by dissection, and the organic seat of their sad decline was revealed to the medical gaze. Vital norms arose only when the individual gaze of the doctor or the
anatomist could be statisticalised in these ‘machines for cure’. But vital norms were social too: as Foucault was fond of pointing out, medicine was the first ‘social’ science. The norms of longevity, morbidity, reproduction and so forth arose in projects for the government of collective health and sickness. They were mapped out through statistics of birth, death, rates and types of morbidity. They emerged in practices ranging from sewage systems to insurance that regulated the population in the name of health. All these processes of biopolitics were conditions for the emergence of the notion of the idea of the vital normativity of the living human being and the human population.

So my point is that social and vital norms of individuals and populations have always been inextricably intertwined at the very heart of medical knowledge. It was in the universal and compulsory practices of schooling that the idea of ‘normal development’ in the child was formed, including normal physical development, and all the techniques of weighing, measuring, assessing were invented: they solidified the idea that there were biological norms of height, weight and development and that deviations were biomedical abnormalities - slow development, obesity and so forth. Our norms of procreation, which radically changed the vital lives of women, emerged within domestication of sexuality and reproduction in the family. Our beliefs about which human ills were normal and which were treatable arose from the transformation of the home into a machine for creating and maintaining hygiene. Norms of the labouring body arose from the penetration of the gaze of welfare medicine into the workplace, which also gave us the apparently natural life course - the times when one was too young to labour, the times of labour, and the times of retirement. And so forth.
So perhaps we can say, in medicine as much as in pedagogy, that from the nineteenth century onwards, the idea of the norm involved an overlaying of social and vital ways of thinking. More precisely, the term ‘normal’ condensed a statistical, a social, a moral and a medical judgement. The normal was that which was average, was socially desirable, was good or virtuous, and was healthy. The norm combined or aligned the register of the statistical - the central point in the normal distribution which captured the regularities found in populations of numbers - and the register of the social and moral - the judgments of authorities about the desirability of certain types of conduct - and located these twin registers in a medical field of judgments of health and illness.

Pathology was abnormality in all these senses. We are very familiar with this move in the case of intelligence. For the statisticians of intelligence such as Karl Pearson, this was merely one exemplar of the fact that many, if not all, mental and moral qualities were normally distributed in the population, as were other conditions, such as tuberculosis, which manifested an inherited weakness of constitution. But it went much wider. Thus, as Ian Hacking has pointed out, at the turn of the nineteenth and twentieth centuries, many thought that the tendency to commit crime was both biologically based and normally distributed in the population. His example is Charles Goring's *The English Criminal* of 1913, which tried and failed to confirm Lombroso's view that there were physical stigmata of criminals, but nonetheless argued that 'the criminal diathesis' was normally distributed in the population. As Hacking (2001: 149) puts it, "The assumption of normalcy, the bell-shaped curve, the Gaussian distribution, is enormously powerful as a mathematical tool for analysing the tendency to criminal behavior." But there is no reason to believe that these variations at the molecular level of base pairs gene sequences will be normally distributed, or be
seen to correlate, in the same way, with tendencies to socially desirable or undesirable conduct. It is in this sense that I suggest that, in this molecular gaze, we may be seeing the emergence of a new way of thinking: variation without a norm and perhaps, even, anomaly without abnormality.

These, however, are not the only changes that are contributing to this waning of the logic of the norm. Across the nineteenth and twentieth centuries, ideas about normality arose out of zones gridded by government, that is to say, concerned with ‘the conduct of conduct’. But, at the start of the twenty first century, new forces are judging vitality in relation to different objectives. There are the commercial organisations such as the pharmaceutical companies whose interests are the bottom line - profit. There are professionals ranging from doctors to research scientists where humanistic aspirations are linked to mundane concerns for advancement in their careers, fame and fortune. There are insurance companies with their criteria for benefits and so forth. There are medical institutions with their decisions as to who to treat, at what ages for what conditions, under what criteria – for example, should there be ‘treatment’ for infertility, or plastic surgery for children with Down’s syndrome – decisions that are simultaneously concerned with efficacy, equity and economy. There are philanthropic bodies, such as NGOs, charities, pressure groups and campaigning organisations who now play a key role in demanding the rights to health, and to treatment for all manner of conditions and persons. And, of course, the subjects and patients themselves now play a key role in shaping our judgements as to what is ‘a suitable case for treatment’.

In this new configuration we have examinations galore, but they do not seem to operate in terms of hierarchical observation, distribution of individuals according to their qualities, and normalising judgement against institutional expectations. There are
plenty of charts of normal functioning, and graphs of distributions, but these do not seem to be standards to judge some as failing and to open them up for control. These new ways of assessing and intervening upon persons seem to operate differently. Perhaps the implications will become clearer if I turn to my second main thesis, the move from risk to susceptibility, in the context of the new molecular genetics.

From Risk to Susceptibility

In the early days of human genome project, when the term ‘geneticisation’ came into fashion amongst critics, it was often thought that the sequencing of the human genome would establish such a single ‘normal’ sequence, a composite or ‘consensus genome’. It was suggested that this sequence would serve as a norm of health against which all discrepancies would be judged as morbid abnormalities (e.g. Flower and Heath 1993). A new form of molecular surveillance was often predicted that would categorise individuals as healthy or pathological on the basis of the sequences of bases on their genome, and would divide them up and administer their lives in the light of this implacable biological truth.

But the draft sequence of the human genome published on 11th February does not produce a single ‘normal’ sequence. The first surprise was that there were far fewer genes than had been anticipated - around 31,000 sequences coding for chains of amino acids as opposed to the 100,000 that had been predicted. This seemed to mark the defeat of that simple reductionism in which pathology is written in the genes in the form of a simple mutation, with one mutated gene equalling one disease. On the publication of the draft sequence of the genome, Craig Venter of Celera Genomics, who once hoped to be the legal owner of the human genome, pronounced ‘The notion that one gene equals one disease, or that one gene produces one protein, is flying out
of the window. The post genomic project is regrouping around the genes that control
genes, or around the interactions between genes, or around functional genomics, or
around proteomics.

Perhaps as important, the sequencing seemed to show that there were millions
of loci on the genome where individuals differed from one another by as little as a
single base in the chains of As, Cs, Gs and Ts that make up 'the genetic code' – an A
is substituted by a C, for example. These variations that comprise such single
nucleotide polymorphisms, or SNPs, do not seem to function along the axis of the
normal and the pathological or the healthy and the sick. The two genomes (DNA
sequences) that each of us carry differ from one another as well as from those carried
by other individuals. Every sequence identified as a ‘gene’ now seemed to be marked
by such variation. In the human genome, to quote a recent article on
pharmacogenomics 'the normal is rare.' Or, perhaps, one might say that there is no
normal human genome - variation is the norm. This is a variation so complex and
multidimensional that it cannot easily be mapped onto the earlier 'Mendelian' ideas of
genes as units of inheritance, single entities which exist in a small number of alleles,
some of which are normal, others pathological. We seem to have a geneticisation of
variability without the reciprocal positing of a norm.

In the nineteenth century, the arguments about the inheritance of pathology all
went along the same lines - an inherited predisposition was triggered by an exciting
cause - loss of fortune, masturbation and so forth. But in the contemporary style of
biological thought, while vulnerabilities are inherited, and genome substitutes for
constitution, there is not a single inherited diathesis, or a constitution that can be
healthy or degenerate. We all carry genomic vulnerabilities to different conditions,
but neither the vulnerabilities nor the conditions are molar, defects of the person.
Instead they are small, discrete, molecular. And in the process, the vocabulary of genetic abnormality seems to mutate. There is still a logic of absolute genetic pathology. Disorders such as Huntington’s arise inescapably from a specific identified set of repetitions in the base sequences in a particular gene region. But even that fatal logic is not a logic of fatalism as (Novas and Rose 2000). In any event, such apparently implacable genetic pathologies are merely the extreme point of a rather different perception. One might call this ‘risk’, but this would be to merge it into a family of related but distinct ways of thinking. So let us term it ‘susceptibility’.

Previously the clinical assessment of risk in biomedicine was probabilistic. It might operate in terms of family history – on the basis of the pattern of illness amongst his or her relatives, an individual could then be told that they had a one in ten chance of developing a disease or giving birth to a child with a particular condition. Or it might operate by locating the individual in a risk table on the basis of measures that the epidemiology had linked to increased probabilities: age, gender, body mass index, blood pressure, lipid levels or whatever. An individual with a certain combination of these factors could then be told that his or her chance of a heart attack in the next ten years was less than 10 percent. But the new molecular gaze promises something different, a precise appraisal of one’s individual, specific susceptibilities on the basis of the sequencing of one's own DNA and the identification of a particular pattern of bases in particular locations on a specific chromosome. By the close of the twentieth century, an increasing number of medical classifications of illness were being designated with such apparent genetic precision. For example, one condition involving fronto-temporal Dementia and Parkinsonism is known as FTDP-17 because it is linked to a number of mutations in a specific region of chromosome 17. Increased susceptibility to breast cancer has been linked to the mutations known as BRCA1 and
BRCA2 on chromosome 13. Researchers claim that specific features of personality such as novelty seeking, or psychiatric disorders such as particular types of manic depression, arise from the synthesis or non-synthesis of particular proteins, and that these are controlled by polymorphisms at specific loci on particular genes. The mapping of the human genome, and the identification of increasing numbers of SNPs will merely accelerate these processes.

The genetics of alcohol dependency is now imagined in these terms. As we know, in the late nineteenth century and into the age of eugenics, alcoholism had a dual role in the inheritance of pathology. On the one hand, alcoholism was one amongst many manifestations of an inherited tainted constitution, whose passage down the generations could be visualised in a genealogical table whose blacked out squares and circles - marking the affected men and women in the lineage - showed the defective germ plasm coursing down the generations. On the other hand, alcohol abuse had a deleterious effect on the germ plasm, and so was a factor that led those with already weakened constitutions to frank breakdown. Pedigree charts of alcoholism and its links with feeble-mindedness, sexual immorality, criminality and the whole catalogue of manifestations of degeneracy were common in eugenic advice up to the outbreak of the Second World War. Such images of inherited weakness were instantiated in guidance to those contemplating marriage - who were well advised to check the family history of their prospective spouse for signs of these defects - and for those with such a history, who should think very carefully of their social duty before procreating. In the post war years, all the usual techniques were deployed to estimate heritability. Notable are the Scandinavian studies of drinking behaviour in identical and fraternal twins and adoptees, many based upon the very detailed census information and registers of alcohol abuse maintained in these countries. Most of
these studies claimed that, even when adopted, male biological children of alcoholics had a much higher risk of themselves becoming alcoholics, a risk about four times higher than biological children of non-alcoholics. From 1970s onwards, probabilistic attempts to estimate group risk were supplemented by studies that tried to identify markers - sites on specific chromosomes linked to the ‘inheritance’ of alcoholism – often based upon isolated ‘inbreeding’ populations. Blum and Noble(1990) claimed to have gone beyond the detection of markers, and to have identified a specific gene linked to alcoholism, a variant of the gene for the D2 dopamine receptor. They later argued - despite other studies failing to replicate their findings - that this was a 'reward gene' that leads to reinforcement for a wide range of compulsive behaviours.

Such 'gene for' explanations remain the stuff of popular journalism. For example, on 9 April 2001, Metro, the local London free paper headlined 'Scientists seek alcoholism gene' and went on 'A gene may be responsible for turning drinkers into alcoholics and British scientists are determined to identify it' (p. 2). But actually, behavioural genetics now takes a rather different line. Alcohol dependence is now conceived of as a matter of polygenic susceptibilities, in which a range of variations (polymorphisms) on a number of different genes contribute, in different but specific ways, to an increased susceptibility - or sometimes a decreased susceptibility - to alcohol dependency. These are not 'genes for alcoholism', but genes that control the synthesis of the proteins involved in production and transportation of neurotransmitters, receptors, enzymes, cell membranes or control of ion channels regulating neurones.

As far as addiction is concerned, the belief that there is a genetic element has not weakened, but it is now posed in terms of risk, or ‘vulnerability’. Linkage studies
from the 1980s onwards suggested that so-called ‘vulnerability genes’ might be found on Chromosomes 1, 2 and 7, and a ‘protective’ locus had been found on Chromosome 4. This protective locus was near the region of that gene that contained the alcohol dehydrogenase gene cluster, which includes genes encoding isozymes that accelerate the metabolism of ethanol to acetaldehyde. Interest was raised because this was thought to be the basis of the aversive ‘flushing reaction’ that prevents many Asians from regular or heavy drinking (Reich et al. 1998). Research goes on apace in genetically engineered mice to correlate coding sequence polymorphisms with behavioural responses to alcoholism. Hood and Buck report that variation in the GABA sub A receptor gamma 2 subunit is associated with genetic susceptibility to ethanol-induced responses such as taste aversion and motor incoordination, which may be motivationally negative in relation to alcohol.

Many who think that environment may have a role to play still retain this form of argument. Gurling and Cook (2000) agree that much genetic research has tended to ignore psychosocial issues, but point out that psychosocial research has tended to ignore biological variables. They suggest that once specific genes involved in alcoholism have been identified, new environmental factors that trigger susceptibilities will be identified. They predict that the comorbidity of alcoholism with anxiety, depression, and antisocial personality may soon be understood in the context of genetic effects from specific genetic susceptibility loci. And commenting on the implications of the publication of the map of the human genome, Nestler and Landsman (2001: 834-35) assert ‘Epidemiological studies indicate that 40-60 percent of an individual’s risk for an addiction, whether it is to alcohol, opiates or cocaine, is genetic.’ But we are not talking here about the inheritance of a single ‘gene for’ alcoholism. Rather what is at stake are the genes that regulate different aspects of the
neurotransmitter systems now thought to be involved. Thus Nestler and Landsman discuss the possible involvement of genes whose products regulate the sensitivity of a particular type of receptor, the G-protein coupled receptor, thought to be the initial target for drugs of abuse. Even with the current level of knowledge of the human genome, receptor desensitisation potentially involves at least four gene families, each family containing genes relating to a number of distinct subtypes of particular sorts of protein such as kinases, arrestins, phosducins and signalling regulators. Together, it seems, these constitute 'addiction vulnerability genes' – that is to say, susceptibilities.

This kind of argument has implications for medical practice and treatment. Enthusiasts claim that genetic screening will soon be able to reveal these individual susceptibilities by identifying these polymorphisms in particular locations on specific chromosomes. In the context of the development of cheap, automated methods of gene screening, and the likely increase of genetic screening, of prospective parents, of foetuses, of schoolchildren, of offenders, of psychiatric patients, of customers on a private basis, it is likely that awareness of one’s precise inherited susceptibilities prior to any signs of a pathology will spread from the few to the many. Genetic susceptibility will be one of Ian Hacking's looping kinds. The aspiration, of doctors and of the vulnerable or susceptible individuals themselves, will be to develop treatments that are precisely targeted at the specific abnormalities that do, or may, generate the disorder. Hence the promise of pharmacogenomics, in which medicine will be tailored to individual susceptibilities and pharmaceutical companies will add niche marketing to their immensely profitable catalogue of products for mass production.

In this sense, then, the rise of the idea of susceptibility greatly extends the ambit of disease, and the powers of the doctor to engage those who are neither
phenomenologically nor experientially ill. Such persons may indeed live healthily for years, never become ill, or become incapacitated or die in a hundred other ways. But nonetheless, as everyone has their own susceptibilities, everyone becomes, in potential at least, asymptotically ill and a suitable case for medical tutelage. Of course, this argument can certainly lead to all manner of coercive practices of control of individuals on the basis of their anomalous biology. But, before we rush too quickly to condemn geneticisation, reductionism, determinisms and the like, we need to pause. The idea of risk assessment conjures up images of certainty and calculability and hence, perhaps, a certain fatalism. But in the world of susceptibilities, biology is not destiny. The management of uncertainty is the norm. Molecular presymptomatic diagnosis gives no calculability to the ‘when’ or the ‘how’ of illness or death, nor to the manifold life decisions that must be made between the moment of diagnosis and this imagined end point.

What forms of self-government arise in this new space of uncertainty? Some suggest that individuals and their families are left in what Robert Proctor (1995: 247) has termed ‘enlightened impotence.’ Others predict fatalism, anxiety, guilt, fear, stigmatisation and discrimination without hope of remedy (e.g. Nelkin and Tancredi 1989). These possibilities exist. But advocates of behavioural genomics think the implications are different. For we are not dealing with single genes and unitary pathologies but with variations in multiple loci in multiple gene systems, each of which may itself be modulated by intra- and extra-cellular 'environmental' factors in the course of development and in the mature individual, resulting in a whole variety of different types and levels of susceptibility to particular disorders in specific environments (McGuffin, Riley and Plomin 2001). In this field of multiple possibilities, pathways, interactions and dependencies, the consequences of a genetic
and neurochemical account of mental illness are not fatalism. On the contrary, most advocates claim that the diagnosis of genetic susceptibilities will help individuals take responsibility for the management of their condition, for the control of their vulnerabilities, for the use of individually tailored behavioural and pharmacological correction of the underlying error or deficiency, thus enabling the susceptible individual to maintain themselves in their everyday life.

Of course, there remains the problem of a new and refined form of genetic discrimination, in which insurance eligibility, employment and the like, once linked at a rather gross level to family history of health and illness, will become molecularised. I have argued elsewhere that it is somewhat unlikely that the worst case scenarios of the critics will come about (Novas and Rose 2000). As all diseases and pathologies come to be seen as having a genetic basis, and all such bases come to be seen as polygenetic matters of susceptibility, the binary distinction of 'genetic' and 'non-genetic' diseases will be impossible to maintain. As we all become re-configured as bio-genetic persons, to exclude all with susceptibilities for illness from insurance or employment would, indeed, be to exclude all. The more likely outcome is not so much exclusion but modulation. This would be the requirement that, if we are to be employed, receive insurance or whatever, we continually monitor our susceptibilities in the light of all that might provoke them. And that we adjust our forms of life, activities, insurance cover, financial planning and the like in the light of them. We would be obliged to sculpt our lives in terms of our own particular genetic and biological individuality. And maybe these will soon seem no more strange or reductionist that what have become common sense attributes of our somatic existence - height, weight, body shape or tastes.
We can already see signs of the new forms of activism that are arising in this complex field. That is to say, the emergence of new ways of crafting one's life and modulation of a lifestyle in the light of one's susceptibilities. The potential sufferer is to become skilled, prudent and active, an ally of the doctor, a proto-professional, and to take their own share of the responsibility for managing their bio-genetic selves. Those who are designated at genetic risk and their families are not passive elements in the search for remedies. Such persons are increasingly demanding control over the practices linked to their own health, seeking multiple forms of expert and non-expert advice in devising their life strategies, and asking of medics that they act as the servants and not the masters of this process. Persons identified as susceptible to a particular condition have an investment in scientists fulfilling their promises and discovering the basis of, and the cure or treatment for, genetic conditions.

Contemporary biomedicine, like its predecessors, is one of the key sites for the fabrication of the contemporary self – free yet responsible, enterprising, prudent, encouraging the conduct of life in a calculative manner by acts of choice with an eye to the future and to increasing their well being. Genetic personhood is one of ‘genetic responsibility’. And genetic responsibility induces new forms of biological community, what Paul Rabinow (1996) has termed ‘biosociality’ (see also Novas and Rose 2000; Rose forthcoming). Consider, for example, the Genetic Alliance which, since the mid-1980s has sought to foster 'a dynamic coalition of consumers and professionals to promote the interests of children, adults and families living with genetic conditions' bringing together almost 300 support groups with consumers and health care professionals, 'creating partnership solutions to common concerns about access and availability of quality genetics services.' Those with similar genetic susceptibilities, or their families, gather into support groups, run websites and email
discussion lists, raise funds for medical research, engage their own medical experts, and develop and disseminate practices for crafting a life ‘at risk’ and a new ethics of the susceptible.

This shift from implacable abnormalities to manageable susceptibilities is entirely consistent with the wider reshaping in practices for the government of persons. As is well known, Gilles Deleuze (1995) has suggested that contemporary societies are no longer disciplinary, in the sense identified by Foucault – they are societies of control. Where discipline sought to fabricate individuals whose capacities and forms of conduct were indelibly and permanently inscribed into the soul – in home, school or factory – today control is continuous and integral to all activities and practices of existence. We are required to be flexible, to be in continuous training, life-long learning, perpetual assessment, continual incitement to buy, to improve oneself, constant monitoring of health and never-ending risk management. In these circuits, the active citizen must engage in a constant work of modulation, adjustment, improvement in response to the changing requirements of the practices of his or her mode of everyday life.

The ambition of these new technologies for governing the self is not merely to return the individual to a fixed norm of civilised conduct as housewife or worker, a once-off programme of normalisation. Rather, its ambition is the restoration, and continual, long-term maintenance of the free, autonomous, individual obliged to choose and to take responsibility for his or her life as if it were an outcome of acts of choice. But further, biological psychiatry seems to be offering the possibility of the calculated modification and augmentation of specific aspects of self-hood. In its new neurochemical and psycho-pharmacological guise, it is contributing to the idea of the flexible, manipulable self – manipulable not only in the service of projects of
normalisation, but manipulable by the person him or herself in the service of enhancement of capacities. These new ways of thinking about genetic and neurochemical selves are thus deeply implicated in the continual process of modulation of capacities that has become the life’s work of each active citizen.

**Desire and the Neurochemical Self**

How do these arguments relate to the general question with which I began, the pharmaceutical government of desire?

*First*, I have suggested that the government of addiction has become ‘targeted’. Alcoholics Anonymous, as Mariana Valverde (1999; see also chapter 15) argues, works on identity. It is the whole person who is pathological. 'I am Nikolas and I am an alcoholic.' Whilst in many respects we are still in the age of ‘the addict’ and ‘the alcoholic’ – in which these features are inscribed within and the truth of a whole ‘deviant personality’ – we are also beginning to see a more precise and specific form of address. This does not attempt to reshape a life or normalise a personality, but to isolate a malfunctioning process, and a related set of problematic beliefs, cognitions and life skills, and to engineer the interventions that will precisely address this very specific pathological complex with the minimum of collateral damage. The aim is to enable the individual to re-enter the circuits of everyday life where he or she will re-engage with the cybernetics of control built into education, employment, consumption, leisure and the like. Whilst many professionals still think of their clients as having damaged, defective or dangerous identities, I think we are beginning to see signs of a shift from strategies of normalisation of the deviant to measures seeking the correction of specific anomalies: interventions which work not by eliminating
autonomy, freedom and choice in the individual so affected, but by locating them in a regime requiring a constant work of adjustment.

Of course, such attempts at targeting often prove surprising. Prozac is the clearest example. On the one hand, this carefully sculpted molecule designed to target one specific condition is now offered as the treatment not merely for mild to moderate depression, but for a whole range of other disorders: anorexia, bulimia, obsessive compulsive disorder, panic disorder and much more. Indeed, now that Prozac itself has gone out of patent, Eli Lilly, the manufacturers, are marketing the same drug - fluoxetine hydrochloride - in a different packaging for Premenstrual Dysphoric disorder, a candidate diagnosis in DSM IV apparently marked by severe mood and bodily disorders around the time of menstruation and affecting 3-5 percent of menstruating women in the United States. Yet whilst this appears to run counter to the molecularisation of diagnoses and treatments, or, perhaps, to question the project of DSM IV for the finer and finer distinction of types of disorder, other even newer drugs claim to target particular receptors even more precisely - the 5HT2 receptor, for example - thus once more claiming to re-divide psychiatric disorders in molecular terms. And, at the same time, one molecule and one target seems insufficient. In relation to depression for instance, we now have a range of third generation products that act on serotonin and norepinephrine, or serotonin and dopamine. Biovalue, here, seems to demand constant innovation, and the cycling from the specific targeted cure - the magic bullet - to the wonder drug that will cure all seems endemic to marketing and perhaps to the very project of commercial psychopharmacology itself. Yet, despite this cycling, in any of these phases, it is not the pathological individual that is targeted but a molecular anomaly.
Second, I have argued that addiction itself is being rewritten in terms of the susceptible neurochemical and genetic self. Having resisted medicalisation for so long, addiction becomes, at least in part, not a disease of the person, or of the will, but a disease of the brain. Of course, as Mariana Valverde has shown, this short-circuiting of the will is only partial. For in the new 'flattened' self, in which the organs and bodily processes are in ever closer proximity to thought, emotion, cognition and action, a role still remains for techniques for the reconstruction of the will. Cognitive and behavioural therapies have a new role, to equip the vulnerable individual with the insight, the skills and the self-mastering capacities required to live a life in the knowledge of his or her susceptibilities, and to take responsibility for avoiding the actions, settings or relations that might provoke them.

Finally, I would like to return to biovalue, and its relation to this work of modulation of the somatic self. The best selling drugs these days are not those that treat acute illnesses, but those that are prescribed chronically - Premarin, Lipitor, Omeprazole, Prozac, Viagra. The power to reshape life by drugs seems to extend way beyond what we previously understood as illness. Biomedicine has already rewritten the norms of reproduction, its timetables and its kinship relations. Hormone replacement treatment is already rewriting the norms of female ageing. Drugs for ‘panic disorder’ such as Alazopram are rewriting the norms of social interaction. So the capitalisation of the power to treat intensifies the redefinition of that which is amenable to correction or modification. This is not simply blurring the borders between normality and pathology, or widening the net of pathology. We are seeing an enhancement in our capacities to adjust and readjust our somatic existence according to the exigencies of the life to which we aspire. These capacities for enhancement involve the co-production of a drug, the condition it will treat, and the desire and
demand for it. Where Foucault analysed biopolitics, we now must analyse bioeconomics, for human capital is now to be understood in a rather literal sense, in terms of the new linkages between the politics and economics of life itself.
Notes

1 This paper was presented at the Conference on Risk and Morality held at the University of British Columbia in May 2001. It was conceived as a companion piece to that given in the same session by Mariana Valverde and is indebted to her comments and suggestions. I have reframed it here to stand on its own and I am to blame for the mistakes. An earlier, rather different, version of this paper was presented as ‘Normality and Pathology in a Biological Age’, given as a public lecture in the Faculty of Humanities, University of Copenhagen, 9 March 2001.

2 I discuss the 'opening up' of this psychological space in Rose (1985).

3 Actually, as Leonard (1992) points out, there is considerable controversy over these speculations. The benzodiazapine complex shows these 'plasticity effects' in animal brains, but there is little evidence of changes in human brains in post-mortem studies, and the experimental changes, in any event, require much higher doses than those that produce functional tolerance when used clinically. What is important for our purposes is that while there is dispute over the mechanisms and specifics, the thought style remains unchallenged, and defines the form of possible explanations and of criticisms of those explanations.

4 http://www.niaaa.nih.gov/press/1999/discover-text.htm. The study is J. Lewohl, et al. 1999. " G-protein-coupled inwardly rectifying potassium channels are targets of alcohol action ", Nature Neuroscience, 2 (12): 1084-1090. Lewohl and her associates identified the effect of ethanol on the G-protein-coupled inwardly rectifying potassium channel (GIRK). GIRKs are widely distributed in the brain and play a major role in regulating inhibitory responses in the central nervous system and GIRK channels were shown to be sensitive to ethanol at a concentration below the legal
level for intoxication (10mM). They suggested that this high sensitivity to relatively minor changes may influence substantially the capability of neurons to communicate.

5 This is one focus of my 1985 study, The Psychological Complex (London: Routledge, especially Chapters 3-6): ‘Individual psychology derives its conception of its object from the statistical normativity of the population. The norms which it proposes are not those of life but those of large numbers. The possibility of a knowledge of the individual, for the psychology of the individual, is not provided through a conception of the psyche, its processes, its homeostatic mechanisms, the laws of its development and the abnormalities to which they can give rise. It is founded upon a metaphysic of the quantification of qualities and the laws of variation in populations…. It was founded through the identification of norm in the register of the statistical with norm in the register of the social. The operation which made individual psychology possible was the identification of statistical norms of variation with social norms of expectation. The abnormality which was so crucial in the founding of a medical notion of bodily norms was a disturbance in its object, the body itself. But the abnormality around which individual psychology was organised was not an abnormality of a life process, or one specifiable in terms of ease and dis-ease. It was an abnormality in terms of a norm of functioning specified by particular social apparatuses. The unease which enabled the normativity of individual psychology to be established was constituted by the objectives of government rather than the vicissitudes of the psyche. It was the school, the courts, the police and the army which provided the psychology of the individual with those whom it would have to be able to construe as abnormal’ (p.229).

7 The ‘public’ genome identifies some 1.42 million single nucleotide polymorphisms. Craig Venter’s group identify 2.1 million SNPs, very few of which, less than 1 percent, seem to result in variations of the proteins coded.

8 ‘In his 1997 book, Pharmacogenetics, Wendell W. Weber quotes from Somerset Maugham's account of his experiences as a young medical student. ..."I have always worked from the living model. I remember that once in the dissecting room when I was going over my 'part' with the demonstrator, he asked me what some nerve was and I did not know. He told me; whereupon I remonstrated, for it was in the wrong place. Nevertheless he insisted that it was the nerve I had been looking in vain for. I complained of the abnormality and he, smiling, said that in anatomy it was the normal that was uncommon. I was annoyed at the time, but the remark sank into my mind and since then it has become forced upon me that it was true of man as well as anatomy. The normal is what you find but rarely. The normal is the ideal. It is a picture that one fabricates of the average characteristics of men, and to find them all in a single man is hardly to be expected." Maugham's observation -that the normal is rare -is at the heart of the challenge and promise of pharmacogenomics' (Wendell W. Weber (1997) Pharmacogenetics, Oxford: Oxford University Press, quoted in Norton, 2001: 180).

Thanks to Oonagh Corrigan for this quote.

9 For a discussion of this theme in Nazi eugenics, see Proctor (1988: 239-40).

10 See the review of these studies in (Grove and Cadoret 1983).

11 These claims and the contrary findings, are reviewed in Holden (1994).
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