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The rise of neurogenetic determinism

Steven Rose

Neurogenetic determinism claims to explain everything - from violence in the streets to sexual orientation - in terms of properties of the brain or genes. These claims draw on the explosion of new genetic and neuroscientific techniques, but reflect a much older reductionist fallacy. This article analyses a sequence of falsely taken reductive steps and examines their consequences for both biological and social thinking.

We are now halfway through what in the US has been called the Decade of the Brain. Europe, always slower to move on such matters, has just belatedly started its own such Decade. And we are even further into the massive international $3 billion-odd exercise known as the Human Genome Programme, the attempt to map, and subsequently to sequence, the entire DNA alphabet of the human chromosomes. (Identifying just what these DNA strands might do, what the genes might mean, is, as will become clear, a rather different matter, though often elided in popular consciousness.) For a neuroscientist like myself, it is an incredibly exciting time to be in the lab, at the computer or in the library. New results come flooding

in at an almost impossible rate to digest. From the gene sequences of molecular biology to the windows onto the brain produced by the new imaging techniques, extraordinary pictures of complexity at all levels, from the chemical through the cellular to the systemic are emerging; of the brain as a dynamic entity, an ever-shifting sea of electrical and magnetic fluxes, of chemical currents, of growing and retracting cellular connections, of coherent time-locked oscillations which some have even speculated form the basis of conscious experience. But data has far outstripped theory; rival schools, of connectionists (who claim that brain/mind properties can be simulated in the distributed architecture of parallel processing computers) and chaos theorists (who deny a permanent 'seat' to any mind/brain process) strive to make sense of an information overload that almost inhibits meaning.

'New results come flooding in at an almost impossible rate to digest'

Just as the Decade of the Brain has produced dramatic advances in information, so it has also generated ever more strident claims that neuroscience is about to 'solve' the brain and in doing so usher in a new era of what Delgado, an earlier enthusiast for brain surgery to cure violence, once called a 'psychocivilised society'. The emerging synthesis which I call neurogenetics offers the prospect of identifying, ascribing causal power to, and if appropriate modifying, genes affecting brain and behaviour. Neurogenetics claims to be able to answer the question of where, in a world full of individual pain and social disorder, we should look to explain and to change our condition. It is these claims, rather than the excitements of brain theory for its practitioners, which I wish to address here. If the reasons for our distress lie outside ourselves, it is for the social sciences to understand and for politics to try to resolve the problems. If however the causes of our pleasures and our pains, our virtues and our vices, lie predominantly within the biological realm, then it is to neurogenetics that we should look for explanation, and to pharmacology and molecular engineering that we should turn for solutions. Social and biological explanations are not necessarily incompatible, but at any time the emphasis given to each seems to depend less on the state of'objective' scientific knowledge than on the sociopolitical Zeitgeist. In the context of rising public concern about levels of violence, an ideology which stresses personal responsibility, and denies even the correlation between poverty and ill-health, is likely to reject the social in favour of the individual and his or her biological constitution.

Of course this is to simplify, to imply that the world is divided into mutually

incommensurable realms of causation in which problems are *either* social *or* biological. This is not my intention; the phenomena of human existence and experience are always and inexorably simultaneously biological *and* social, and an adequate explanation must involve both. Even this may not be enough; both social and biological sciences deal with the world observed as object; personal experience is by definition subjective and any account which excludes this personal element from our attempts to understand the world falls into the reductive mechanical materialist trap against which both Marx and Engels inveighed. But such unity of subjective and objective may be even harder to achieve than that of the biological and social, and I cannot even begin to approach that issue here. (I tried to talk about it, however inadequately, in my recent book The *Making of* Memory.[1])

Let us then remain in the world of the objective. Clearly, for any serious scientist to deny the social in favour of the biological or vice versa would be unthinkable; we are all interactionists now. However, it is by their deeds that one must judge, and in any search for explanation and intervention it is necessary to seek the appropriate level which effectively determines outcomes. Whilst only the most extreme reductionist would suggest that we should seek the origins of the Bosnian war in deficiencies in serotonin reuptake mechanisms in Dr Karadzic's brain, and its cure by the mass prescription of Prozac, many of the arguments offered by neurogenetic determinists are not far removed from such extremes. Give the social its due, the claim runs, but in the last analysis the determinants are surely biological; and anyhow, we have some understanding and possibility of intervention into the biological, but rather little into the social.

This is not a new debate; it has recurred in each generation at least since Darwin's day, and most recently in the 1970s and 80s in the form of the polemical disputes over the explanatory powers of sociobiology.[2] What is new is the way in which the mystique of the new genetics is seen as strengthening the reductionist argument. At its simplest, neurogenetic determinism argues a directly causal relationship between gene and behaviour. A man is homosexual because he has a 'gay brain' itself the product of 'gay genes' and a woman is depressed because she

1. *The Making of Memory*, Bantam, London 1992.
2. See for example, E.O. Wilson, *Sociobiology: the New Synthesis*, Harvard University Press, Cambridge Mass.1975; S.PR. Rose, R.C. Lewontin and L.J. Kamin, *Not m our Genes*, Penguin, Harmondsworth 1984; R Kitcher, Vaulting Ambition: *Sociobiology and the Quest for Human Nature*, MIT Press, Cambridge, Mass. 1985.

has genes 'for' depression.[3] There is violence on the streets because people have 'violent' or 'criminal' genes;[4] people get drunk because they have genes 'for' alcoholism;[5] and there may be genes 'for' homelessness, according to the then Editor of the leading US journal *Science.* (The other day I even came across a claim that there might be genes for 'compulsive shopping'; there are clearly no limits to the power of the alphabet soup of DNA.) What isn't due to the genes may be left to biological insults occurring during pregnancy, birth defects or early childhood accident.

In a social and political environment conducive to such claims, and which has largely despaired of finding social solutions to social problems, these apparently scientific assertions become magnified by press and politicians, and researchers may argue that their more modest claims are traduced beyond their intentions. Such Pilatism, however, is hard to credit when so much effort is put by researchers themselves into what Nelkin has described as 'selling science'.[6] The press releases, put out by the researchers themselves, which surrounded the publication of leVay's and Hamer's books and papers (see footnote 3), claiming to have identified 'the' biological cause of male homosexuality, and raising a host of alarmist social and ethical speculations were couched in language which leaves little need for media magnification.

Reductionism

It is my argument that such naive neurogenetic determinism is based on a faulty reductive sequence by which complex social processes are regarded as 'caused' by, 'explained by' or 'nothing but' the workings out of biological programmes based in the brain or the genes. This reductive sequence runs through a number of steps which I shall outline later (see pp 65-9); the core issue, however, is reducibility, which, as Medawar once remarked, comes not as second but as first nature to natural scientists.

Thus when Popper, giving the first Medawar lecture to the Royal Society a few

3. See S. Levay, *The Sexual Brain* , MIT Press, Cambridge, Mass., 1993; D. Hamer, S. Hu, V.L. Magnuson, N. Hu and A.M.L. Pattatucci, *Science,* 261, 1993, pp 321-327; D.B. Cohen, Out *of the Blue: Depression and Human Nature,* Norton, New York, 1994-
4. A. Reiss and J. Roth, *Understanding and Preventing Violence,* National Academy Press, Washington DC 1993.
5. M. Galanter (ed) *Recent Developments in Alcoholism,* Plenum, New York 1994-
6. D. Nelkin, *Selling Science,* Freeman, New York 1997.

years back, offered eight terse reasons why biology was irreducible to physics (of which the fourth was that 'biochemistry cannot be reduced to chemistry'), he incurred the wrath of the distinguished assembly, moving the Nobel-Prize winning crystallographer Max Perutz into a vigorous response. His life's work, after all, had been to demonstrate the relevance of chemistry to biology, and a couple of weeks later he published a reply to Popper, basing his case on the way in which the molecular structure of haemoglobins varied amongst species depending on their environment. Contrast, for instance, the haemoglobin of a mammal which lives at relatively low altitudes, like a camel, with that of a related species, the llama, which lives at high altitude in the Andes, where the air is much thinner and the demands on the oxygen-carrying capacity of the blood therefore differ. The structures are subtly different, in each case better fitting the conditions which its owners inhabit. Is this not clear evidence that human physiology and biochemistry not merely depend upon, but are reducible to, the chemistry of their component molecules? Game set and match to Perutz?[7] I think not, but can not here argue that case in more depth. Suffice it to say that no amount of analysis, however detailed, of the molecular structure of haemoglobin can lead to an understanding of the function of that molecule as an oxygen carrier in a living animal, in other words its *meaning* for the system of which it is a part.

This is not of course at all to deny the power of reductionist analysis as part of our attempt to understand complex systems, nor does it reflect on reductionism as a methodology by which to experiment - there is essentially no other way to work. And it says nothing about abstract philosophical concerns with theory reduction. I am concerned here simply with the efforts to attribute causal explanation of complex social affairs through appeals to neurotransmitter metabolism, brain structures and genes. It is not necessary to enter into a full-blown defence of irreducibility to identify the flaws in the claims of neurogenetic determinism to explain complex social phenomena. Such phenomena are of their essence historically contingent and framed by meanings which the reductive process loses as surely as the information content of the page on which these words are printed is lost from a chemical study of the paper and ink which comprise it. And the issue at stake is not the formal philosophical one, but the question of the appropriate level of organisation of matter at which to seek causally effective

7. M. Perutz, Trends *Biochem. So.,* 13, 1988.

determinants of the behaviour of individuals and societies.

The US Violence Initiative

Let me take a specific example of considerable current concern: the explanation and treatment of the wave of violence which seems to be spreading through the societies of the industrialised world. The debate about the causes of violence long predates the current furore; only the language in which it is cloaked changes. Two decades ago the focus was not genes but chromosomes, when it was claimed that there was a higher than expected prevalence of men carrying an extra Y chromosome amongst those incarcerated for violent crime. And at the turn of the twentieth century, for the followers of Lombroso, it was physiognomy rather than genes which predicted criminality. Before the time of modern science, it was simpler still; it was sufficient to invoke original sin, or predestination. Even if the extra Y has now gone the way of physiognomy and sin, predestination (albeit now spoken in a medicalised hush rather than a hell-fire rant) still lies at the heart of the argument.

Hitherto, in Britain at least, the focus of explanation has been on personal life history; the impoverished rearing practices of single mothers or the laxly disciplined schooling of the 1960s. But in the US even this explanation is being discarded in favour of a return to original biological sin; the fault, we are told, lies in our (or rather *their)* genes. The argument was put most clearly in 1992 by the then Director of the National Institutes for Mental Health, Frederick Goodwin, in his proposed Federal Violence Initiative. Noting that violence was concentrated in the US inner cities, and especially amongst blacks, who have, he argued, inherited a cocktail of genetic predispositions, to diabetes, to high blood pressure, and to violent crime, he argued for a research programme to identify some 100,000 inner-city children and to investigate the genetic or congenital factors which predispose them to such violent and antisocial behaviour. A few years previously, the psychologist Richard Hermstein coauthored with James Q. Wilson *Crime and Human Nature* - in many ways the forerunner of Herrnstein's more recent coauthorship of *The Bell Curve* - which equally focused on the proposition that violent crime in the US is the prerogative of the poor and black and that its origins lie in 'failures' in their biological constitution.

Now there are many obvious objections to such a proposal. Some point to the fact that these discussions always seem to focus on working-class crime; no-one

seems to study the heritability of the tendency to commit business fraud, or the biochemical correlates of wife-beating amongst middle-class men. Others worry about the complex and sometimes contradictory web of meanings involved in the very concept of violence. On the one hand an *identical* act - of a man picking up a gun and shooting another at close range - if sanctioned by the state in times of war becomes an act of heroism worthy of a medal, but if carried out in the midst of a drugs deal in a Manchester pub is a crime punishable by a long term of imprisonment. On the other hand, all sorts *of different* acts are lumped together: Cantona's attack on an abusive football fan, fights between demonstrators and police, the Russian bombing of Grozny, all merge, as if one word, violence, fits them all, and their underlying causes are all the same.

'Predestination, through original biological sin still lies at the heart of the argument'

Goodwin's proposal led to charges of racism, and he has subsequently left the NIMH, but a modified version of his proposed Initiative, targeted on specific inner-city areas such as Chicago, is up and running, and estimated to cost some $400 million. A conference based on Goodwin's premises, blocked in the US, was held under the prestigious auspices of the CIBA Foundation in London in January of 1995, and the postponed conference in the US eventually got under way - in the face of pickets alleging racism - in September 1995. Not surprisingly, psychologists and psychiatrists, geneticists and molecular biologists have looked longingly at this particular pork barrel. In 1994 I was telephoned urgently by a well-known California-based therapist, just off to Washington to present a proposal to study biochemical and immunological 'markers' in 'violent, incarcerated criminals'. Would I collaborate with him, he asked, in analysing serotonin levels in fluids derived from spinal taps/ Serotonin is a neurotransmitter whose metabolism is affected by a number of well-known drugs including, as it happens, the now notorious Prozac. To say nothing of the ethics of performing this type of operation on a - literally - captive population, the thought that such a study might provide a causal explanation for the endemic violence of US society is just the sort of simple-mindedness that the Violence Initiative fosters.

Amongst child psychologists the key word has become 'temperament'. This nebulous property is, they claim, to a significant degree heritable. Jerome Kagan suggests that some 10 per cent of the infants he studied show, from a very early age, a tendency to shyness which in later life expresses itself as aggression. To bolster

this deterministic argument, he reports that he finds an analogous pattern of behaviour in kittens which grow into aggressive cats. Adrian Raine and his colleagues have studied a cohort of Danish males, now in their mid 20s, and shown that children born with birth complications, those who were products of an unwanted pregnancy and failed abortion, and those institutionalised during the first year of life, committed a disproportionate number of violent crimes (murder, rape, armed robbery); they concluded that 'biological factors play some role in violent behaviour - and the role is not trivial.'[8] That children with such a desperate history become damaged and even criminal adults is an observation which would scarcely surprise even the most socially deterministic criminologist; the inclusion criteria for his sample are likely to cluster with many other impoverished aspects of the growing child's life history. Most, however, would probably regard Raine's conclusion as a leap of faith justified only by a commitment to biologistic thinking.

'Is there something unique about the genotype of the US population which dramatically predisposes it to violence?'

No biologist could doubt the premise that individual differences in genes, and during development, help shape a person's actions and distinguish how one person behaves in a given context from how another behaves; nor that a study of the mechanisms involved in these developmental processes is of great scientific interest. But that is neither the reason why nor the way in which 'violence research' is currently being conducted. Rather, it is framed within a determinist paradigm which seeks the causes of social problems in individual biology, and it is fostered by a political philosophy - on both sides of the Atlantic - which rejoices in the privileges which come with inequalities in wealth and power and rejects steps to diminish them. The rate of violent crime and of incarceration is higher in the US than in any other industrial country. Can it really be the case that there is something unique about the genotype of the US population which so dramatically predisposes it to violence.[7] Furthermore, rates of violence are not static; in both the US and the UK, violent crime has markedly increased in recent years - in the US the death rate amongst young males increased 154 per cent between 1985 and 1994. Such fluctuations between and within societies are quite incompatible with any genetic explanation.

8. A. Raine, R Brennan and S. Mednick, mimeo paper at AAAS annual meeting, San Francisco, 1993.

The reductionist cascade

What this account demonstrates is the first two steps in a reductive cascade which characterises all such determinist thinking: *reification* and *arbitrary agglomeration.* In this section I will outline a series of reductionist assumptions which constitute the bases for determinist thinking. Alongside *reification* and *arbitrary agglomeration,* I will also describe: *improper quantification,* belief in *statistical normality, spurious localisation,* and *misplaced causality.*

Reification converts a dynamic process into a static phenomenon. Thus violence, rather than describing an action/activity between persons, or even a person and the natural world, becomes instead a 'character' - aggression - a thing which can be abstracted from the dynamically interactive system in which it appears and which can be studied in isolation. The same process occurs with 'intelligence', 'altruism', 'homosexuality', etc. Yet if the activity described by the term violence can only be expressed in an interaction between individuals, then to reify the process is to lose its meaning.

'Comparably crude generalisations in my study of memory in day-old chicks would be rejected out of hand'

Arbitrary agglomeration carries reification a step further, lumping together many different reified interactions as all exemplars of the one thing. Thus aggression becomes a portmanteau term within which all the many types of event and process catalogued above can be linked; all become manifestations of some unitary underlying property of the individuals, so that identical biological mechanisms are involved in, or even cause, each. Take, for example, the descriptions offered in a recent widely cited paper by Hans Brunner and his colleagues, associating a point mutation in the gene which codes for a particular brain enzyme concerned with neurotransmission with 'abnormal behaviour.'[9] The 'behavioural phenotype' in eight males in this family is described as including 'aggressive outbursts, arson, attempted rape and exhibitionism', activities carried out by subjects living in different parts of the country at different times' across three generations. Can such widely differing types of behaviour, described so baldly as to isolate them from social context, appropriately be subsumed under the single heading of aggression? It is unlikely that such an assertion, if made in the context of a study of non-human animal behaviour, would pass muster

9. H.J. Brunner, M. Nelen, X.O. Breakefield, H.H. Ropers, and B.A. van Oost, Science, 262, 1993.

certainly if I made comparably crude generalisations on the basis of such sparse data in my study of memory in day-old chicks, the paper would rightly be rejected out of hand. Yet the claims the Brunner paper makes have become part of the arsenal of argument employed, for example, by the Federal Violence Initiative.

A third error in the use of this form of argumentation is *improper quantification.* Improper quantification argues that reified and agglomerated characters can be given numerical value. If a person is violent, or intelligent, one can ask how violent, how intelligent, by comparison with other people. IQ is one well-known example, but the quantification of aggression is also revealing, for it illustrates another feature of the reductionist cascade which leads to neurogenetic determinism, the use of an animal model. Place an unfamiliar mouse into a cage occupied by a rat, and often the rat will eventually kill the mouse. The time taken for the rat to perform this act is taken as a surrogate for the rat's aggression; some rats will kill quickly, others slowly or even not at all. The rat which kills in thirty seconds is on this scale twice as aggressive as the rat which takes a minute. Such a measure, dignified as *muricidal behaviour,* serves as a quantitative index for the study of aggression, ignoring the many other aspects of the rat-mouse interaction, for instance the dimensions, shape and degree of familiarity of the cage environment to the participants in the 'muricidal' interaction, whether there are opportunities for retreat or escape, and the prior history of interactions between the pair. And just as time to kill becomes a surrogate for a measure of aggression, so this behaviour in the rat is transmogrified into drive-by gangs shooting up a district in Los Angeles. Genes which affect the muricidal interaction are claimed to have their homologues in the human, and therefore to be explanatory factors here too.

Of course, in an entirely trivial sense this could be true. A genetic defect which leads to blindness in rats may have its homologue in humans, and blind humans are, one would assume, less likely to pick up a gun and fire than sighted ones. But this is not what the determinists mean when they make their causal claims for a specific genetic origin for violence.

Belief in *statistical normality* assumes that in any given population the distribution of such behavioural scores takes a Gaussian form, the bell-shaped curve. The best known example is IQ, the tests for which successive generations of psychometricians refined and remoulded until it was made to fit (almost) the

approved statistical distribution - a manipulation exploited to the hilt in *The Bell Curve*.[10] But to assume that the entire population can be distributed along a single dimension to which a single numerical value can be ascribed is to confuse a statistical manipulation for a biological phenomenon. There is no biological necessity for such a unidimensional distribution (even for continuously varying genetic traits), nor for one in which the population shows such a convenient spread. (It is perfectly possible to set examinations in which virtually everyone scores 100 per cent; the British university penchant for 10 per cent firsts, 10 per cent thirds and 10 per cent fails, with everyone else comfortably in the middle, is a convention, not a law of nature.) Yet the power of this reified statistic should not be underestimated. It conveniently conflates two different concepts of 'normality', implying that to lie outside the permitted range around the norm is to be in some way abnormal, not merely statistically but in the sense which ascribes normative values. Thus homosexuality is abnormal in that only a small percentage of the population are gay or lesbian, and it has been, at least until recently, normatively unacceptable both legally and religiously. When Herrnstein and Murray called their book *The Bell Curve* they played precisely into these multiple meanings of reified normality.

Having reified processes into things and arbitrarily quantified them, the reified object ceases to be a property even of the individual, but instead becomes that of a part of the person - this is *spurious localisation*. Hence the penchant for speaking of, for example, schizophrenic brains, genes - or even urine - rather than of brains, genes or urine derived from a person diagnosed as suffering from schizophrenia. Of course, everyone ought to know (and does, at least on Sundays) that this is a shorthand, but the resonance of 'gay brains' or 'gay genes' does more than merely sell books for their scientific authors; it both reflects and endorses the modes of thought and explanation that constitute neurogenetic determinism, for it disarticulates the complex properties of individuals into isolated and localised lumps of biology, permitting neuroanatomical debates to range over whether gayness is embedded in one or other hypothalamic region or, alternatively, a differently shaped corpus callosum in the brain. Aggression is similarly 'located' in the limbic system, probably the amygdala. In the 1970s US psychosurgeons proposed to treat inner-city violence by amygdalectomising ghetto

10. R.J. Herrnstein, and C. Murray, *The Bell Curve-Intelligence and Class Structure in American* Li/e,The Free Press, 1994.

militants." Things are a little more sophisticated today; a localisation in the brain can also take the form of some chemical imbalance, probably of neurotransmitters, so aggression is now 'caused' by a disorder of serotonin reuptake mechanisms, and drugs rather than the knife become the approved approach. Raine claims to be able to detect reductions in the neural activity of the frontal cortex in 'murderers' as opposed to 'normal' individuals by means of brain scans, and hence to be able to predict 'with 80% certainty' from this biological measure the likelihood of a person being a violent killer. It is not clear what such a measure might show in the brain of a Saddam Hussein, a Ratko Mladic or a Stonnin' Norman. Presumably these would feature amongst Dr Raine's 'normals'.

It is at this point that neurogenetic determinism introduces its *misplaced sense of causality*. It is of course probable that during aggressive encounters people show dramatic changes in, for instance, hormones, neurotransmitters and neuro-physiological responses, all of which can be affected by drug treatments. People whose life history includes many such encounters are likely to show lasting differences in a variety of brain and body markers. But to describe such changes as if they were the *causes* of particular behaviours is to mistake correlation or even consequence for cause. This issue has dogged interpretation of the biochemical and brain correlates of psychiatric disorders for decades, yet it still continues. When one has a cold, one's nose runs. But the nasal mucus is a consequence and not a cause of the infection. When one has a toothache it may be sensible to alleviate the pain by taking aspirin, but the cause of the toothache is not too little aspirin in the brain. Such fallacies are however an almost inevitable consequence of the processes of reification and agglomeration, for if there is one single thing called, for instance, alcoholism, then it becomes appropriate to seek a single causative agent; complexity is hard to deal with within the neurogenetic agenda.

'The cause of toothache is not too little aspirin in the brain'

Consequences of determinism

A number of single gene defects are known to lead to drastic dysfunctions of mind and brain. Huntington's disease, with its seemingly inevitable progress towards neurological collapse in middle age, is the classic example, but there are of course

11. V.H. Mark, and F.R. Ervin, *Violence and the Brain,* Harper and Row, London and New York, 1970.

many others; Lesch-Nyhan syndrome, Tay-Sachs disease; the list of rare but devastating conditions is long... But neurogenetics consistently overstates its case, moving seamlessly from single to many genes, from genes with predictable consequences in virtually all known environments to genes with small or highly variable effects, whose norm of reaction extends so far as to prevent any claims to predictability. In only a few per cent of all Alzheimers cases is there a clear genetic involvement; while evidence identifying gene markers for manic depression and schizophrenia have been advanced with extensive publicity, and then quietly withdrawn. At best, the hunt for the genes 'for' these conditions may be able to identify anomalous cases in which the genetic effect is to mimic a more widespread phenotypic condition (geneticists speak of phenocopies to emphasise the primacy of their genetic explanations in such cases; I have proposed the term 'genocopy' to help geneticists appreciate their more limited contribution). What both concepts emphasise is the extent to which multiple pathways may lead to a final common biochemical or behavioural endpoint. What both mask is the possibility that the endpoints may not be in every sense identical. Some diagnosed depressions are ameliorated by one type of drug, some by another, and these distinctions have even been made the basis of diagnosis, in which the pharmacological response rather than the clinical syndrome is made the basis for defining the disorder from which the individual suffers - once again insisting on the primacy of the biochemical over the behavioural, the biological over the social.

There are four main negative consequences of such determinism. The first is limited to the study of biology itself: the damage it does to conceptualising living processes.The primacy given to genetic causes fosters, even amongst researchers whose day-to-day practice ought to convince them otherwise, a linear view of living processes, in which the key to life itself lies in the one-dimensional string of nucleotide bases of DNA, the mythopoeic genome. Witness the metaphors with which molecular geneticists speak of the goals of the Human Genome Project - the 'Holy Grail', the 'Code of Codes', 'the Book of Life', 'genes for' particular conditions, as if the entire four dimensions of an organism - three of space and one of developmental and life history - can be read off from this linear code, like a telephone directory.

Few popular writers are more guilty of this than Richard Dawkins. From his first book The *Selfish* Gene to the most recent, *River out of Eden*, he has shown a rhetorical gift for making plausible the gene's eye view not merely of the individual

but of the entire living world. Of course genes aren't selfish - this is a term which can only sensibly be applied to an organism, not a part thereof, but, whereas in myl970s book *The Conscious Brain,* the elision in the title was a deliberate act of paradox, Dawkins seems really to believe that this is the way the world really is. Practising evolutionary and population geneticists and molecular biologists recognise that there are multiple mechanisms at play in the processes whereby species evolve or go extinct, and by which new species are formed. They include, of course, classical natural selection and possibly sexual selection, but they also may include genetic drift, molecular drive, founder effects and many others. But it remains the case that, for Dawkins as for Darwin, explaining how natural selection *per se* generates new species is a great deal harder than explaining how it succeeds in enabling already existing species to evolve so as to get better at doing their own specific things. Furthermore, the selective processes on which Darwinian evolution depends operate at many levels, that of the gene, the genome, the organism, even the population. For Dawkins only the gene, the primary DNA 'replicator', counts. It is a version of what in the 1930s was called 'bean bag genetics' in which each gene is seen as an individual discrete unit; the history of population genetics since then, in the hands of Sewall Wright, Dobzhansky and others, has been to transcend such simplicities, but the popularising schemata of Dawkins cannot deal with complexity. This is why he continually reduces organisms to genes and genes to text - to *information.* 'Life', he writes, ' is just bytes and bytes and bytes of digital information.'[12] He may think that he is nothing but a rather primitively designed PC, but I doubt that the rest of us see ourselves that way. 'Information' is not what life is about; it is about 'meaning', and the one is not even formally reducible to the other.

To clarify what I am getting at, consider the phenomenon that I study myself, memory. Memories are believed to be encoded in the brain in some manner based on the establishment of connections between nerve cells such as to create potential novel circuits - a little crude as a description but it will do for the present. Computer modellers from amongst the Artificial Intelligentsia have had a field day producing wiring diagrams of how such circuits could be created, and an entire theoretical universe has grown up over the past decade, called connectionism, which claims to be able to model memories this

12. R. Dawkins, River *out of Eden,* Weidenfeld and Nicolson, London 1995, p 19.

way. At a recent workshop on memory, an enthusiastic Oxford-based modeller claimed to be able to calculate that in primates a particular brain structure, the hippocampus, could encode precisely 36,000 memories. For him, a memory was synonymous with a particular bit of information. How many bits of information do I need to remember the peculiar sadness of the long September shadows, the colour of my son's hair, or even what I had for last night's dinner? These are simply not calculable in terms of information theory. Yet they are central to life - and I suspect they are for the Oxford populariser Dawkins as much as the Oxford connectionist. Neither they, nor my personal and intellectual engagement with them, are simply bytes and bytes and bytes.

What *do* genes do?

So what should one be talking about when one discusses genes? The shorthand phrase of a gene 'for' a condition is profoundly misleading - after all there aren't really even genes 'for' blue or brown eyes, let alone such complex and historically and socially shaped features of human existence as sexual desire or urban guerillas. The cellular developmental and enzymatic route which results in the manufacture of particular pigments involves many thousand genes; the route which leads to the behavioural manifestations we call desire clearly involves genes, but cannot sensibly be regarded as embodied in them. What there are, of course, are differences between genomes (that is, the entire ensemble of genes that any organism possesses). Thus in any particular genome, the absence of a particular gene may result in the emergence of differences in eye colour. The biologist looking at the effects of particular genetic mutations or deletions studies the functioning of the system in the absence or malfunction of a particular gene. Furthermore, the system is not a passive responder to absence or malfunction, but seeks, by means of developmental plasticity, to compensate for any deficit.

A considerable disservice was done to biology by the historical chance which meant that in the early years of this century two separate sub-disciplines emerged; genetics, which essentially asks questions about the origins of differences between organisms, and developmental biology, which asks questions about the processes which ensure similarity. The careless language of DNA and molecular genetics serves to widen this gap rather than help bridge it so as to open the route towards the synthetic biology that we so badly need. As is well known, chimpanzees and humans share upwards of 98 per cent of their DNA,

yet no-one would confuse the two phenotypes. We have no idea at present about the developmental rules which lead in one case to the chimp, in the other to the human, but this, surely one of the great unsolved riddles of biology, seems a matter of indifference to most molecularly oriented geneticists.

The other consequences of determinism reach out beyond theory. If the homeless or depressed are so because of a flaw in their biology, their condition cannot be the fault of society, even if a humane society will attempt, pharmacologically or otherwise, to alleviate their distress. This victim-blaming in its turn generates a sort of fatalism amongst those it stigmatises: it is not our fault, the problem lies in our biology. Such fatalism can bring its own relief, for less stigma attaches to being the carrier or transmitter of deficient genes than to having been morally responsible. It is striking that in the US, leading gay activists have embraced the gay brains/gay genes explanation for their sexual orientation on the explicit grounds that they can no longer be held morally culpable for a 'natural' state, nor can they be seen as dangerously likely to infect others with their 'perverse' tastes.

The final consequence is the subversion of scarce resources. Funding for research and treatment becomes misfocused. The orientation of research funds in Russia towards the molecular biology and genetics of alcoholism is one good example; no rational attempt to explain the prevalence of vodka-sodden drunks on the streets of Moscow would seize on the peculiar genetics of the Russian population as its starting point. Similarly the Violence Initiative - directed towards seeking the origins of violence in American society in terms of the genotypes of blacks and poor inner city whites, problems of 'temperament' in toddlers and deficiencies in serotonin reuptake mechanisms in incarcerated criminals - is clearly going to keep a generation of psychologists, neuropharmacologists and behaviour geneticists in research funds for a good few years to come. One of the keys to success in science is to identify the appropriate level of analysis at which to seek the determinants of complex phenomena. Yet when the differentials between rich and poor are so great and widening, where the potential rewards of violence may be so great (and if large enough can even be socially sanctioned) - and especially where, as in the US, there are said to be more than 280 million handguns in private ownership, to look to biology to provide a determining explanation of what is going on is an expensive and foolish diversion.

Even in less dramatic instances, emphasis on genetic explanations and

molecularly-oriented research prevents researchers from seeing and studying the obvious. The almost universal conviction amongst biological psychiatrists that schizophrenia is a genetic disorder means that they are unable to respond to the suggestive epidemiological evidence that the diagnosis of schizophrenia in the children of black-white relationships in Britain is severalfold higher than that of either of the parental populations.[13] No genetic model fits this finding as well as an explanation in terms of the racism of the society in which these children grow up. Yet it is well known that with a little ingenuity any phenotypic distribution can be explained genetically, granted appropriate assumptions about the incomplete or masked effects of genes, technically known as partial penetrance and incomplete dominance. It is not hard for a behavioural geneticist to offer as an alternative that the data could be accounted for by assortative mating - i.e. that you must be mad to begin with to have a relationship with someone of a different colour from yourself.

There is no doubt that the dramatic increases in neuroscientific knowledge are changing and enriching our understanding of brain and behaviour. There is equally no doubt that, wisely and appropriately employed, the new knowledge offers the potential to diminish the degree of human suffering, at least in relatively wealthy industrialised societies. But until the neurosciences and genetics can be broken out of their reductionist mould and relocated within a more integrated understanding of the relationships between the biological, personal and social, abandoning their unidirectional view of the causes of human action so as to recognise the appropriate, determining level of explanation for complex phenomena - that is until we can stop looking for the key under the lamppost because that is where the light is, even though a moment's thought will tell us we lost it a long way further up the road - their potential for good remains limited and for misapplication substantial and disturbing.

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13. G. Harrison, *Schiz. Bull,* 16,1990, pp 663-671.