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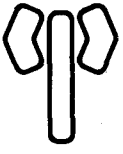

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S. J. Judge

Ideology and the Nature of Man

According to Rose, Kamin and Lewontin, the authors of *Not in our genes*, the recent history of the relationship between Christian Faith, Science and Society runs something like this:

> 'Darwinisn wrested God's final hold on human affairs from his now powerless hands and relegated the deity to, at the best, some dim primordial principle whose will no longer determined human action. The consequence was to change finally the form of the legitimating ideology of bourgeois society. No longer able to rely on the myth of a deity who had made all things bright and beautiful and assigned each to his or her estate—the rich ruler in the castle or the poor peasant at the gate—the dominant class dethroned God and replaced him with science. The social order was seen as fixed by forces outside humanity, but now these forces were natural rather than deistic. If anything, this new legitimator of the social order was more formidable than the one it replaced.' (p. 50–51) '"Science" is the ultimate legitimator of bourgeois ideology.' (p. 31—Double quotes are the authors' own, not mine.)[1]

What do the authors mean by this? What they principally have in mind is the political application of what they call *biological determinism*:

> 'Biological determinists ask, in essence, Why are individuals as they are? Why do they do what they do? And they answer that human lives and actions are the inevitable consequences of the biochemical properties of the cells that make up the individual; and these characteristics are in turn uniquely determined by the constituents of the genes possessed by each individual. Ultimately, all human behaviour—hence all human society—is governed by a chain of determinants that runs from the gene to the individual to the sum of behaviours of all individuals. The determinists would have it, then, that human nature is fixed by our genes. The good society is either one in accord with a human nature to whose fundamental characteristics of inequality and competitiveness the ideology claims privileged access, or else it is an unobtainable utopia because human nature is in unbreakable contradiction with an arbitrary notion of the good derived without reference to the facts of physical nature.' (p. 6)

1. Rose, S., Kamin, L. J., and Lewontin, R. C. *Not in our genes: biology, ideology and human nature*. Harmondsworth, Penguin (1985). Also published under the same title in New York by Pantheon in 1984—with Lewontin as first named author.)

The sort of statement that makes Rose *et al.* 'see red' is that attributed to Patrick Jenkin (British Minister for Social Services) when he said in a 1980 TV interview:

> 'Quite frankly, I don't think mothers have the same right to work as fathers. If the Lord had intended us to have equal rights to go to work, he wouldn't have created men and women. These are biological facts; young children do depend on their mothers.' (p. 6)

Their book has a two fold task:

> 'we are concerned first with an explanation of the origins and social functions of biological determinism in general ... and second with a systematic examination and exposure of the emptiness of its claims vis-a-vis the nature and limits of human society with respect to equality, class, race, sex and "mental disorder".' (p. 8)

Origins and social functions of biological determinism

I pass over quickly their attempt to carry out the first part of that task, because the authors content themselves with a flamboyant piece of pseudohistory that they themselves would be the first to characterize as unscholarly and ideologically-motivated if it were to come from the pens of their political opponents. This is a shame, for they touch on material which is worthy of more careful attention. For example, on the social consequences of the belief that schizophrenia has a genetic component they say:

> 'The father of psychiatric genetics, Ernst Rudin, was so convinced of this that, arguing on the basis of statistics collected by his co-workers, he advocated the eugenic sterilization of schizophrenics. When Hitler came to power in 1933, Rudin's advocacy was no longer merely academic. Professor Rudin served on a panel, with Heinrich Himmler as head, of the Task Force of Heredity Experts who drew up the German sterilization law of 1933.' (p. 207)

Certainly this is disturbing reading—but it is only the starting point for anything in the way of a serious study of the relationship between geneticism and its social consequences and you will look in vain in the book for anything more than this sort of argument by shocking example.

It may well be, as Bateson[2] says in a balanced review of the book, that 'it is very convenient for those who have great power, prestige and possessions to attribute their good fortune to some unchangeable aspect of their natures' but Rose *et al.* have certainly not given us a

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2. Bateson, P. Review of *Not in our genes. New Scientist*, January (1985) p. 24.

serious historical and social study of the extent to which genetic determinism has played such a role.

Is there evidence for genetic determination of social behaviour?

The second part of the book tries to show that

> 'no one has ever been able to relate any aspect of human social behaviour to any particular gene or set of genes, and no-one has ever suggested an experimental plan for doing so. Thus all statements about the genetic basis of human social traits are necessarily purely speculative' (p. 251).

This is at last something that we can 'get our teeth into'. The authors address three areas: the notion of 'IQ' and its heritability, the notion that schizophrenia is genetically determined, and sociobiology.

Sociobiology. It is sociobiology which really fills Rose *et al.* with indignation:

> 'Sociobiology is a reductionist, biological determinist explanation of human existence. Its adherents claim, first, that the details of present and past social arrangements are the inevitable manifestations of the specific action of genes' (p. 236)

No doubt it would help their argument if this were true, but according to Dawkins,[3] 'Unfortunately, academic sociobiologists . . . do not seem anywhere to have actually said that human social arrangements are the inevitable manifestations of genes . . . Rose *et al.* cannot substantiate their allegation about sociobiologists believing inevitable genetic determination, because the allegation is a simple lie. The myth of the "inevitability" of genetic effects has nothing whatever to do with sociobiology, and has everything to do with Rose *et al.'s* paranoic and demonological theology of science. Sociobiologists . . . are in the business of trying to work out the conditions under which Darwinian theory might be applicable to behaviour. If we tried to do our theorizing *without* postulating genes affecting behaviour, we should get it wrong.' Bateson, with whom Rose was once a scientific collaborator, takes some trouble to spell out just how ill-informed and illogical Rose *et al.* are in discussing one of Bateson's own interests—whether animals other than man tend to avoid mating with individuals that are familiar from early life.[2] Rose *et al.* claim that

> 'the nonhuman evidence is at best fragmentary; the prediction seems to be supported by observations of some baboon populations, and by unfortunate extrapolations from the behavior of new-hatched Japanese quail. . .' (p. 137).

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3. Dawkins, R. Review of *Not in our genes. New Scientist*, January (1985) p. 24.

According to Bateson, 'The principle is, in fact, based on more than a dozen quantitative studies of at least eight separate species, including the chimpanzee and *adult* Japanese quail. Unabashed, our experts go on to refute any evidence that might happen to exist with the "common observation of fairly indiscriminate mating among domestic or farmyard animals". This is not simply ill-informed and silly, it reveals deterministic thinking of the type they revile elsewhere. If animals behave in one way in one set of conditions, it does not follow that they will behave in the same way in another set of conditions. Indeed, the studies, which the authors purport to know about, show clearly that many animals will mate with members of the opposite sex they were brought up with, but prefer not to when given the choice.'[2]

Much the best parts of the book are the sections dealing with IQ, and with the cause of schizophrenia. These stand out from the rest of the text by reason of their relative lack of propaganda and by the careful consideration of data. I found them very interesting reading—and all the reviewers I have read were clearly of the same opinion. The material is presumably the work of Kamin, the experimental psychologist who first drew attention to the suspiciously coincidental correlation coefficients in what were purported to be separate studies by Burt of the heritability of IQ. Others (Gillie[4] and Hearnshaw[5]) then went on to demonstrate the frauds that Burt had perpetuated in the latter part of his life. Kamin has written a book about IQ[6] and seems to have ceased doing experimental science and become a sort of scientific 'bloodhound', searching for evidence of Burt-like frauds in other fields. This is by no means a dishonourable occupation and his work is, unlike a great deal of the rest of the book, worthy of our attention. I should say that I am not a psychiatrist or a geneticist and so my comments on this field are those of an outsider who has read a good deal, but not all of the material cited by Rose *et al.* Since the IQ controversy is a relatively well-known matter, I shall not discuss it in this paper, but instead examine the critique advanced by (I assume) Kamin of the evidence for a genetic factor underlying schizophrenia.

Schizophrenia. The first point to make is that there is simply no question of anyone now believing that schizophrenia is entirely

4. Gillie, O. *Sunday Times*, (London), 24 October 1976.
5. Hearnshaw, L. S. *Cyril Burt: Psychologist* London, Hodder and Stoughton, 1979.
6. Kamin, L. *The Science and Politics of IQ.* Potomac, MD; Erlbaum, 1974.

genetically determined. For example, according to a standard British textbook for psychiatrists: 'The strongest evidence for *predisposing* factors comes from genetic studies, but it is clear that inheritance cannot be the complete explanation and that environmental factors must be important as well.'[7] So the first point to make is that Rose *et al.* are setting up a 'straw man'. But they do more than this. They also attempt to show that the evidence for any genetic component is negligible. I am going to discuss this claim in some detail, because it is supported by a considerable amount of careful discussion of the data and, whether in the end one rejects the criticism or not, it certainly shows that the data are not nearly so clear as one might have expected from a casual reading of the textbooks.

There are three kinds of evidence addressing the issue of whether schizophrenia has a genetic predisposing factor. It has long been known that schizophrenia tends to run in families. In the general population (certainly in Northern European countries and North America and probably elsewhere) the risk of becoming schizophrenic at some point in one's life is approximately 1%. Amongst the siblings of schizophrenics, however, the risk of becoming schizophrenic is approximately 10%.[8] Rose *et al.* do not contest these data, but neither they nor those working in the field would make any strong conclusions from them.

The second kind of evidence comes from twin studies. Almost since the condition which we now call schizophrenia was first suggested as a distinct type of psychosis by Kraepelin at the end of the nineteenth century, psychiatrists have attempted to examine the relative importance of genetic and environmental factors in the etiology of schizophrenia by comparing the frequency with which schizophrenia occurs in monozygotic (MZ) and dizygotic (DZ) twins. There are several difficulties with this line of attack. The first is that both schizophrenia and twinning are quite rare events, so their conjunction occurs only in a very low proportion of the population, making the location of subjects a very onerous task. Nevertheless, several such studies have been made. A second difficulty is that determined believers in non-genetic explanations can always argue that identical twins 'usually share a disproportionate segment of environmental and interpersonal factors in addition to their genetic

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7. Gelder, M., Gath, D. and Mayou, R. *Oxford Textbook of Psychiatry*, Oxford, OUP, 1983.
8. Gottesman, I. I. and Shields, J. *Schizophrenia: the epigenetic puzzle*. Cambridge, CUP, 1982.

identity'. That quote, incidentally, is not from Rose *et al.* but from one of the supposed arch villains—Seymour Kety.[9] Rose *et al.* first note, quite correctly, that a large proportion of all the twins ever studied for this purpose were located by a man called Kallman. Kamin obviously thinks he suspects another 'Burt' here, and indulges himself in a variety of attempts to discredit Kallman. Kallman certainly was a eugenist of the 'old school' who believed that schizophrenics should be sterilized to stamp out the disease. He worked in Germany until 1938 when, as a Jew, he was forced to leave. Rose *et al.* are not, however able to come up with any hard evidence that Kallman faked his data in the way that Burt did. In his study reported in 1946, Kallman found that 69% of his MZ twins were concordant for schizophrenia, whereas only 11% of DZ twins were. Similar results were obtained by Rosanoff in 1934 and Slater in 1953 (references in Rose *et al.*), but more recent studies have, on the face of it, found lower concordance rates for MZ twins and a smaller difference between MZ and DZ concordances—i.e. weaker evidence for a genetic component. The usual explanation for this difference is that the earlier workers surveyed chronically hospitalized patients who were probably as a population more severely ill. Gottesman and Shields have a long discussion (see chapter six of their book) as to why the discrepancies between older and newer studies are perhaps not as great as they seem from Table 8.2 of Rose *et al.*[8] The reader who really wants to judge for himself the truth of the matter is directed to Gottesman and Shields' book. I should, however, like to draw attention to the disingenuous summary sentence on p. 217 of Rose *et al.*, which will, I think, indicate that not all the bias is on the side of the geneticists. They say

> 'The concordance of MZs reported by modern researchers, even under the broadest criteria, does not remotely approach the preposterous 86% figure claimed by Kallman.'

There are two pieces of trickery hidden in this statement. First, the notorious 86% figure is not Kallman's actual finding for concordance but an extrapolation (by Kallman) based on what is now universally agreed to be an erroneous correction for age. The intention of such corrections is to allow for the fact that schizophrenia sometimes does

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9. Kety, S. S., Rosenthal, D., Wender, P. H. and Schulsinger, F. The types and prevalence of mental illness in the biological and adoptive families of adoptive schizophrenics. In: *Transmission of Schizophrenia*, Eds. Rosenthal, D. and Kety, S. S., Oxford, Pergamon. 1968.

not appear until latter in life, so unless all co-twins survive through the whole risk period for schizophrenia and are followed up for that whole period, the concordance rates found are underestimates of the true rate.

Rose *et al.* try to test the notion that environmental effects may, after all explain the data by two comparisons (Tables 8.3 and 8.4). They compare the incidence of schizophrenia amongst DZ twins and siblings of schizophrenics and show that in two studies (out of six) there is a significantly higher risk of schizophrenia in DZ twins than in siblings. Since, as they point out, DZ twins are no more genetically alike than siblings, the difference must be environmental. And they have a similar argument on the basis of comparing same-sexed and opposite-sexed twins. But this is a muddle: no one contests the existence of environmental factors, and their demonstration says nothing about whether there is or is not a genetic factor.

Rose *et al.* are able to show that at least in two cases, there has been a certain amount of conjecture involved in deciding whether some twins were or were not schizophrenic. But 'two swallows do not make a spring' and it seems, at least in the case of the Gottesman and Shields study that the very fact that they reported their inability to interview one twin attests to their honesty.

Rose *et al.* then turn to the third kind of study—adoption. The aim of this is to study the rates of occurrence of schizophrenia either in adopted children of schizophrenic mothers, or in the biological relatives of adopted children who subsequently become schizophrenic. The most famous adoption study is of the latter kind and was carried out by Kety *et al.* in Denmark, making use of the national records there of adoptions and (separately) of psychiatric treatment.[9] Kety *et al.* found 33 schizophrenia adoptees from the Greater Copenhagen area, and chose a control sample of 33 adoptees matched for the usual social variables. They were able to locate approximately 150 biological relatives of each of the two groups, and then to look at the incidence of psychiatric disorder in the records of these as well as the adopted relatives. This study is usually taken to establish conclusively the importance of genetic factors in the etiology of schizophrenia. What is not always made clear, and something which Rose *et al.* duly pounce on, is that chronic schizophrenia was no more common among the biological relatives of the controls—to be precise there was one in each group. What Kety *et al.* did find, however, was that 'schizoprhenic spectrum' disorders were significantly more common amongst the biological relatives of the schizophrenics than amongst those of the control group. When one looks at the diagnostic classification scheme used by Kety *et al.*

these spectrum disorders include 'border-line (schizophrenic) state', 'inadequate personality' and 'uncertain (schizophrenia)'. Rose *et al.* assert that:

> 'without the inclusion of such vague diagnoses as "inadequate personality" and "uncertain borderline schizophrenia" there would be no significant results in the Kety study.' (p. 222)

This is true. But the unbiased reader should note several points not mentioned by Rose *et al.* The largest group—the 'border-line schizophrenics' (B3 in Kety's classification scheme) is not mentioned by Rose *et al.* There are six of these amongst the biological relatives of the schizophrenic adoptees and only one amongst the biological relatives of the controls. So, including the one chronic schizophrenic in each group there are 7/33 schizophrenics amongst the biological relatives of the schizophrenic adoptees and 2/33 amongst the controls. This value approaches, but does not reach, significance at the 5% level. The real conclusion from these data is that the sample is too small. Furthermore, it should have been clear in advance that this was a likely outcome. If we take the estimate from other studies of the incidence of schizophrenia amongst relatives of schizophrenics as about 10%, then it is easy to calculate from the usual binomial probability formula that the expected number of schizophrenics amongst the relatives is 3.0 ± 1.7 (S.D.). In other words, one would not have expected the study to show a significant result when schizophrenia was strictly diagnosed. Kety *et al.* have gone on to extend their study in two ways—first by enlarging the sample to include the whole of Denmark and secondly by actually interviewing the relatives rather than relying on diagnoses from the national psychiatric records. As Rose *et al.* say, the results of this larger study are not yet reported in full, so one must suspend judgement. According to Gottesman and Shields, (Table 7.4) the results are now significant, but cases of 'uncertain schizophrenia' are still included in the tally so that the sceptic will still not be convinced.[8] Rose *et al.* further suggest that there is a source of bias in the data which potentially undermines the validity of the whole method:

> 'From data kindly made available to one of us by Dr. Kety, we have been able to demonstrate a clear selective placement effect ... When we check the adoptive families of the schizophrenic adoptees, we discover that in eight of the families (24%) an adoptive parent had been in a mental hospital. That was not true of a single adoptive parent of a control adoptee'—suggesting that the schizophrenic adoptees 'acquired their schizophrenia as a result of the poor adoptive environments into which they were placed.' (p. 223)

I have not time to consider all of the issues raised by Rose *et al.*, but the reader will see that there is certainly room for doubt about the issue of whether schizophrenia has a genetic basis. The writer, whom I assume is Kamin, has undoubtedly done a good job for the opposition (though his bias is at least as evident as that of the geneticists) and it is not at all obvious to me as an outsider that the issue has been laid to rest.

Perhaps it is true that the difficulties of interpreting the data on genetic factors in the etiology of schizophrenia are greater than one might think from the textbooks, but what does this establish? Does it really carry the vast consequences that Rose *et al.* suggest? No one is now sterilizing schizophrenics. No one (geneticist or environmentalist) knows how to cure schizophrenia, although it has been discovered empirically that some drugs alleviate the symptoms in some patients. If there is a tendency to slide over the difficulties with the data as to whether schizophrenia has a genetic component, may I suggest two alternative hypotheses to the one proposed by Rose *et al.* as the explanation for this tendency? The reasons I suggest are, first, that all scientists prefer simple hypotheses and the genetic hypothesis about schizophrenia was the simplest available. Secondly, despite all Rose *et al.* say, the truth or falsity of the matter has no clear consequences for modern psychiatric practice.

The final chapter of the book is an attempt to say something positive about what they propose as an alternative to 'biological determinism'. What Rose *et al.* propose as 'an alternative world view' is 'dialectical biology'. And what is dialectical biology? Shorn of its flag-waving label, it turns out to be the familiar moderate view that human behaviour is the result of an interaction between nature and nurture. Well, if that is radical science, we can all be radical scientists!