

7 Environmental Effects on Brain and Behaviour

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So far this book has considered various aspects of the performance of individuals and groups, as measured by IQ tests and other ways, and also what can reasonably be concluded concerning the genetics of such performance. In all these accounts, there has been remarkably little said about the organ of the body which is above all responsible for behavioural performance and that complex set of attributes defined as intelligence – the brain. Rather, the individual and his brain have been treated somewhat as a black box, with particular sorts of output. It is the purpose of this chapter to examine the structural, physiological and biochemical features of the brain which are relevant to an understanding of those aspects of its performance subsumed under the general title of intelligence, and to consider the evidence relating to factors which can influence this performance. Much of this evidence comes, inevitably, from animal studies, and it must always be borne in mind when evaluating it that extrapolation upwards to men is beset with difficulties. It is no more desirable socially, nor sound scientifically, to be ratiomorphic or chimpomorphic about humans, than it is to be anthropomorphic about animal behaviour.

Brain structure and performance

A nervous system may be defined as an organized constellation of cells (*neurons*), specialized for the repeated conduction of an excited state from receptor cells or from other neurons to effectors or to other neurons. It is essentially a device, first, for the reception of sensory information, second, for its processing, storage and comparison with past information, and third, for making decisions about how to act, on the basis of all this data, and instructing the effector organs of the body accordingly. The greater the storage and processing capacity of the system, the more effective it will be at these tasks.

Effectively to perform them, the nervous system needs to show two complementary features. If it is to be able to function at all, it needs to have a set of built-in, programmed responses. Particular patterns of sensory input must result in certain predictable outputs. A tap on the knee must result in a knee jerk, pain in a limb to its removal from the stimulus. These are examples of the *specificity* of the nervous system. In addition, however, it must have the capacity to respond to new information in a novel way. If a particular type of food tastes bad, it should not be eaten again. If a given type of activity produces reward, it should be repeated. This represents the *plasticity* of the system. Specificity is the genetically programmed, invariant response of the system, whilst plasticity is the learned result of experience. For any species, behaviour is a result of the sum of – often the tension between – the plasticity and specificity of its nervous system.

In all vertebrates, and particularly in mammals, the largest group of specialized cells performing the functions of the nervous system is in the brain, and an examination of human performance must, therefore, begin with examination of the human brain. The adult human brain weighs 1300 to 1500 grammes – heavier than most organs in the body. In appearance it is dominated by the large, convoluted, walnut-like masses of the cerebral hemispheres, which fold over and bury beneath them practically all other structures. The hemispheres themselves consists of a skin, three to four millimetres thick, of 'grey matter' above an internal core of 'white matter'. This thin skin is the cerebral cortex, and is densely packed with

nerve cells. The white matter beneath gains its characteristic colour from the high concentrations of a fatty substance, myelin, which forms the insulating sheath round the many nerves which run to and from the neurons of the cerebral cortex. Of the many structures of the brain, it is the cerebral cortex which is most concerned with conscious behaviour, the processing and analysis of incoming sensory information, and decisions as to appropriate motor responses. The cortex is the most plastic part of the brain, concerned with learning, memory and the coding of the experience of the individual.

The evolution of intelligence and consciousness

Thus, in an examination of factors which affect intelligence and performance, we may guess that the area most relevant to our study will be the cerebral cortex, although clearly many other regions of brain and body will have a part to play. Deficiencies in sensory input or motor output will affect performance. So will factors relating to attention and alertness, controlled by regions lower in the brain. One way of approaching the question of human intelligence may then be to ask how the human brain resembles, or differs from, the brains of other animals. The human brain is not, by any means, the largest of all. Elephants, dolphins and whales for example, all have heavier brains, but these animals also all weigh a great deal more than humans. It is a reasonable postulate that the more body cells there are, the more brain cells will be needed to control them. So a fairer estimate of brain weights is to relate them directly to body weights. When this is done the human brain ranks amongst the highest. But the differences are not dramatic. Clever as dolphins are humans are a great deal cleverer, and this is not reflected in any massive difference in the ratios. More striking perhaps, is the difference in the size of the cerebral cortex of humans compared to other species. In the evolutionary path to man, the development of the brain is characterized by a progressively greater dominance of the cortex.

The increase in cortex size, however, is not so striking as the increase in learning capacity and plasticity of the brain, which is the unique feature of humans. Fishes can learn simple avoidance and discrimination responses, pigeons to count, rats to run

mazes or press levers for rewards, dogs to round up sheep and perform other complex tricks, dolphins to communicate with humans, chimpanzees to use simple tools, generalize and even, in some recent experiments, to construct sentences and use language symbols. All these are examples of brain plasticity: they are not inherited, but learned skills. In general it may be argued that, in the evolutionary path to man, there is an increase in plasticity, and a relative diminution in specificity. The specific 'innate' responses of organisms with smaller brains, from the fluttering of a moth towards a flame to the complex community relationships of ants and bees, are all based on specificity rather than plasticity. Their responses, at their most complex, as in the bee's capacity to make maps of the external world and to communicate them to the fellow members of its hive, are striking. The learning capacity of the bee on the other hand, is almost negligible. But with the human there is a quantum jump in performance, intelligence and consciousness.

What structural feature does this performance depend upon? In part, clearly on non-brain features. The human hand is capable of more complex manipulations than that of the ape. The structure of the human vocal and auditory systems is better adapted to making and interpreting complex sounds. But there must also be a combination of brain features which enable these advantages to be exploited. These features are clearly not just brain size, or cell number, or even cortex size. It seems probable that there is another crucial aspect of the organization of the brain which is relevant to performance. This is its connectivity.

Within the nervous system, neurons are connected to one another in such a way that signals arriving, say, from sense receptors, can be transmitted down other nerves to the effector organs, like muscles. The complexity of the instructions that can ultimately be transmitted depends on how much information, about present and past events, can be collated and computed before the final message is transmitted. Without going into the microscopic structure of the brain system, it is fairly obvious that the more neurons that are involved in the making of any decision, the more complex are their interactions and the more information the final message will be based upon.

The point at which one neuron makes contact with another,

across which information can pass between them is called the *synapse*, and each neuron makes many synapses. The synapses are thus the main information processing devices of the nervous system, for they enable a whole set of incoming data to be compared, collated and either acted upon or not. The synapse is the decision point of the brain; it has been likened to the yes/no gate of a computer. The capacity of the brain to store information depends not only on the number of its cells, but the number of synapses between them, which determines the number of possible interactions. And there is some evidence that this number is larger in men and monkeys than other animals. The neurons of the rat cerebral cortex may each make only some 10^2 - 10^4 synapses; in monkeys and some regions of the human cerebral cortex the figure may be an order of magnitude higher, from 10^4 - 10^5 . There are estimated to be 10^{10} neurons in the human cerebral cortex, perhaps 10^{14} synapses. This is 30,000 times as many synapses as there are humans on earth, and we may postulate that the specifically human aspects of intelligence and performance are indeed some function of the number of neurons and their connectivity. To ask the question 'what determines intelligence?' can then be rephrased in neurobiological terms as 'what factors decide or influence neuronal cell number and connectivity in the brain?'

But to ask the question only in these terms misses one highly significant point. In listing the features which distinguish man from the apes, we did not include one of the crucial ones; his capacity to live and communicate in social groups. Because of this capacity to communicate with his fellows, first in words, later in the more permanent form of writing, the information available to the human, even from early days (30,000-100,000 years ago) surpassed by orders of magnitude that available to his evolutionary neighbours. Storage and transfer of information, outside the brain, became possible, and the experiences of one individual could be transferred to another, even across generations. Hence the social evolution of man, which is so important in understanding the present situation, could begin. The difference in performance, and doubtless intelligence, of the men of today from the earliest of *Homo sapiens* is enormous, precisely because of this factor. The cranial capacity of the

early humans does not suggest they had a greatly different brain size from ours, yet the range of their activities was much more limited. Yet the 30,000–100,000 years that have elapsed since the early days of man is far too short a time for major genetically derived evolutionary changes to have occurred. To ignore the social environment in which man operates, to ignore the fact of man becoming, and to postulate any model of brain function as an absolute outside this social environment, is to be guilty of an error of cardinal significance.

The development of the brain

So far we have been concerned with the performance of the human brain compared with that of the animal. But what distinguishes the performance of one human brain from that of another? This is clearly the key question with which this book is concerned. One obvious distinction might appear to be brain size. Some people have heavier brains than others. But when expressed in terms of the brain weight to body weight ratio, there is surprisingly little difference between sexes, races or individuals. Post-mortem examination of Einstein's and Lenin's brains revealed no significant differences in terms of weight ratio or obvious structures from that of the average human. The performance differences must depend on micro-structural differences at the level of synaptic interactions in the living brain which no post-mortem study of pickled sections can reveal. Whilst one thus cannot detect in individual adults the cause of differences in performance, an examination of the pathways of development of the brain during infancy may help to do so.

Amongst all the organs of the body, the brain is unique in that the neurons are a non-dividing cell population. Each of us is born with very nearly his full complement of neurons, a very few more being formed in the first months after birth, and the brain at birth is closer to its adult size than any other organ of the body. It comprises 10 per cent of body weight compared with 2 per cent of the adult. By six months old the brain is 50 per cent of its adult weight, at a year 60 per cent, at five years 90 per cent, at ten years 95 per cent. The nine pre-natal months are thus a period of enormous brain cell growth, and

most of the key brain structures are established at birth. The developments after birth are of three kinds. First, the proliferation in the brain of a type of cell other than the neurons (glial cells) which have a predominantly supportive role in brain function. Second, the development of the synaptic connections of the neurons. Third, the laying down of the myelin sheaths of the nerves, which enhances their function, and hence the development of the characteristic brain 'white matter'. There is a complex but ordered pattern to this development which is the expression of the genetic programme of the organism in interaction with the environment.

It is during this pre- and post-natal development that the interplay of specificity and plasticity becomes of such major significance for the organism, and it may be as well to clear up here some of the misconceptions that abound in this area. The genetic programme of the individual is an expression of the DNA content (the genes) of the egg and sperm from which he develops. But this genetic programme can never be expressed without an environment in which the expression is to occur. If the environment is inadequate, the individual simply dies. From the first moment of cell fusion, there is an interplay between the genes and the environment of the most complex and interactive kind. During development, the pattern expressed on any gene becomes part of the environment of all the other genes. Even marginal differences in the external environment may induce a variety of changes in the nature and quantity of the protein being expressed upon the genes. It has been observed in a certain mosquito, for example, that a phenotypic female can be produced from a genotypic male simply by exposing the organism during development to an elevated temperature (29°C). In female rats, implantation of testosterone, a male sex hormone, into the brain of the young animal can produce male sexual behaviour in the adult. To attempt to parcel out hereditary and environmental influences during such developmental sequences is meaningless.

The interaction of genetic programme and environment is not really difficult to understand, yet it has been made the subject of vast oversimplifications. Thus in a recent book one psychologist referred, in apparent good faith, to the existence of 'high

'IQ genes'. Brain performance depends on structural interactions, which in turn depend on changes in a large number of interacting biochemical systems. A recent analysis shows a considerably higher percentage of the brain's genome – its DNA complement – is switched on to the manufacture of protein than in any other organ. The genes of the brain are producing more different types of protein – perhaps 30,000 different ones in all – than any other part of the body. A large number of these proteins presumably play a role in specifying brain structures and hence performance. Only in very rare cases – the one or two genetic diseases in which one of the genes is absent, as in phenylketonuria, for example – is it possible to specify the effect of the absence of a particular gene in relationship to function. As Bodmer points out, phenylketonuric children, if untreated, suffer considerable brain and performance damage. The gene which prevents phenylketonuria, because it produces a key enzyme which helps metabolize the substance phenylalanine, is present in normal children, and if they are compared with the phenylketonuric children, it is obviously by this definition a 'high IQ gene'.

But even here the child can be spared brain damage if its environment is modified at birth. If for example it is fed a diet containing no phenylalanine, it will develop practically normally. Hence the environment has 'triumphed' over the genetic deficiency of the individual. The fact is that, unlike a simple trait like eye or hair colour, brain performance is profoundly complex. Not only does it depend on a very large number of genes, but it displays a plasticity such that if one or several of these genes is modified the performance of the others is affected so as to tend to compensate. To talk of 'high IQ genes', or to try to disentangle the genetic programme from the environment in which it is expressed is both disingenuous and misleading.

What can be studied are the differences between the developmental pathway which is taken in a 'normal' environment – that is one varying over rather small limits – and one in which quite substantial changes outside these limits have occurred. The problem of interpretation, however, remains; for plasticity, that is the capacity for continued interaction with the environment, is itself programmed into the genetic specification of the human.

If the brain were rigidly and deterministically specified it would be useless as a brain.

Certain parts of the system are, however, laid down fairly rigidly. A supreme example is the pattern of pathways and interconnections of the visual system, from the retina of the eye via various intermediate neurons to the visual region of the cerebral cortex. If these pathways were not specifically programmed the organism would not be able to receive and analyse visual input and would be disadvantaged. What are not specified, however, it would seem, are the connections of the visual analyser cells of the cortex themselves. Much of this region of the brain is relatively undeveloped at birth; electrical activity develops in it only slowly and myelination is also retarded compared with other brain regions. The newborn child may be able to see, but it is doubtful if it can analyse or understand what it sees. The development of this capacity to analyse depends on subsequent interaction of the environment with the brain and can indeed be modified, as will become apparent subsequently.

Environmental effects upon brain structure and performance

I now turn to an examination of the way in which such modifications of the environment can and do affect both brain structure and brain performance. One of the severest environmental alterations which can be provided, short of actually physically damaging the brain, is to deprive the organism of food. When this is done in the adult the body weight declines sharply, but the brain weight is relatively unimpaired. The biochemical defence mechanisms of the body protect the brain against being used as a food reserve practically until death from starvation. The body sacrifices almost every other organ in preference to the brain.

But in infancy and during brain development the situation is different. Work on experimental animals, particularly that of Dobbing, has shown that if they are malnourished or undernourished for periods during weaning, or in the period just following, which are the times of rapid brain growth, then, not only will body weight be dramatically affected; but brain growth itself will also be retarded. Even if the animal is sub-

sequently fed freely, with as much food as it can eat, the brain growth may never catch up. During infancy in the rat or the pig for example, there are thus certain sensitive periods during which, if brain growth is impaired by malnourishment, the effect will last for the lifetime of the individual. In the case of the rat, this malnourishment can be achieved by the relatively simple method of taking two litters of pups born the same day, mixing them and returning a few - three say - to one mother and the rest - which may be up to fifteen or twenty - to the other. The 'large family' pups will have permanent deficits in brain weight which cannot be remedied after weaning, however good the diet they are on. Under these circumstances, the brain weight/body weight ratio stays permanently outside the normal limits.

In addition, there is a deficiency in the level of brain DNA (which is presumably a measure of brain cell number) which is never retrieved in these undernourished animals. Such deprivations will also result in performance deficits. Baird and her colleagues have shown that rats malnourished from conception, birth, or weaning, made more errors when tested on a Hebb-Williams maze than controls, either during their period of malnourishment or after a period of five weeks of rehabilitation.

How far are these observations relevant to the human situation? Rats are different from humans, not only in the obvious ways, but also in terms of the state of the development of their brain at birth. Compared with humans, rats are born relatively retarded. A baby rat is blind and naked. Biochemical studies show that a major spurt in its brain development occurs in the first two post-natal weeks, when a large variety of structural and biochemical systems rapidly mature, including the glial cells, synapses, and myelin formation. This then forms a critical developmental period for the animal. The human situation is different. Relatively more of the human infant's brain development has occurred prior to birth, and Dobbing has suggested there are two critical periods in human brain development, one pre-natally, the other the period of glial proliferation and myelination over the first eighteen months to two years post-natally.

To extrapolate from rats to humans is neither easy nor

sensible. But in many parts of the world, child malnourishment occurs, and its scale is terrifying. The key problem is protein deficiency, and it has been estimated that more than half of all the children in the world are 'at risk' from effects of protein deficiency, manifested in the most serious cases as the disease Kwashiorkor - but even in less serious cases the deficiency is on a scale likely to cause irrevocable impairment of brain development. This is the most serious nutritional disease in the world, affecting most strongly the children in the lower socio-economic groups. Three hundred and fifty million children under the age of six are probably thus affected.

The relationship of brain-weight to nutritional status is revealed clearly by studies in Latin America, which show that severely undernourished children have a reduced head circumference compared either to high socio-economic groups within their own country, or to American or western European children. Coupled with the small head size goes small body stature, increased age of puberty, and a variety of other features as well, not directly relating to brain performance. Nonetheless, even when the calculations are made on an estimated brain-weight to body-weight ratio, such undernourished children, or adults who have been undernourished in childhood, show deficits. We may expect to find that the critical periods for malnourishment in humans fall both pre-natally and in the first post-natal years.

Similarly, performance deficits occur in undernourished children. Cravioto and his group have studied such children in Mexico, and shown that compared with the children of rich parents in the same country, they have reduced sensori-motor skills and capacity to associate across modalities - pretty basic performance deficits which clearly will manifest themselves in such measures as the IQ tests.

Children in America, Britain or western Europe are unlikely to be as undernourished at those in Latin America or India. Nonetheless, longitudinal studies on British children have shown that small body size tends to be correlated with low socio-economic status and large family size. That is a polite way of saying that they come from a family with not much money to spend on food. Once again the critical question is how far

actual performance is affected by such poverty and malnutrition. Whilst it becomes extremely difficult to sort out many of the other related variables in such studies, the longitudinal studies suggest that low IQ scores are also associated with low socio-economic background of the parents, large family size and poor maternal state of health during pregnancy. Pasamanick and his associates have found that deficiencies of maternal diet associated with low income can produce complications of pregnancy and parturition followed by intellectual retardation and behavioural disorders in the children. Harrell and his co-workers added nutritional supplements to the otherwise deficient diets of pregnant women, and found significantly raised IQs in their children at ages two, three and four compared with the children of mothers without the supplements. Whilst gross undernourishment is infrequent in Britain, malnourishment and the diseases of deficiency are disturbingly frequent. It would seem probable, therefore, that a considerable number of performance deficits in children from deprived backgrounds are associated with long term impoverishment of this sort.

In terms of human welfare and politics such effects are surely of great significance, because the obvious way of remedying them lies to hand. Elaborate arguments as to genetic effects on intelligence, which present a prescription for social and political inaction because they are apparently at best irremediable, can only hinder the eradication of this monstrous situation.

However, it may be argued that the discussion does not come to grips with the issues of whether less extreme environmental situations can produce effects on brain performance. In this context, the work of groups studying the effect of changed environments on brain chemistry and performance is of relevance. Some of the most detailed of these have been made by the group of Bennett, Krech and Rosenzweig, who rear littermate rats from birth in one of two types of condition. One type 'environmentally impoverished', where, although fed enough, the animals are caged individually in conditions of low sensory stimulation of light or sound, out of sight of their fellows. Their handling is reduced to a minimum, or avoided altogether. The comparison group is reared in an 'enriched environment' living in a communal cage, and handled often, with plenty of

'toys' to play with and objects to explore. At the end of some weeks in either of these two conditions, the animals are killed and certain characteristics of their brain chemistry examined. Environmentally enriched animals are found to have a thicker cerebral cortex than the impoverished ones, and certain brain enzymes, notably those associated with synaptic transmission, such as acetylcholinesterase, alter in concentration. As little as one hour a day of the enriched experience is enough to cause measurable differences in these parameters. Very recently, this group also claimed that there are significant differences in the size of the synapses in the cerebral cortex of these animals when viewed electron-microscopically.

These changes relate to brain structure and chemistry. The question of performance changes remains to be described. These have been studied to some extent by the Bennett, Krech and Rosenzweig group, who do find performance differences between their two groups of animals.

The recent experiments of Blakemore and Cooper, and of Hirsch and Spinnelli, show that quite basic brain mechanisms are environmentally modifiable. In the Hirsch and Spinelli experiments, kittens were reared under conditions of controlled visual experience, so that one eye was exposed only to vertical lines and the other simultaneously to horizontal lines. At the conclusion of the training period, it was found that the cells of the two halves of the brain responded differently to visual inputs, each side responding best to the type of pattern that was familiar to it through the training. Early experience in these animals has directed subsequent physiological performance, even in as apparently genetically 'programmed' a response as the development of the analytical capacity of the visual system. Many other 'innate' and 'specific' behavioural responses are now known to be a good deal more plastic and modifiable, and less specific, than was once believed.

But the classic, and perhaps most relevant examples of the effects of early environmental experience on behavioural functions remain the well-known experiments of the Harlows, who showed that rearing monkeys in isolation resulted in an apparently permanent inability to form normal social, sexual or parental relationships in later life.

How far this type of experiment is related to the human experience is of course subject to all the cautions about extrapolation that have already been made. Environmental impoverishment to the extent involved in these studies, such as rearing in the dark, is obviously rare. The contrast between the impoverished and enriched environment of humans, even at its most extreme, cannot be so dramatic as this. Nonetheless, the differences are there, and all the evidence from the animal studies must lead us to suspect differences in humans in response to environmental changes as well. What can be less easily separated in the human situation are the nutritional and environmental effects, as children from impoverished environments are more likely *a priori* also to suffer nutritional disadvantages. The most relevant studies in this regard are the longitudinal observations made in Aberdeen and the experiment of Skodak and Skeels, referred to by Professor Bodmer (see chapter 5).

Transgenerational effects

The thrust of the argument so far advanced has been that environmental effects on brain performance and brain structure are profound and must always be so confounded with the genetic ones that parcelling out is meaningless. But we come now to the most important class of effects of all from the point of view of this argument, effects which, in the human situation are formally totally indistinguishable from the genetic ones, and yet are consistently ignored by those who would claim to have characterized an isolable genetic component in brain performance. These effects are referred to as transgenerational.

We can discuss them first in terms of the animal experiments. It has already been pointed out that nutritional deficiencies in childhood result in permanent changes in brain chemistry for which a subsequent adequate diet does not compensate. Recent experiments have also examined the effect of these deficiencies in the second generation. If malnourished female rats are then allowed to produce offspring, what will be the effect of the maternal infantile malnourishment on the brains of the offspring? Several intriguing experiments have shown that the undernutrition of the mother is reflected in low brain weight in the offspring. The most recent results are those of

Zamenhof and his group, who earlier had shown that protein deficiency in infancy in the rat results in lowered DNA (lower cell numbers) in the adult. They then mated the females amongst these infantile undernourished animals with normal males. Amongst the offspring, whether weaned by their own mothers or by normal foster mothers, there was also a highly significant reduction in DNA content, and hence probably in cell number, in the brain.

Such effects are transmitted between one generation and the next. Yet they are not genetic but environmental. Presumably after an adequate number of generations have been adequately nourished, they are reversible. Such effects are also reflected in performance. Again the Harlows' work may be cited. Not only is social behaviour in monkeys reared under deprived conditions itself abnormal, but this abnormality reflects itself in the rearing of the animals' own infants. Thus the rearing of the second generation is affected by the childhood experiences of the previous one.

In other words, the Harlows' monkeys are demonstrating the truth of the well known observation that the sins of the parents are visited upon the children. Except that in this case – and maybe in most other cases as well – the parents themselves are more sinned against than sinning. Children reared in deprived conditions are 'at risk' in two ways. Not only are they more likely to suffer from diet-linked complications of pregnancy, but the consequences of this are more likely to ensure that they grow up and live in the same adverse conditions as their mother. Such handicaps combine to sustain the very conditions from which they are derived, so the vicious circle tends to be repeated for generation after generation. Clearly the correlations between IQs of parents and children are bound to be high, and give a superficial impression of genetic determination. To fail to appreciate this is to ensure that bad science is done and bad social policy advocated.

Conclusions

Of course, this all ought to be self-evident. Everyone knows, from their own experience, the extent to which their adult behaviour reflects their childhood experiences, and indeed,

anyone with any degree of self-consciousness at all will be aware how much his own childhood experiences affect the rearing of his own children. And if we did not know it from our own experience – that non-scientific guide – we would know it from the work of the psychoanalysts. It is easier to understand and accept these effects in humans than it is in animals. And indeed, it is obvious from our experience, and should not, perhaps, need to be said, that ‘enriched experience’ in childhood modifies subsequent performance. It seems almost bizarre that we should have been put in a position of needing to reaffirm some of these statements.

Brain structure and chemistry determine performance, and brain structure and chemistry are themselves subtly but profoundly affected both by immediate environmental influences and by those stretching back beyond our own generation and into an indeterminate distance into the past. Brain structure and performance are affected most seriously by the world tragedy of protein starvation in the developing countries, which puts 350 million children at risk, and of malnutrition and appalling social environment in the industrial societies of Europe and the United States. In situations in which parents are expected to bring their children up in the slum conditions of the Gorbals or Notting Hill or Harlem, fed badly and overcrowded at home, deplorably educated in overcrowded schools, condemned to lives in which unemployment may alternate with the alienated labour of an oppressive social order, the miracle of it all is the human capacity to triumph in spite of adversity, to succeed despite the odds. This is the key to human social evolution, to man becoming; his capacity to transform his environment positively.

What is quite intolerable is that, rather than throwing their weight behind the obvious measures which will immediately improve the environment and hence the performance of such a large proportion of humanity, certain self-styled advocates of ‘pure science’ – whatever that might mean – should continue to attempt to cloud the situation with a set of spurious arguments which have the effect – whatever the intention of their advocates – of justifying the present situation.

Further reading

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