Ann. Hum. Genet. (2000), **64**, 363–374
Printed in Great Britain

A review of *Dysgenics: Genetic Deterioration in Modern Populations*

W. D. HAMILTON

Editorial Note

This book review arrived here at the beginning of this year with a very apologetic note from the author, Bill Hamilton, about the long delay in producing the review and its 'rambling essay format'. Sadly, Hamilton died just a few weeks later from complications following malarial infection. Several tributes have been published, such as *Science* (2000), **287**, 2438, and we continue the tributes with a final unedited manuscript from the hand of this unique, colourful and idiosyncratic evolutionary theorist.

Dysgenics: Genetic Deterioration in Modern Populations. Human Evolution, Behaviour, and Intelligence Series. By R. Lynn (Series Editor: S. Itzkoff). Westport, Connecticut and London: Praeger. 1996. Pp. 237. £47.50 (hardback).

In a sense a dominance hierarchy has only one satisfied individual – she or he at the top. If the hierarchy is bottom-numerous rather than linear, as is the case with most human hierarchies, it is all the more true that the vast majority of people are dissatisfied, wishing they were higher up, a thought which provides a basic reason why democracies (and especially, within democracies, such institutions as their state school systems) have to be unstable. We see a wobbly pyramid, and particularly within that pyramid we see certain side stairs all human examples have by which demagogues skip up a level or two so as to shout down to the restless base that the whole structure is somehow 'wrong'. Under a different system, the demagogue shouts, 'You could be higher too'.

A similar image, I believe, can also reduce our surprise not only at the never ending objections to Neodarwinism but, taking the level more relevant to this review, explain the intrinsic popularity of the nurturist side in the 'nature *vs.* nurture' debate. Neodarwinism is just too ruthless in its realism to please a majority of people: even a faint implication that an attained low station in life or education has been inevitable is too much for that hope that we all must have, the simple wish to be higher; so it is too for the feelings the average person has about their children in schools.

Demagogues by definition have to be popular; almost equally they have to paint all those who speak out against them as deluded doom-sayers, scheming or fearful rightists, and the like. All of this sketches a background – a steep slope of average human preference – lying behind all the topics covered in Richard Lynn's *Dysgenics*. His very title guarantees demagogues to be girding against him; it is important to note, however, that among these will be not just the movers and shakers who write the 'PC' books with titles like *The Iniquities of IQ*, and *Wonderbrained Woman*, even if such authors are the most influential; others girding in gentler ways are simply the sunny optimist we all know in the office, and the neighbour at home telling us, almost without thinking, 'Believe me, it wasn't your Tom who failed, it was the school'.

One has to be brave, thick-skinned, and very persistent to swim against such popular anti-realistic currents. Richard Lynn, discussing the large bank of evidence that still steadily accumulates on heritability of aptitudes and differentials of fertility, shows in this book that almost all of the worries of the early eugenicists were well-founded in spite of the relative paucity of their evidence at the time. Correct both in their intuitions and in their assessment of the tentative data available, for most of the past hundred years Lynn shows that they have been unfairly derided. The concerns they had about declines in health, intelligence and conscientiousness are matters that we should still be much concerned with; yet at the same time he admits the blunt and contrary fact that all over the world where it is measured, intelligence, or cognitive ability as it is now more commonly called, seems to be shooting up, thus confounding at least the most direct versions of the selection formulas that he and others have all been using. Something is evidently wrong here and I will come back to it at length. First, however, let me make clear that by the 'early eugenicists' above I mean mainly

such pioneers as Galton and Pearson and their true followers, *not* those political demagogues who simultaneously created their own interpretation of Darwinism and chose immediate, forceful action in various directions without much consideration or data. Many new activists who claimed to follow Darwin or Galton were indeed sometimes absurdly bigoted and far more radical in their proposals than any evidence of their time justified. It was they who caused the unfortunate political movements to one side or another which ended in the mid-century giving the whole field of eugenics a bad name.

These stampedes, as I think they can reasonably be called, had their climax in the mass racist events in East Europe and Russia around the time of World War II. It is good to see these diversions in so-called eugenics at last, in this book, treated with calm and in proper proportion. As Lynn makes clear in his first chapter the real successors of Galton and Pearson were eminently liberal and largely non-political scientists with reputations usually already built on what they had done in genetic and theoretic fields quite unrelated to human breeding. This had also been the case with the founders themselves. Names here which probably most biologists will know, at one time or another attached to work or statements about dysgenics and/or eugenics in the first half of the century are Fisher, Haldane, Muller, Burt, Julian Huxley. Probably because of distaste for the over-enthusiasts, all except Julian Huxley and perhaps Herman Muller, drifted away from involvement in 'eugenics movements' *per se* as the movements became more politicised and activist during the early central years.

To repeat, the book's main theme amounts to a claim that all the aspects of apparent dysgenic process which had worried the pioneers from Darwin and Galton onwards through that list of names and others, were real and continue today. Scanty as the data had to be at the beginning of the century, it did not mislead: again and again Lynn's chapters end, after reviewing comparatively vast amounts of more recent evidence, stating about the early eugenicists concerns: 'Once again, the research evidence has shown that they were right'.

For the most part I don't have time or competence to check that all the modern studies carry conviction for me in their details. Still, I would doubt very much that dozens of scientists and psychometrists have all been making laborious shams of data collection or deliberate distortions in their results through political or personal biases; as for detailed studies of opposite tendency, showing there is nothing to be concerned about, there seem virtually none published. Instead from the other side tend to come lengthy and finicky critiques spiced with *ad hominem* point scoring – Sir Cyril Burt's father being Galton's doctor gives the type. In one case where the attempt was made by various writers of nurturist and leftist persuasion to convince the public – claiming even proof – that one psychometrist, Sir Cyril Burt, had published fraudulent data, it later came to light that the tirade that had been either wrong or grossly exaggerated in a very high proportion of the claims it had raised (Joynson, 1989; Fletcher, 1991; Mackintosh, 1995). Muddle a-plenty of a minor nature was indeed evident in Burt's late-life publications on IQ and heritability but no case of fraud has been proven. Stimulated by the earlier tirades to look up some of Burt's books and papers, a field I have hardly ever touched, it seemed strange to me that a man collecting and writing so much about remediations for handicapped people, working and running clinics in London's poorest districts, should ever have been seen as an elitist bigot. What I read suggested the attacks on his early work must have been primed by a mere handful of paragraphs, seldom even making up whole pages (and these within large books that otherwise hardly addressed the topic of genetic quality), in which he dared to state that he regarded some of the conditions he described as hereditary, thus inevitable and likely to re-create their problems if their bearers had children. In spite of so much clear refutation of evidence, the writers of the original attacks (which began with what I can only call a cowardly immediacy once Burt had died and it had become known that all his notes and records had been burnt – this on the advice of one of his detractors) have never changed their accusations and continue to republish them (Rose, Lewontin & Kamin, 1989; Gould, 1996). Papers at least half-heartedly supporting of the tirades still emerge but the focus appears to have shifted more towards showing muddle and repetition by an old man as opposed to claims of purposeful inaccuracy (Butler & Petrulis, 1999). The idea that Burt led his field away from the truths now generally accepted, or even proposed

unsustainably high values of heritability, seems now to be practically abandoned (Mackintosh, 1995).

Even at the time of the attacks various other studies had shown either the same results as Burt had claimed or values still more favourable to the 'nature' side. Slight elevation of the key heritability value has continued (Rushton, 1994; Eysenck, 1995). In Cambridge University in the late 50s, and partly on the basis of Burt's results, I was taught that about one half of the variance of human intelligence could be thought of as due to genetic factors. Herrnstein and Murray (in a book jumped upon within the past decade rather as Burt's work had been and for the same sort of reasons, though the attack this time is more on the side of the book's supposed racism) set the estimate only at 0.6 (Herrnstein & Murray, 1994): now Lynn on the basis of yet more sets of identical twins (and notable among these, more sets reared apart) gives the value at 0.82. Soon quite possibly we will know the identity of some of the genes that cause the variation concerned.

Reverting to Lynn's theme, it is such estimates combined with fertility differentials that exist all across the civilised world that suggest that human attributes of health, intelligence and conscientiousness should all be in steady decline. Yet on the health side Olympic records continue to be broken; on the intelligence side almost all the mass estimates show IQ as quite dramatically rising throughout the present century. Has something overwhelmed heredity? What can be happening and do we still have cause for concern?

In the chapter he devotes to this paradox, Lynn concludes, I think probably correctly, that the largest factor in the mass rise in intelligence has been worldwide improvement in nutrition. In essence this is the very same factor as has made possible the parallel rapid rise in human stature, as also, in the background, the great growth in world population. It is implied that undernourished humans from foetal life onwards put their meagre resources into 'bodily' systems (in which I would include the immune system) before they put it into the 'modern luxury' of their line – the enlarged neocortex of the brain. This is an idea I will come back to; for the present please just note it as making passable evolutionary sense. But such offsetting by nutrition, Lynn notes, has to have a limit: 'These environmental improvements are bound to be subject to diminishing returns. When their impact is exhausted, and if dysgenic fertility continues, phenotypic intelligence will begin to decline' (p. 112).

Could it be that, although all Lynn's predictions are correct regarding the genetic basis, nevertheless the human psyche plus its underlying necessities of physique and health are going to prove so infinitely plastic that, under the ministrations of not only better nutrition but an ever increasing scientific programme of 'medical nurture', our species can indefinitely keep ahead of any amount of decline in its 'nature'? I very much doubt it and would not want to see such a course pursued for reasons I'll come to later. Others may be more hopeful and many seem to be judging by how hopes of gene therapy and the like are enthusiastically pursued. But obviously in such a course we have to realise we are ceasing to be the free individuals we are now and have been in the past.

In any case, Lynn's resolution of the paradox seems to me plausible but perhaps not enough: reading, I had a sense of some more radical point needed for the degree of the discrepancy. Casting around in what I might call the hinterland of my own field, and what I know of Lynn's, for ideas that might help, and fossicking in remote, uncertain country in both respects, I am going to dare to suggest various factors.

My first two concern potential special trends in the upper and lower tails of the distribution of intelligence respectively, which seem neglected by Lynn. The first is that perinatal and surgical gynaecological skill is permitting the birth of larger-headed babies than formerly. There certainly was a mortality which used to curtail perinatal survival of the largest babies (Karn & Penrose, 1951), and this probably also affected the largest head sizes most. Since head size is positively correlated with intelligence (Penrose, 1961; Lynn, 1990; Rushton, 1995), the upper tail of intelligence may be becoming less 'thinned' than it once was. In other words newly permitted large heads may be adding their 'grey matter', as one might put it, to re-thicken the upper tail.

Since these extra births will tend to be within families which are also on the whole at the upper end of the IQ distribution whereas the parent-child correlations have been based on the whole range, they affect the reliability of Lynn's estimates of change. As regards dysgenesis,

however, they affect it the wrong way: had all babies been born rather than some of the bright ones suppressed by perinatal death, the parent-offspring correlation in IQ would have been higher than was actually estimated and the hereditarians' predictions based on family sizes would have been gloomier still. In spite of this, if the trend that I postulate is real and substantial, it has to be in the long-term beneficial, and in so far as the family size differentials come to reduce or reverse, it must add to long-term eugenesis. I have little idea whether the magnitude of this 'tail effect' is at all significant, but the dreadful rates of perinatal death revealed in fiction and biographies of the last century suggest that they might be. The hospital situations of rampant puerperal fever that Ignaz Semmelweis campaigned against for sure had no-one measuring live- and still-born heads and/or correlating this with the fate of the mother, so we don't know for certain about them. Yet it is hard to imagine that the larger heads were not indeed correlated with more tissue damage on both sides and therefore more chance of infection. Looking outward to a vastly different time scale, the same idea is more simply suggested by the evolutionary expansion of the human female pelvis: no factor except a very long-standing problem with babies too large-headed to pass the birth canal can plausibly explain evolution of this obviously athletically handicapping development in women.

Current manifestations of such an effect are, of course, a different matter from the eugenic aspect: in this regard they can well be contributing to the secular increase in brain size and IQ (as well documented in Lynn's Chapter 8). In so far as caesarean births are responsible for the change this may be a dangerous course for humanity to persist in if we value long term freedom (Hamilton, 1988); but in so far as the release of extra large human heads comes simply from better hygiene, better midwifery manuals, better survival of premature babies, better postnatal remediation of slight birthing damage to babies in normal births, and so on, such stricture is largely avoided. To summarise, this is a factor doubtless contributing to the mass phenotypic trend but, unlike the factor of nutrition, it may have a small eugenic component as well: the point is that more genetically brainy babies are now growing instead of dying and Lynn's calculations neglect them.

My second suggestion is of a differential this time affecting heritability from the lower tail of IQ. Human rates of extra-pair paternity within apparent families may be an important factor counteracting Lynn's trends. None of his calculations take this into account and yet most estimates of western populations set the rate quite high, at about 10% (Baker & Bellis, 1995). Moreover as required and as would be expected from sociobiological reasoning, these rates are highest lower on the social scale (Cerda-Flores *et al.* 1999). Within Lynn's general scheme this is just as is needed for the effect to be more pronounced even than the high figure of 10% would suggest. As with the other suggestion, the factor again means that the estimates of heritability Lynn relies on should again have been higher; therefore again we may have to think about how much the 'extra-pair' effect is offsetting what should have been still gloomier predictions. However, with this factor it is possible to imagine that if the impaternity rate in the 'lower tail' is high enough and if the phenotypes chosen by women for their infidelities are 'upwardly disparate' enough, the dysgenic situation for intelligence Lynn describes might not even exist.

As I already hinted, this is a rather gloomy solution for a gloomy problem: invoking it is like begging the dastardly Red Knight to help rescue the maiden chained to the rock, Sir Lancelot being too sick to be called upon. Or perhaps it's like going about the job by yourself but, applying chisel to chain, you notice an irritated instead of a thankful look from the maiden – you're not quite what she was hoping for. Alas, we have to describe what exists, not such stories.

My third suggestion (and confessedly a reluctant line for me) is that the mass rise in IQ might not be due to nutrition but instead due to some 'epigenetic' process. It seems necessary to discuss this because it is sure to be raised by some as a 'more natural' alternative to all the possibilities I have suggested above and we need to see what the scope of such a suggestion is. Might it be that Neodarwinism either doesn't work at all for intelligence or that it works but is far less than the whole story?

Occasional but persistent, like tropical jellyfish drifting to Britain, one or another new version of a Lamarckian modification washes to the sea walls of evolutionary theory almost every year. I believe that fundamentally most come in the

great ocean current of popular appeal that I outlined in my first paragraphs and that, as with the real jellyfish, most of such theories are moribund even as they arrive. But recently a more serious claim has come which invokes such striking facts of secular change that it perhaps deserves to be taken more seriously. Amongst the changes this version highlights is that we have already seen: the rapid mass rise of IQ in the civilised world. Miles Storfer (Storfer, 1999) depicts not only this rise but, in parallel to it, facts I did not know concerning a current upsurging of human short-sightedness. Early onset myopia apparently is increasing rapidly in almost all countries and cultures where medical records exist, implying seemingly all cultures literate enough to have records or to be starting them. It includes peoples such as Eskimos amongst whom until recently myopia was virtually unknown whereas now examples of the condition begin even in early childhood. Storfer argues that the change cannot be Neodarwinian microevolution nor even plasticity within a lifetime – the condition begins so early it cannot be brought on by your own childhood's close studying of books but rather has to have been induced by whether your parents closely so studied (and, a most surprising claim in Storfer's scenario, particularly by whether your *father* did so!). This is not the place to go into whether the evidence for the effect being epigenetic, rather than due to within-lifetime effects, stands up; I myself am not convinced by the evidence I have so far followed-up from his citations. However, epigenesis in such a feature as human myopia would be a very dramatic fact if confirmed. If further, as in Storfer's additional claim, similar findings apply to brain development, the trajectory of mass IQ then escapes from Lynn's discussion (no oversight is implied here, of course, since Storfer's review came later). It therefore seems a worthwhile exercise to accept the claims at face value, including epigenetic inheritance in forebrain development, and then to ask: can such effects negative the IQ dysgenesis Lynn predicts? And: how can such claims integrate with evolution theory? The problem – the apparent non-fulfilment of the dysgenic predictions – is a big one and justifies a far-ranging search.

Chemical impulses experienced during an individual's lifetime and passed into the development of offspring have been known for some time. In the 1920s when they were studied by Jollos in Germany they were given the name 'Dauermodifikationen' (Jollos, 1921; Jollos, 1934). Since that time many more similar effects have been found (Jablonka & Lamb, 1995). Characteristically they occur in small opportunist animals and in weedy plants, fungi, and bacteria. If environments of successive generations are positively auto-correlated there is nothing surprising, or fundamentally non-Neodarwinian, in this providing we acknowledge that some developmental impulses, other than by the code of the genes, can be passed through the single-cell zygote. Organisms presumably can 'learn' by natural selection ways of anticipating what developmental strategies will be advantageously started very early in their offspring and make provision for them. Enough chemical modification (including of course, as we now know, patterns of methylation of DNA) goes through the unicellular gate to provide changes of the environment in which the new generation's gene activity initiates. Primary codes on the double helices can begin their operation subject to switches set by one or other or both parents. These can be parents acting either in concert or discord (Haig, 1993). Regarding more concordant and uniparental 'direct guidance' to offspring, we expect this to be evolved especially whenever the waves of environments creating positive autocorrelation have been very numerous in the past and they really are waves – that is, they end and they repeat. The system needs to be arranged so that if reinforcement is not provided by parents in a given generation the effect fades away – either immediately or over a few generations. This is exactly what Jollos described. If on the other hand the environmental change was one that a species has never experienced before, it is very hard to see how a species can have any epigenesis prepared for it. Not understanding how any species, including our own before scientific thinking began, can have any foresight for changes that have never occurred before, I find it almost impossible to imagine how a species can ready a programme for changes that are not repetitive.

Compared to some other animals including our cousin chimpanzees, humans are a weedy opportunist species. We are more fertile than chimps and much more fertile than some other animals – some dung beetles, for example, seem to be restricted to maxima as low as four or five offspring for their entire brood. Given *H. sapiens*'

weedy opportunism – obvious enough perhaps in the way we have spread and conquered new habitats – it is pertinent to ask if our line has been *repeatedly* encountering periods where myopia and/or extra intelligence have been especially advantageous, these periods having been interspersed with others where these qualities were less required. It doesn't seem very likely. But there is another route to another kind of a 'modification' that may still rescue this general line of explanation.

As a primate line we have certainly a very long exposure to a condition of living socially. Almost certainly, as shown by present behaviour in chimps, this includes a great importance attached to sharing within social groups. Instead of looking for 'waves of need', may this social importance have continued long enough to have generated epigenetic machinery that readies offspring for what a parent 'foresees' socially as the necessities and opportunities of the next, with each parent seeking 'feasible fits' for its offspring into the group – fits which probably include taking into account the parent's own social position? (Status itself is indeed heritable in some macaques though this is most plausibly through simple learning.) Here the environments for which modifications are required are coming not as waves in time but as variations in a kind of 'social space' in which gene contributions and therefore lineages may show slow convection-like rise and fall (Hamilton, 1991).

Repertoires of development that include appropriate social switches certainly have been evolved in connected clonal associations, such as those of stemmed and stoloniferous hydroids, as also in some disconnected forms (Francis, 1979). Moving to non-clonal associations, the 'soldiers' of Francis among social sea anemones are suggestively paralleled by the 'soldiers' among ants and termites. Both kinds of soldier morphs appear where and in such numbers as are required for colony defence (Harris, 1961). Turning back to humans we may see signs of such readiness to take diverse paths existing in the striking findings about the personality differences connected with different birth order (Sulloway, 1996). Likewise we may see them at a genetic polymorphism level (which may be expected often to precede plasticity). Several genera of new world monkeys have extensive genetic polymorphisms for colour vision (Shyue, Hewett *et al.* 1995; Jacobs, Neitz *et al.* 1996; Lucas, Darvell *et al.* 1998) and it has been plausibly suggested that this, given a degree of sharing in the troupes, may be connected with a group advantage in having diverse specialists in the use of colour and monochromatic vision. Some animals are good at seeing the distant fruiting trees, some at breaking crypsis of nearby insects, some see better (the scenario can suggest) the distant connections of lianes in the dim understories of the forest and thus became leaders in finding a path for the group. Having a *genetic* polymorphism for such vision specialism within a group seems a clumsy way but, at a stage awaiting more plastic solutions, one can imagine it. So far, apart from our human colour-blindness variation which seems to hover at the threshold of a frequency suggesting a polymorphism, similar extensive colour-vision morphs have not been found in old world primates. So far as I am aware the possibility of potentially more efficient *plastic* specialisms in vision propensity that become exaggerated during development have not been looked for in any primate. If they are found they may, as parallels, begin to make Storfer's claims on myopia more understandable – in other words may suggest all these effects to have a sufficiently extensive social background for epigenetic machinery to have evolved. The idea would then be that modern living conditions have come to tell almost all parents that the specialism associated with being myopic (so conducive to adaptive efforts to become literate) is now so rewarding that they should head their offspring towards it – push them farther in the way the parent itself has been striving.

Since maintaining and using a big brain is very costly in energy and since typically the troupe in the tree tops needs at most a few of its number to be the path-picking leaders while the rest direct their specialisms towards other matters, including (it may be) their colour vision and (as must be) their reproduction, similar considerations could apply to a specialism in neocortical increase generally. A widespread 'morph' change, then, offers another naturalistic explanation for the mass rise in IQ in humans: suddenly, to cope with modern civilisation, more and more need to become 'specialists' in thinking; perhaps the predispositions have long stood ready – plastic for ourselves and epigenetic for our children. This view of course is not unlike (and in the epigenesis part it even out-does) the sanguine view of all nurturists. But, in so far as it works all

and nothing is proven, it is obvious that the 'caste' product is much less uniform than is a soldier termite. Perhaps we should feel relieved.

Along such lines I can see how Storfer's claims for a seeming 'dauermod' or caste-like epigenesis in humans can be brought into evolutionary theory: even if not uniformly able to respond, this view would say, all of us or more of us are now specialising the same way, more putting family resources into the same caste-line out of a formerly much wider repertoire. Many lines in the old repertoire are probably now obsolete (for example in the past one once fashionable specialism may have been tuning the olfactory brain axis of one's offspring to the scent of fruiting fig trees or the sweat of enemy groups).

All this of course is a digression from the topic of the review. Returning to it, the important thing to note is that, just as with the factor of simple nutrition, all the epigeneses suggested here are bound to have their limits. You may do better going for one of the specialisms evolutionarily prepared for by treetop ancestors; still, you shouldn't go so far out on that branch, whatever it is, as to forget having children, or at least not so far as to forget making sure that someone in your 'plastic' family is having them. In short an epigenesis argument concerning rising IQ cannot for the long term, whether concerning nutrition or something else, provide an escape from Lynn's conclusions.

Storfer, however, goes further and touches an idea that his effects may be not merely epigenetic engineering (as through methylation patterns perched upon an unchanging DNA) but may be gene conversions paving new development pathways. In so far as he is implying a back and forth between two rather fixed states [as was suggested by Fitch and Atcheley for some genetic phenomena in mice (Fitch & Atchley, 1985)], this does not escape from the strictures above except in the changes being made more sudden. But in so far as he is implying one mutation or conversion step carrying the phenotype in a particular reproductively competent direction while also making more likely another similar step to be taken (say, by a further duplication of the same locus), he is bringing in the usual problems of concept of orthogenesis: how does the process stop? – how reverse? – must it not go lethally extreme? If this is happening in respect of intelligence it would indeed provide an escape from Lynn's conclusions but surely this is not a reassuring solution: one needs only to think of an orthogenesis continuously operating on myopia to see that. If we postulate a mechanism providing eventual restraint we must ask how the restraint itself was evolved and again I can only think that there must have been many similar microevolutionary bursts within families in the past and the modifiers of those that switched on and off best are now established. This seems to need a very improbable time period and repetitive pattern. Thus it is more dubious even than my symbiosis of social forms above. In short while I am happy to imagine evolving patterns of methylations effected by parents I am much less so for series of locus duplications and 'return' excisions. In any case, short of imagining a mystic intelligence in species which foresees the future, both paths lead back ultimately to the cases and restrictions of the previous discussion.

To summarise so far, dramatic and important as epigenetic trends might continue during perhaps the next hundred years if Storfer's claims prove to be right I still do not see these trends as providing any ultimate escape from Lynn's conclusions. If we pretend to be at all concerned about the condition of humanity in distant future generations, the themes of Lynn's *Dysgenesis* still need to be read and understood. If we want to reach a distant future still with our special status as the world's most rational and constructive being, and if we wish to arrive there other than by a series of painful catastrophes (in the course of which we will see most of what we hold dear and try to conserve at the moment swept away, and this happening both in the way of our material treasures and our treasures of human character) then we need to think of ways to correct the trends he describes. It will not be easy but must be attempted. On the side of success, fortunately a majority of people do actually want intelligent offspring. Thus it is still possible to imagine the feat will be achieved, once the confusing disinformation of the demagogues has been set aside, *still within democratic procedures*.

Two other loosely connected matters remain, in the one case arising and in the other conspicuously not arising, in Lynn's book, that I would like to discuss. One is Lynn's chapter on dysgenesis in health and the other is his missing chapter on where all the variation for the heritability he so emphasises can be coming

from. Both these fill or should fill gaps between the ideas already covered. Lastly, attention certainly deserves to be given to Lynn's separate but important topic of the dysgenesis of conscientiousness. In some ways it is the most fascinating and serious of all the themes of the book. However I have to avoid this one: Lynn, covering it in three chapters towards the end of his book, does an excellent job with the facts. To comment usefully would require more long digressions both on deleterious mutation and on sociobiology.

His coverage of dysgenesis of health, all in just one chapter, is, in my opinion, not very adequate. It is shown that a number of well characterised genetic diseases are under at least a weak dysgenesis in the sense that we are medically curing them and thus allowing their recessives (and sometimes heterozygous conditions too) a cessation of the counterselection through death or disability that formerly kept the genes at very low frequency. The mutation-selection balance has been changed in their favour. One of the book's few graphs suggests severe mental disability has increased something like threefold in Denmark between 1888 and 1979 – an alarming implication if true, but not much is adduced to back up the idea that this is due to changes in the Danish gene pool as opposed to changes in detection, categorisation, etc. I believe [and have written (Hamilton, 1965; Hamilton, 1988; Hamilton, 1991)] that the changes Lynn points out are serious and that cumulatively they will eventually even become destabilising for civilisation. But I also believe these conspicuous cases of genetic disease, plentiful as they may be (Lynn mentions a recent count of about 4000) are far from the whole story about what we are doing to ourselves, and that actually there is a bright side to this gloomy cloud too. Indeed it is so bright that it might conceivably show an escape, again, from his central paradox. Lynn's oversight of this seems indexed in his quick dismissal of infectious disease and resistance to it, with these playing no important part in the chapter.

Suppose the cystic fibrosis gene is at its present frequency (generally admitted too high to be due to mutation alone) because it provides resistance during some kinds of intermittent epidemic. For the present argument let us accept the idea that the mucus of the condition, although becoming habitat to pathogenic mucus-specialising microbes, at times protects against the other pathogens that prefer to reach the epithelial cells directly and are more dangerous. *Vibrio cholerae* and *Salmonella typhi* have been suggested; the mechanism is probably more subtle than being checked by mucus but that will not affect the argument (Pier *et al.* 1998). The cystic fibrosis gene in single dose then, while not ideal, may provide a degree of protection when diseases such as deadly cholera and typhoid strike. Given further that such epidemics are erratic, the cystic fibrosis gene may exist, or have existed, in an ever-changing low frequency for a long time. Most of that time, through segregation and the chance unions that form the lethal homozygote, it is descending in frequency; sometimes however it sharply rises under the positive selection of heterozygotes in an epidemic. Next suppose that this is just one of a multitude of such cases of intermittently favoured and disfavoured genes concerned with resistance. The pathogens (and even the quasi-mutualists) against which genes of these other unknown polymorphisms help may be under much milder selection than genes for resistance to cholera and typhoid. This may be partly why they are harder to detect and few are yet known. Due to the intrinsic instability of all host-parasite population interactions (Eshel & Akin, 1983), all these various conditions are likely to have gene frequencies oscillating over generations to some degree while due to linkage such oscillations are bound mutually to interfere. A model that tries to put together the effects of a number of quasi-independent oscillating gene frequencies of this kind is frequency dependent and, it seems, allele protection against extinction in such a system is assisted by moderate to tight linkage (Hamilton, 1993). Recombination in this system thus (a) facilitates flexible response to pathogens, (b) maintains its base-polymorphism – and (c), from (a) and (b), maintains sex even when mutations to asex are possible (Hamilton *et al.* 1990). The important outcome for the present theme, however, is simply the variability of general health. Given that most of the time it is not good to experience even minor heterozygous effects of a gene like that for cystic fibrosis, we can expect a final steady-state health result for the population as a whole that is, as usual, a *polygenic bell-shaped distribution*. This is exactly what we generally observe. Accessory to this curve, of course, will go other 'bell' distributions for many associated characters including

stature, muscularity, and, for sure, given the expensiveness of the trait, intelligence.

The crux yet again is that brains are costly for maintenance: 20% of our metabolism – so much energy, all the time – do we really need this big thing? One line trying to explain the surprising size and estimate is that large brains may have come to human heads through the same evolutionary mechanism that brought the exterior wattles, crests and colours to heads of crowned cranes, and antlers to the heads of reindeer, horns to ibex. They have appeared through sexual selection as displays providing, through costliness, un-bluffable signals of a bearer's standing in 'good-genes' (Hamilton, 1975; Hamilton & Zuk, 1982; Hamilton, 1990). If so, what we know of parallel and well studied animal systems leads on to a notion that high cortical development in humans will only be only expressed in so far as requirements for other more vital human physiological systems are satisfied. It will be as if some extra channel opens increasingly to allow high cortical development once the basic brain that sees to daily needs has been constructed.

This idea is a bit akin to imagining a person's IQ projecting from their skull like the horn of an *Oryx*. (Measure it? Of course, that's easy – but go at it from the side, with caution!) Horns, as expected for extreme products of sexual selection, are indeed very variable but an obvious objection to the parallel applying particularly to the sexual selection that may underlie the upward tail of the IQ distribution, is that, while horns fit well with a adolescent overspill idea, brain development comes much too early. It occurs long before the growing foetus or juvenile could reasonably guess at what level of health, and hence surplus, it is going attain. However, there is more than one escape from this objection. One has already been hinted at in my speculations on social epigenesis.

The whole idea of sexually selected characters being revealers of health in respect of infectious disease, and hence revealers of 'resistance genes' against such disease (Hamilton & Zuk, 1982), is predicated on the idea that the oscillations in usefulness and therefore frequency of alleles like those determining cystic fibrosis are sufficiently long (period > 2) to lead substantial positive autocorrelation in fitness over generations. Such autocorrelation or heritability (as viewed with some surprise hitherto; the disease perspective having been neglected) is actually what is observed. Thus it is certain both from the model and by the facts, that the healthy individual's offspring will indeed be above average in health, and that even in a random mating system advantage should persist for several generations to come. In this argument sexual choosiness is not the only outcome; it will also be unsurprising if human mothers and fathers are adapted to bet on the outcome to the extent of having evolved ways to imprint offspring, where possible, with patterns that either accentuate or diminish brain development in preparation for expectable adaptive lifestyles. If a long established heritability of 'true fitness' – that is, of health – has fuelled the development of various sexual ornaments in other animals [the connection of these with 'good genes' is well established in many cases (Möller, 1994; Grob, Knapp *et al.* 1998; Petrie & Kempanaers, 1998; von Schantz, Bensch *et al.* 1999) but is not always strong (Möller & Alatalo, 1994)] there should be no surprise to find an additional epigenetic step connecting a more utilitarian feature – cognitive ability – within a similar environmental autocorrelation. We know small organisms do it for certain features; why should big ones not, especially large but weedy opportunists such as us? Daphnia water fleas facilitate 'helmets' for offspring when reliable environmental cues, which may include (but don't have to derive from) actual presence of predators, 'tell' them that helmets will be valuable defence for their offspring; wild radishes similarly switch on, seemingly, extra toxin (Agrawal *et al.* 1999). Many characters featuring epigenetic inheritance are probably prepared for in a previous generation in a similar way (Jablonka & Lamb, 1995).

We here find ourselves reviving parts of the argument already given about Neodarwinian epigenesis as a step in resolving Lynn's paradox. There is a very important difference in the present tack, however: into the topic we have brought a very broad explanation for the underlying variation and one that makes it virtually impossible for this variation to be selected away. Improved nutrition and condition of life may indeed underlie the mass upward movement of IQ. Lynn is still right that this must reach limits – this for the same reason that optimal nutrition and even antibiotics provided to male deer will not create antlers of unlimited

size. But in the new view the implication of heritabilities, even those calculated from monozygotic twins reared apart, may turn out a little less dire than he thinks. If the whole astonishing sweep of human intelligence from genius to moron is indeed based on such a shifting sand of genes promoting health as I have suggested – that is upon alleles which even switch their '+' and '−' signs every so often as the various pathogens and their pathotypes rise and fall – and if the switches, working through degrees of health, affect IQ as well, we may end with less cause to worry about the seeming *long term* implication of the class fertility differentials. In a nutshell, the differentials may be indeed, on the scale of the next hundred years, working against both average health and average IQ; yet in spite of this, on the scale of millennia, they may not matter. In the present view nothing is irrevocably lost to the human population whether the paradoxical downward steps predicted by Lynn's analysis continue or not. However (a) the short term effect in its making us on average less able to lay out and enjoy the riches of human thought is still obviously regrettable, and (b) it is likely that not all of the variation is of such a deceptive developmental or epigenetic kind as I suggest. We have no idea at present how much might be, or really whether any is. Presumably some more fundamental mutations affecting human brain development are in transition in human populations. In so far as they are, it remains true Lynn's differentials are beating them back and they are also worsening the state of any mutation-selection balance.

There remains yet another slightly differing escape from the paradox that deserves brief attention. In the mid 1900s Lionel Penrose, as if already sensing that mass IQ was not in such regress as the predictions foretold, offered a model to explain how decline might not occur even while one admitted IQ to be largely genetic and thus, at least in the broad sense, heritable (Penrose, 1955; Penrose, 1961). He had two alleles at a single locus in polymorphism heterotic for fertility but monotonic for intelligence and such that there is an overall negative association. Lynn covers Penrose's idea but points out that '[it] does not match the evidence which indicates a linear decline in fertility with rising intelligence'. Perhaps Penrose had infertile extreme geniuses in mind; or perhaps (and not exclusive to such an idea) he believed in some kind of a differentiation in families – a social-insect-like pattern. Contra Lynn, I see some hope for the idea in either of these ways provided we extend the number of loci and as in my last discussion bring in again epigenetic inheritance and parental physiological decisions – in short a kind of compote of all I have discussed. Again I refer to Sulloway's human-brothers review which has shown rather clearly that special different life strategies within human families exist (Sulloway, 1996). We may expect the effect he found to repeat to a limited degree within small inbred groups such as we believe to have existed over most of our past in the palaeolithic. A global model based on changeable polygenes for health such as I have suggested, couldn't yield a stable outcome for IQ if 'intelligence monotonic upwards' goes with 'fertility monotonic downward', class by class; however, it is possible that the model could stabilise as Penrose's did or even be globally eugenic, if negative associations existed in all of a set of small groups in a metapopulation while the differential rates of reproduction between the groups were sufficient with more expressed intelligence in a group meaning faster growth. The idea here is a bit like that of the groups of dry-wood termites differentiating their 'soldiers' (the first evolved caste in termites) and their young queens, with 'intelligence' imagined as the group-beneficial analogue parallel to the defence trait of the termite soldiers. We know that the families of termites manage to differentiate rather precisely the proportion of soldiers that each group's current size requires (Harris, 1961) (this had even been noted in work by Grassi in Italy in the last century). Given Sulloway's finding of fairly distinct pathways existing in human family development, then rather as if we were seeing in our own families a faint delineation of a future caste, or at least a dispersal morph, it is possible to imagine celibate human genius as just the extreme of a more modest normal human differentiation. Children (and perhaps, as suggested above, even foetuses), that find themselves far out in the local distribution of the set of health 'polygenes' that particularly have side effects on neuronal development [say through molecular mimicries of pathogens to neuronal epitopes (Brown, 1997)] might tend to specialise in intellectual development to the benefit of their family or group while at the same time reducing their drive towards individual reproduction.

Inclusive fitness/group selection arguments (which are basically equivalent) could easily 'justify' such courses.

Such indirect adaptive outcome may have played an evolutionary part in the ready acceptance of 'intellectual babies' rather than real ones that seems indeed to occur, much on Penrose's lines, in the extreme upper tail of the distribution of human IQ. Unfortunately whether or not such an effect did in the past help to stabilise levels or was actually eugenic, it cannot provide us with an optimistic view of the present situation. Lynn's fertility differentials are already often averaged from massive and heterogeneous samples and also involve comparisons outside these groups, such as comparing Africans with Europeans; all, however, still show the differentials leaning the same way. This means that at least for the present such an effect is not working, and one may wonder, with individualism as strong at it is in humans, whether such kin-group selection ever could have been important enough. Looking back only a few centuries, however, to the new expansions of population that came about immediately after the great age of exploration, with this in part having being permitted through applications of the efforts of dedicated, infertile European intellectuals [technicians, mathematicians, scientists as described striving to improve global navigation by Sobel (1996)] we see that the present situation may be anomalous. To my mind the facts don't rule out effects like Penrose's as modified here having been important, nor rule against their becoming so again in the future. The idea of genetic symbioses of aptitudes existing in human societies deserves much more modelling attention than it has had. Contra Sober and Wilson (Sober & Wilson, 1977) kin relatedness in such models is very unlikely ever to be absent; however, apart from this caveat, the group selection perspective that these authors prefer on human evolution can certainly be used; a lot of the gene-frequency changes in humans may indeed have been accountable in the past to small group levels (Hamilton, 1996). Castes within groups, like the seeming genetic colour-vision specialists comprised in New World monkey groups, seem a possible example. It must be remembered that even if these formed specialist endogamous castes within groups, like castes within Indian villages, and even if between such castes there was no relatedness, the support and permissiveness accorded by one caste to another can be considered, in so far as the castes are interdependent, as being still a kind of kin altruism directed to descendants – everyone benefits if the 'colour-blind' monkey specialists or the human sweeper caster continue to be present in the troupe/village so that being kind to them helps your descendants. In any case along with these endorsements to 'group selection' in human evolution has to go a warning that using the notion certainly doesn't invoke any more of a 'liberal utopian' mind set among humans than individual or kin-selection does; if anything deeds done in the name of groups have been more illiberal and awful than acts of normal selfishness or nepotism (Hamiliton, 1996).

This ends my list of suggestions of how Lynn's central paradox of his book might be resolved either completely or made to disappear in temporary ways that are different from the suggestion Lynn himself makes. Most of my ways are admittedly very speculative: however the paradox itself is such a gaping hole and so important for our future, it has seemed to justify me, a jackdaw on the chimney pot, throwing down a variety of sticks in the dark, hoping at least one of them will lodge and start a platform for a nest – down there perhaps it may make combination with that stick which Lynn himself has more securely placed in his brave and fertile book.

REFERENCES

Agrawal, A. A., Laforsch, C. & Tollrian, R. (1999). Transgenerational induction of defences in animals and plants. *Nature* **401**, 60–63.

Baker, R. R. & Bellis, M. A. (1995). *Human Sperm Competition*. London: Chapman and Hall.

Brown, P. (1997). Over and over and over.... *New Scientist* **155**, 27–31.

Butler, B. E. & Petrulis, J. (1999). Some further observations concerning Sir Cyril Burt. *British Journal of Psychology* **90**, 155–160.

Cerda-Flores, R. M., Barton, S. A., Marty-Gonzalez, L. F. *et al*. (1999). Estimation of nonpaternity in the Mexican population of Nuevo Leon: A validation study with blood group markers. *American Journal of Physical Anthropology* **109**, 281–293.

Eshel, I. & Akin, E. (1983). Evolutionary instability of mixed Nash solutions. *J. Math. Biol*. **18**, 123–133.

Eysenck, H. J. (1995). Burt as hero and anti-hero: a Greek tragedy. In *Cyril Burt: Fraud or Framed?* (ed. N. J. Mackintosh), pp. 111–129. Oxford: Oxford University Press.

Fitch, W. M. & Atchley, W. P. (1985). Evolution of inbred strains of mice appears rapid. *Science* **228**, 1169–1175.

Fletcher, R. (1991). *Science, Ideology and the Media: The Cyril Burt Scandal*. New Brunswick, USA: Transaction Publishers.

Francis, L. (1979). Contrast between solitary and clonal lifestyle in the sea anemone. *Anthopleura elegantissima*. *American Zoologist* **19**, 669–681.

Gould, S. J. (1996). *The Mismeasure of Man*. London: Penguin Books.

Grob, B., Knapp, L. A., Martin, R. D., *et al*. (1998). The major histocompatibility complex and mate choice: inbreeding avoidance and selection of good genes. *Experimental and Clinical Immunogenetics* **15**, 119–129.

Haig, D. (1993). Genetic conflicts in human pregnancy. *Quarterly Review of Biology* **68**, 495–532.

Hamilton, W. D. (1965). (Human Diversity. K. Mather). *Population Studies* **19**, 203–205.

Hamilton, W. D. (1975). Gamblers since life began: barnacles, aphids, elms. A review. *Quarterly Review of Biology* **50**, 175–180.

Hamilton, W. D. (1988). Sex and disease. In *Evolution of Sex*. (ed. G. Stevens & R. Bellig). San Francisco: Harper and Rowe.

Hamilton, W. D. (1990). Mate choice near or far. *American Zoology* **30**, 341–352.

Hamilton, W. D. (1991). Seething genetics of health and the evolution of sex. In *Evolution of Life. Fossils, Molecules, and Culture*. (ed. S. Osawa & T. Honjo), pp. 229–251. Berlin: Springer.

Hamilton, W. D. (1993). Haploid dynamical polymorphism in a host with matching parasites: effects of mutation/subdivision, linkage and patterns of selection. *J. Hered*. **84**, 328–338.

Hamilton, W. D. (1996). *Narrow Roads of Gene Land*. Volume 1. Oxford: Freeman/Spektrum.

Hamilton, W. D., Axelrod, R. & Tanese, R. (1990). Sex as an adaptation to resist parasites. *Proc. Natl. Acad. Sci., USA* 87, 3566–3575.

Hamilton, W. D. & Zuk, M. (1982). Heritable true fitness and bright birds: a role for parasites? *Science* **218**, 384–387.

Hamilton, W. V. (1961). *Termites. Their Recognition and Control*. London: Longmans, Green and Co. Ltd.

Harris, W. V. (1961). *Termites. Their Recognition and Control*. London: Longmans, Green and Co. Ltd.

Herrnstein, R. J. & Murray, C. (1994). *The Bell Curve: Intelligence and Class Structure in American Life*. New York: The Free Press.

Jablonka, E. & Lamb, M. J. (1995). *Epigenetic Inheritance and Evolution: The Lamarckian Dimension*. Oxford: Oxford University Press.

Jacobs, G. H., Neitz, M., Deegan, J. F. *et al*. (1996). Trichromatic colour vision in New World monkeys. *Nature* **382**, 156–158.

Jollos, V. (1921). Experimentelle Protistenstudien. 1. Untersuchungen über Variabilität und Vererbung bei Infusorien. *Archiv. für Protistenkunde* **43**, 1–222.

Jollos, V. (1934). Inherited changes induced by heat treatment in *Drosophilia melanogaster*. *Genetica* **16**, 476–494.

Joynson, R. B. (1989). *The Burt Affair*. London: Routledge.

Karn, M. N. & Penrose, L. S. (1951). Birth weight and gestation time in relation to maternal age, parity, and infant mortality. *Annals of Eugenics* **15**, 206–233.

Lucas, P. W., Darvell, B. W., Lee, P. K. D. *et al*. (1998). Colour cues for leaf food selection by long-tailed macaques (*Macaca fascicularis*) with a new suggestion for the evolution of trichromatic colour vision. *Folia Primatologica* **69**, 139–152.

Lynn, R. (1990). New evidence on brain size and intelligence: A comment on Rushton and Cain and Vanderwolf. *Personality and Individual Differences* **11**, 795–797.

Mackintosh, N. J. (1995). *Cyril Burt: Fraud or Framed?* Oxford: Oxford University Press.

Möller, A. P. (1994). Male ornament size a reliable cue to enhanced offspring viability in the barn swallow. *Proc. Natl. Acad. Sci. USA* **91**, 6929–6932.

Möller, A. P. & Alatalo, R. V. (1994). Good-genes effects in sexual selection. *Proceedings of the Royal Society of London B* **266**, 85–91.

Penrose, L. S. (1955). Evidence of heterosis in man. *Proceedings of the Royal Society of London B* **144**, 203–213.

Penrose, L. S. (1961). Genetics of growth and development of the foetus. In *Recent Advances in Human Genetics* (ed. L. S. Penrose), pp. 56–75. London: J & A Churchill.

Petrie, M. & Kempanaers, B. (1998). Extra-pair paternity in birds: explaining the variation between species and populations. *Trends in Ecology and Evolution* **13**, 52–58.

Pier, G. B., Grout, M., Zaidi, T., *et al*. (1998). *Salmonella typhi* uses CFTR to enter intestinal epithelial cells. *Nature* **393**, 79–82.

Rose, S. Lewontin, R. C. & Kamin, L. (1989). *Not In Our Genes*. New York: Pantheon.

Rushton, J. P. (1994). The equalitarian dogma revisited. *Intelligence* **19**, 263–280.

Rushton, J. P. (1995). *Race, Evolution and Behaviour, a Life History Perspective*. New Brunswick, USA: Transaction Publishers.

Shyue, S. K., Hewett, E. D., Sperling, H. G. *et al*. (1995). Adaptive evolution of colour-vision genes in higher primates. *Science* **269**, 1265–1267.

Sobel, D. (1996). *Longitude*. London: Fourth Estate.

Sober, E. & Wilson, D. S. (1997). *Unto others: The Evolution of Altruism*. Cambridge, MA: Harvard University Press.

Storfer, M. (1999). Myopia, intelligence, and the expanding human neocortex: Behavioral influences and evolutionary implications. *International Journal of Neuroscience* **98**, 153–276.

Sulloway, F. J. (1996). *Born to Rebel. Birth Order, Family Dynamics and Creative Lives*. London: Little, Brown.

von Schantz, T. Bensch, S. Grahn, M. *et al*. (1999). Good genes, oxidative stress and condition-dependent sexual signals. *Proceedings of the Royal Society of London B* **266**, 1–12.