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Genetic fallacies

We used to think our fate was in our stars. Now we know, in large measure, our fate is in our genes.

James Watson, winner (with Francis Crick and Maurice Wilkins) of the 1962 Nobel Prize for Medicine, for discovering the structure of DNA

In previous chapters, I have traced and analyzed the debates over the importance for morality of our evolutionary past by reference to Darwin's theory of natural selection. There is a sense, however, in which this is a one-sided approach. Darwin's work was certainly one of the most impressive and momentous in the history of science, but it was also critically incomplete. As Darwin saw, the theory of natural selection has three fundamental elements: there must be *differences* between organisms of the same species, as a result of which some are *fitter* than others and therefore tend to have more offspring, and these differences must be *heritable*, so that these offspring tend to possess the traits that made their parents fitter. Without heritability, random differences could not accumulate, and speciation could not occur. Evolution is the story of how life on Earth, in all its fantastic diversity, is descended from very simple organisms. But such descent requires that a fine balance be struck between the preservation of traits from one generation to another – so that offspring tend to resemble their parents, and species can be identified – and slow alteration in traits, so that mutations with differential fitness can arise. Though Darwin saw the need for such a mechanism, he had no credible theory to account for it.

It was largely because of his failure on this score that, though most biologists soon accepted evolution, the mechanisms that Darwin proposed were largely rejected. Scientists looked to other evolutionary explanations. All this changed with the rediscovery of the work of an obscure Austrian monk, Gregor Mendel. Mendel had conducted experiments in the breeding of peas, in which he crossed one variety with another. He noticed that some traits appeared to be *dominant* over others, in the sense that if you crossed a variety with one trait with a variety with another, all the plants in the second generation would have the dominant trait. Upon first inspection, it might seem that the *recessive* (as we now say) trait had been “bred out” altogether. But re-crossing the plants in the second generation with each other showed that this was not the case: the recessive trait reappeared in the next generation! Just what was going on?

Mendel realized that the ratio in which traits appeared was an important clue: the recessive trait appeared in around twenty-five percent of the plants in the third generation. This discovery allowed him to guess at the nature of the underlying process. Somehow, inheritance must involve passing on half the properties of parents to their offspring. That much of course, Darwin had known, but he hadn’t been able to explain why traits were not “washed out” in the process. Take Mendel’s own subject, the garden pea. The pea comes in round-seeded and wrinkle-seeded varieties, which Mendel crossed with each other. If offspring inherit from each parent equally, then shouldn’t we expect the second generation of plants to have had seeds that were half-way between the round-seeded and wrinkle-seeded variety – a little wrinkled, perhaps? But that didn’t happen – the entire second generation consisted of round-seeded plants. And the third generation was made up of round-seeded peas and wrinkle-seeded peas in the ratio of 3:1. The traits were not blended, but inherited in stable forms.

Mendel explained the pattern of inheritance by postulating that the appearance of traits was controlled by some heritable element that comes in *pairs*: one from each parent. This, he saw, would explain both why the second generation consisted exclusively of round-seeded plants, and also the ratio in which wrinkle-seeded peas appeared in the next generation. Suppose that the plants in the first generation each had two copies of the same heritable factor (we call them genes), and

that one of these factors is dominant over the other, so that even if a plant receives just one, it will possess the trait associated with that factor. In that case, crossing two different *homozygous* plants – plants that possess two identical varieties of a particular gene – would produce the following results. (In this schematic representation, “*R*” represents the allele for rounded seeds, while “*r*” represents the allele for wrinkled seeds. An *allele* is one of the possible variants of a gene – in this case, wrinkled or rounded):

First generation	RR	rr
		↓
Second generation	Rr	Rr

Since the offspring inherit one gene for seed shape from each parent, and the parents are homozygous for the gene, then no matter which gene they get from which parent, each plant in the second generation ends up with one copy of each allele (the plants are *heterozygous* for that gene). But since the gene for rounded seeds is dominant, each plant *expresses* only that gene: it has rounded seeds.

In the next generation, however, the phenotype for wrinkled seeds reappears:

Second generation	Rr	Rr		
		↓		
Third generation	Rr	RR	rR	rr

Each plant inherits one allele from each parent. Thus, half the plants get the *R* allele from the parent on the left, and half the *r* allele; half get the *R* allele from the plant on the right, and half the *r* allele. As a result, one of the plants is homozygous dominant (*RR*) for seed shape and two, like the parents, are heterozygous (*Rr*). Since *R* is dominant, all three plants have rounded seeds. But one plant is homozygous recessive (*rr*). Since there is no dominant (*R*) present, the recessive phenotype is expressed – the plant has wrinkled seeds.

Mendel’s theory not only explained the ratios in which traits were inherited, but also why heredity did not wash away differences. Since the underlying mechanism was a factor that was passed on as a whole,

the trait associated with it either appeared full-blown, or not at all. Mendel's careful experimentation had revealed the underlying mechanism of heredity. When Darwinism made its triumphant comeback in the early years of the twentieth century, it called upon Mendelian genetics to fill the gaps in explaining inheritance. It was not Darwinism, as Darwin himself had elaborated it, which swept biology, but what came to be known as "the new synthesis," or Neo-Darwinism. The genetic perspective allowed biologists to understand a great many phenomena that had been mysterious to evolutionists who lacked the mechanism provided by Mendel.

Indeed, I have invoked the genetic perspective at crucial points in my analysis of the evolution of morality. For example, the notion of inclusive fitness, which underlies kin altruism, only makes sense in the light of Mendelian inheritance. But we need to take fuller cognizance of the role of the gene. When people invoke biology as a means to understand human behavior, to explain, or explain away, ethics and responsibility, it is most often genes they have in mind, not evolution. It is time to examine the other half of the new synthesis, the mechanism of inheritance itself.

The genetics of behavior

What do genes actually *do*? The most common answer is that they are a blueprint, or perhaps a program. They contain the instructions for making organisms. Francis Crick, who, with James Watson, made the breakthrough discovery of the double helix structure of deoxyribonucleic acid (DNA), the substance out of which genes are composed, put it this way, "DNA makes RNA [ribonucleic acid, which carries the genetic instructions to the parts of the cell where proteins are made], RNA makes protein, and proteins make us."¹³⁷ Thus, our genome is the blueprint that our cellular machinery follows in constructing us. The genome contains the set of plans for building an organism.

When we are engaged in the project of understanding human behavior and morality, this seemingly technical matter is very important. For the way the blueprint metaphor is usually understood, our genes shape, control, or at very least constrain, what it is we are able to

do, think, and desire. Our shared genome explains what we have in common. Equally, our differences are to be explained in terms of the degree to which our genes differ from one another – "it's in the genes," we are often told. More and more, the medical profession looks to the genome to understand disease, and to learn how to fight it. Increasingly, we can test for genes that indicate susceptibility to cancer: for example, possession of the gene BRCA 1 raises a woman's lifetime risk of breast cancer to fifty percent or more. And it is not just our physical characteristics which genes are supposed to explain. Psychological traits and dispositions are also widely held to be under genetic control. In the past few years, researchers have made an astonishing array of claims about the genetic origins of complex behaviors:

1. A disposition to violent behavior is widely believed to have a genetic cause. For example, Terrie Moffitt, from the Institute of Psychiatry at King's College London, claims to have isolated what, on one interpretation, are "genes for violence."¹³⁸
2. A disposition to laziness might have a genetic basis. Professor Susan Ward of Glasgow University predicts that a gene for laziness will be found. She claims that understanding the genetics of laziness should transform our attitude to people who suffer from this condition: "People who don't like exercise are usually seen as lazy, but it may be that it's not their fault. There may be a link between exercise intolerance and genetic make-up which restricts or promotes exercise depending on your genes."¹³⁹
3. Intelligence is very frequently thought to be largely genetic. Professor Gerald McClearn argues that "IQ genes" play a crucial role in determining our cognitive ability.¹⁴⁰ Pinker's *The Blank Slate* is largely devoted to buttressing the case for a genetic basis for IQ.
4. Sexual orientation is in the genes. The discovery of the "gay gene" was widely hailed in the world's newspapers.¹⁴¹ Dean Hamer, the scientist whose research caused this stir, was himself more careful. He claims only that our genes "influence" sexual orientation, not that they *determine* it. Nevertheless, he describes his search, through linkage studies, for the gene or genes which play this role as "Looking for Gay Genes."¹⁴²

We could continue to multiply examples of claims of genes for phenotypic traits: sporting or artistic ability, dyslexia, autism, psychopathy, depression, binge eating, introversion – all of these, and many more, are sometimes said to be caused (or, at a minimum, influenced) by our genes. The language of the genes, and the message of genetic determinism, has been absorbed into our culture. From *The X-Files* to *The New York Times*, our media repeat to us the message that our destiny is in our genes.

Genethics

If we fail to consider the implications of the new genetics for morality and human behavior, we risk leaving a large hole at the heart of our inquiry. We cannot hope to understand the implications of evolution if we don't explore and account for the role of its principal mechanism, the mechanism that rescued Darwinism from obscurity. Moreover, many of the challenges we have faced thus far, especially under the guise of the evolutionary explanations of human psychology, reappear as more narrowly genetic challenges to our ability to elaborate an adequate account of human morality and freedom. It may be, for instance, that we can puncture the theories of sex differences offered by evolutionary psychology, or counter the claim that there are viable and informative explanations of rape as an adaptation. No matter, critics might claim: whatever the explanations for how certain people come to have "genes for violence," or "genes for nurturing," the fact is that they have them. The evidence is overwhelming: in their desires and their dispositions, people are driven by their genes.

If the more lurid claims of the genetic determinists are true, we shall need to rethink our morality and our politics from the ground up. Recall Susan Ward's contention that a gene for laziness might soon be found, and the implications she draws from its possible discovery. If people have a gene for laziness, then their idleness is "not their fault," she tells us. It's in the genes, and what is in the genes we cannot help (and we are never responsible for what we can't help). On the face of it, this seems a reasonable claim. Some people are born with disabilities that are the result of genetic defects, such as cystic fibrosis, which is

caused by a mutation of a gene on chromosome seven. It would be grotesque and immoral to blame someone for being born with cystic fibrosis. They couldn't control their genetic make-up, and we aren't responsible for what we can't control. Similarly, we might think, we cannot blame someone for being born with a gene for psychopathy. And if possession of the gene leads inexorably to the behavior associated with it, then we can't blame them for the behavior either.

Perhaps we can avoid this unfortunate conclusion by recalling that genetic determinism is false. Every reputable scientist working on the genetics of behavior denies that they are committed to genetic determinism. Thus, we might think, the door is opened for the admission of responsibility. Actually, it is not clear that denying determinism admits responsibility. When a scientist repudiates genetic determinism, she has one (or both) of two quite separate and independent claims in mind:

1. The association between the gene and the behavior is statistical: those with the gene have a higher probability of exhibiting the behavior than those without. Thus, not everyone who has the gene will exhibit the behavior, and not everyone who exhibits the behavior will have the gene.
2. Human beings are, sometimes at least, able to resist the influence of their genes upon their behavior. Thus, someone who possessed a hypothetical gene inclining him toward rape could, through sheer will-power, refrain from raping, because he knows that rape is immoral.

Anyone who believes either (1) or (2) holds that genetic determinism (understood as the claim that the presence of gene x invariably leads to the organism exhibiting trait y) is false. But (1) and (2) have quite different implications for human freedom and morality.

All scientists subscribe to (1), or something like it. Every geneticist knows that genes do not cause behavior, or indeed anything else, except in a particular environment, and that the details of that environment can make a crucial difference to the trait shaped by the genes. Thus, Terrie Moffit's research on the genetics of violence, which I previously mentioned, claims that the genetic mutation responsible for making young men violent is triggered by childhood maltreatment. By itself,

the gene doesn't cause violent behavior; instead, its effects are crucially mediated by the environment. If a child who has this mutation grows up in a stable and happy home, he may exhibit no unusual propensity to violence. However, if he is mistreated he is more likely to turn violent than is a similarly treated child who lacks the mutation.

Gene-environment interactionism is certainly true. But it does not answer all our questions about the moral and political implications of genetics; it just raises further questions. We can divide these questions into two kinds: questions about free will and moral responsibility, and questions about political and social policy.

As far as the first set of questions is concerned, the problem is this: the fact that our genes do not cause our phenotypic traits by themselves, but require an environment in which they are expressed, doesn't change the fact that our traits are nevertheless caused. *All* causes have their effects mediated in this way. Striking a match normally causes a flame, but this effect is dependent upon the appropriate conditions being present: there must be a high enough proportion of oxygen in the air, the match must not be (too) wet, and so on. The fact that interactionism is true does not make genes any less deterministic causes of phenotypic effects. Interactionism doesn't give individuals any more control over their traits than genetic determinism (narrowly understood) does, since we do not choose our formative environments any more than we choose our genes. The child who becomes a violent adult as a result of a genetic mutation *and* maltreatment does not seem more responsible for his violence than a (counterfactual) child who becomes violent as the result solely of a genetic mutation.

This set of questions is very, very hard. Fortunately, we can largely ignore them, on the grounds that genetics raises no *special* problems for free will and moral responsibility. Our behavior seems just as much determined whether it is determined by environment alone or by genes alone, so whatever the influence of genes, exactly the same set of issues confronts us. As many philosophers have pointed out, it seems that if our behavior wasn't caused deterministically at all, this would do nothing to restore our freedom. Random behavior, or behavior caused indeterministically (perhaps by the indeterminate decay of quantum particles) seems no freer (perhaps less free) than behavior that is deterministically caused. Genetics is therefore no particular threat

to free will: we have or lack it regardless of the truth of genetic determinism.¹⁴³

That is not to say that we can simply ignore genetics when assessing the moral responsibility of particular individuals. We already take into account people's capacities, and the burdens under which they labor, when we assess moral responsibility. We don't blame young children or the insane for crimes, because we believe that they can't sufficiently understand what they're doing. We also excuse, wholly or partially, people who act under duress: the woman who steals a loaf of bread to feed her starving children is dealt with less harshly than the person who doesn't need what she steals. We need to know whether genes can influence our capacities, or (more likely) place special burdens upon us. It might be, for example, that some people find it more difficult to control themselves because, as a result of their genetic endowment, they have lowered levels of serotonin (a neurotransmitter) in their brain. If this turns out to be true, then perhaps it should diminish their responsibility for their actions.¹⁴⁴

So, despite the fact that genetics doesn't constitute a new threat to human freedom, we still need to understand the precise nature of its influence upon our behavior. This brings us to the second set of questions, concerning social and political policy. Interactionism does not settle any questions here, because we need to know how powerful is the influence of our genes on our traits. It might be, for instance, that the effects of genes (or at least of some genes) are robust, in the sense that they will show up in the same way across almost all environments. Or it might be that we can modify these effects, but not in ways we would like: we can easily modify human intelligence, using crude environmental interventions like drugs or child abuse, but it seems much easier to bring intelligence down than up. Perhaps this is the typical case: perhaps what natural selection gives us is optimal, or at any rate better than the result we could achieve by intervention. We should all be interactionists: we should all recognize that the traits we have are the result of our biological endowment and the environment in which it expresses itself. But recognizing that fact still leaves all the important work to be done. We still need to know the extent to which these traits can be modified, in directions we desire, by environmental interventions.

This question matters, because the extent to which certain types of socially desirable goals are achievable turns on it. If, for example, it turns out that current variation in intelligence is (1) significantly genetic and (2) unmodifiable (for practical purposes) by environmental interventions, then our political choices are greatly constrained. We should only be able to achieve substantial equality by sacrificing goods we value very highly. If we cannot raise the intelligence of the genetically less well endowed, then we might achieve equality by lowering the intelligence of the better off. But that would involve the sacrifice of a very large number of important goods, ranging from the social benefits that the efforts of the talented can bring all of us, to our prized freedom to develop our abilities. If so, perhaps we would be forced to give up on equality. Perhaps, indeed, we should come to think that any attempt even to limit inequalities is illegitimate. But, if we were to discover that the current distribution of intelligence could be altered through interventions which did not carry high costs, then we seem to be obliged to intervene. We cannot rest content with the recognition that interactionism is true. We need to investigate the extent to which intervention is practical and desirable.

There is a second way to deny genetic determinism. We can claim that whatever dispositions our genes may foist upon us, we have the power to resist them. Richard Dawkins sometimes seems to advocate such a view:

We have the power to defy the selfish genes of our birth and, if necessary, the selfish memes of our indoctrination. We can even discuss ways of deliberately cultivating and nurturing pure, disinterested altruism – something that has no place in nature, something that has never existed before in the whole history of the world. We are built as gene machines and cultured as meme machines, but we have the power to turn against our creators. We, alone on earth, can rebel against the tyranny of the selfish replicators.¹⁴⁵

In many ways this seems commonsensical. We all have urges that we do not act upon. Just for a moment, you find yourself ready to punch the driver who cuts in ahead of you. But you think better of it, and fight the urge down. Similarly, you might be genetically programmed for violence, or to eat sugar and fat. But you can control these dispositions,

learn to recognize the warning signs and take evasive measures, or refrain from getting into situations where you know you will not be able to resist (the prudent dieter does not keep chocolate ice cream in the refrigerator). What does it matter if our genes dispose us to one behavior or another? These dispositions are irrelevant to how we can and ought to act, and what we can realistically hope to achieve.

There are two problems for this view. Firstly, though it certainly seems true that we can often resist our desires, we can do so only as long as we have other resources of rational self-control available, which are equally the product of evolution. It may be that some people have fewer such resources, for reasons that can be traced back to their genes. Secondly, even if we are in the fortunate position of being able to rebel against (some of) our genes, our innate disposition will still impose costs and constraints upon us. We resist many of our urges only through effort and planning. The dieter who plans his day so as to avoid temptation imposes costs upon himself, in the form of forgone opportunities and limited spontaneity. Moreover, there is good evidence that self-control is a resource that can be used up: that even the strong-willed eventually give in if the temptations are repeated too often and too soon.¹⁴⁶ If people have strong, morally relevant, genetic dispositions, then we will need to take account of these desires in building our institutions, in our education systems, and in our courts and legislatures. The mere fact that they can sometimes be resisted (by whom? under what conditions?) does not make them morally irrelevant.

Most of the issues I have just sketched, to do with the extent to which genes influence or control behavior, have a close parallel with the questions raised by evolutionary psychology. In this case as in that, simply invoking the naturalistic fallacy is far from sufficient to prevent the claims of these sciences from being directly morally relevant. But there is also a second kind of problem raised by genetics, which has no parallel among those raised by evolutionary psychology. Genetics offers us unique, and uniquely troubling, opportunities not merely to predict human behavior, but to intervene in it.

This has raised the fear, in many quarters, of inequality of a kind and scale unprecedented in world history. Human genetic engineering, it seems, has the potential not only to cure diseases, but also to enhance a range of desirable characteristics, from intelligence to height. These

enhancements are likely to be expensive, and therefore accessible only to the wealthy. As a result, their children will be doubly advantaged over the less fortunate: not only will they have access to all the social goods which money can buy – the best schools, medical care, nutrition, and so on – but they will also begin with a better genetic endowment. If the genetic alterations that their parents provide are of the right sort (technically, if they are the result of germline intervention), then these advantages will be encoded in turn in the genome of *their* children. Generation after generation, the genetic stock of the wealthy might be expected to improve, and their comparative advantage over the less well off increase. Eventually, the purveyors of this scenario fear, the process might lead to the creation of a distinct species, of superhumans.

If this were allowed to occur, critics fear, the bonds of sympathy that connect each of us to every other would be cut or severely weakened. The better off would no longer feel that they have a reason to care more strongly about their fellow human beings than about members of other species; indeed, they might not even believe that they are of the same species. As Buchanan, Brock, Daniels, and Wikler express it, in their important overview of the implications of the new genetics for considerations of justice, “the effectiveness of people’s moral motivation to act consistently on universal moral principles may depend significantly upon whether they share a sense of common membership in a single moral community. But whether this sense of moral community could survive such divergence is a momentous question.”¹⁴⁷ The fear that genetic engineering could be used not just to cure (what we currently recognize as) diseases, but to create such superior beings, or at very least to exacerbate existing inequalities, seems to be the major impetus behind the widely shared intuition that we should distinguish genetic enhancement from treatment, and ban the former. This is an intuition shared across much of the political spectrum, from egalitarian liberals like Buchanan, to conservatives like Francis Fukuyama.¹⁴⁸ All fear that allowing widespread access to genetic enhancement would lead to massive inequality and social stratification.

The fear must be taken seriously. It may well be, for example, that democracy only works if inequalities in wealth and status are kept within certain limits. Greater inequality might lead to a breakdown of

our relatively free, relatively secure, societies. But is the fear realistic? Notice that many of the people who express it are well aware that interactionism is true, that genes have particular effects only within particular environments. They are implicitly suggesting that genetic inequalities are likely to result across the range of practically achievable or foreseeable environments. Are they right? And are they correct in focusing their fears upon genetic interventions? We shall need a much more detailed understanding of the manner in which genes interact with the environment before we can answer these questions, and assess the extent to which genes really constrain our behavior and therefore set the bounds within which our societies must learn to exist.

Genes “for” traits

The extent to which genetic engineering, of the sort that Buchanan et al. and Fukuyama fear, is a live possibility depends upon there being levers which can be manipulated to produce particular phenotypic effects. In the terminology that has pervaded the media, there must be *genes for* those traits. Of course there are such genes – aren’t there? Certainly, many scientists believe that there are: for example, Professor Robert Plomin claims to have isolated “the first specific gene for human intelligence.”¹⁴⁹ More careful, or more prudent, scientists talk instead of genes involved in, or influencing, intelligence, personality, and behavior. Some of these findings are well confirmed. Genes for cystic fibrosis, or for breast cancer, are well known, their variants are mapped, and the causal role they play in development is slowly being unraveled.

But things are not as simple as they appear. There certainly are “genes” the presence of which is predictive of certain outcomes. But in what sense are they *genes for* those outcomes? Imagine that your new car has a defect in it: it has a mechanical flaw in the axle that causes the front wheels to fall off. Would your mechanic look at the axle and remark that your car has a special feature, the function of which is to make its wheels fall off: a design feature *for* breaking down? Surely not. Analogously, it is unlikely we have *genes for* disease or cancer. Furthermore, in the case of your car, it is easy to see the causal relationship between the defect and the result: the axle breaks and the wheels

come off. But with a genetic defect, the relationship might be far more indirect. The gene for breast cancer might play a causal role in bringing about breast cancer. Or it might simply be *correlated* with a high risk of breast cancer. This, too, should make us hesitate before assigning a function to it.

Lenny Moss, a cell biologist turned philosopher, suggests that we can make sense of this talk of a gene for cancer, but only if we accept that it is *just* a useful way of talking, not something that picks out a real object in the world.¹⁵⁰ Moss distinguishes between two, equally legitimate, ways of talking about genes. We can talk about genes as Genes-P (for preformationist), or Genes-D (for developmental).¹⁵¹ A Gene-P is the kind of thing we have in mind when we talk about a gene for cancer (or for intelligence, or aggression). A Gene-P is a gene for a phenotypic trait. But a Gene-P is *not* a physical entity. In fact, Moss argues, the physical basis of a Gene-P is rather the *absence* of a physical entity. For example, I have the “gene for” blue eyes. But that does not mean that somewhere on one of my chromosomes is a sequence of DNA that has the property of causing me to have blue eyes. Rather, I have blue eyes because I *lack* the molecular structures which, in humans, result in brown eyes, and blue eyes is what we typically get when we don’t have brown eyes. Since there are many ways of *not* having a gene – you can have any number of alternative sequences in its place – having blue eyes is not correlated with any particular sequence of DNA. Moreover, the sequences of DNA that I have in place of genes for brown eyes do not necessarily play any sort of role in building my eyes. Similarly, if someone has a gene for breast cancer, she does not have a sequence that other people lack, which causes breast cancer. Rather she *lacks* a sequence, and therefore the ability to make a certain protein, which raises the probability that she will suffer from breast cancer. So a Gene-P, a gene for a phenotypic trait, is not a physical entity. But a Gene-D is a physical entity: it is a particular sequence of DNA. However, Genes-D are not genes for phenotypic traits. They are developmental resources, one set among many in the cell. They work together with the other developmental resources to build organisms, but they do not map on to particular traits.

Both of these ways of talking about genes are legitimate. Genes-P are predictive devices, and their usefulness as such is beyond question.

Genes-D are real sequences of DNA, the stuff which consumes the working lives of molecular biologists. But the gene concept that dominates our culture, the much-hyped notion of the gene, is the result of the illegitimate conflation of these two notions of the gene. *There are no stretches of DNA that are “for” particular phenotypic traits, no Genes-D that are also Genes-P*, Moss claims. Once we recognize this, the hype surrounding the gene should dissipate. Consider the hysteria that surrounded the announcement, in 2000, that the human genome had been mapped. Newspaper headlines around the world announced that “the book of life” (a favorite metaphor) had been decoded, the blueprint of humanity revealed.¹⁵² But all the Human Genome Project did was map the DNA sequences in the human genome. In other words, the genes it lists are Genes-D. We get so excited about this accomplishment only because we (mistakenly) believe that these Genes-D are also Genes-P. But they’re not. They might be useful in identifying Genes-P (since a Gene-P may be correlated with many different Genes-D, which occupy the place where a particular sequence is missing), but they are not genes for traits.

As Moss points out, the discovery by the Human Genome Project that we have far fewer genes than expected – about thirty thousand, around the same number as the mouse, with which we share the overwhelming majority of our genes – ought to have led to a deflation of our gene-hyperbole. Given that we have around the same number of genes as much simpler creatures, our great complexity is unlikely to be the product of our genes alone. Rather, it is the result of the modular architecture of organisms as a whole: the entire cellular machinery, as it develops in human beings, is the source of our complexity. Our “essence” is not contained in our genes, but distributed throughout the developmental resources of the organism.

If this deflationary perspective upon the gene is correct, than many of the hopes and fears that surround genetic engineering are misplaced. The enhancements that are so widely seen as inevitable just may not be technically possible. It is likely that complex traits are the result of a great many genes, *in a particular cellular context*. In other words, when we consider the effects that environments have on phenotypes, we must not limit ourselves to the *external* environment. The internal environment – of cellular machinery, of other genes, of the womb – is

also crucial. So, we cannot simply “lift” a gene from one organism, splice it into the genome of another, and expect to cause a particular phenotypic trait to develop. Genes just don’t work like that. We have an over-inflated view of the power of genes, because the genes(-p) which we have identified so far are genes for gross physical defects, which typically cause their detrimental effects no matter what their environment. But the genes that play a role in the development of desirable traits are not blunt instruments causing traits in simple ways; they work much more subtly, and in a context-sensitive manner. Though we can relatively easily delete a gene, and cause a phenotype to have a defect, improving function will prove much harder. There are no genes for intelligence, if by that we mean genes that are responsible for raised IQs across the range of normal internal and external environments. We are not going to produce designer babies, not now, and perhaps not ever.

Genes and environment

However, the fact that genetic enhancement is likely to prove far harder than is often thought does not stave off many of the threats that genes seem to pose to human hopes. So what if the bases of heredity are far more complex than gene talk seems to suggest? We know that heredity works in *some* manner, after all. Whether we can engineer desirable traits or not, we know that they are passed on. Inability to engineer human enhancements might slow down social stratification, but it will not prevent it. So, for example, Richard Herrnstein claimed. Herrnstein saw this stratification along genetic lines as an inevitable consequence of increasing meritocracy. Pinker summarizes the argument thus:

As social status becomes less strongly determined by arbitrary legacies such as race, parentage, and inherited wealth, it will become more strongly determined by talent, especially (in a modern economy) intelligence. Since differences in intelligence are partly inherited, and since intelligent people tend to marry other intelligent people, when a society becomes more just it will also become more stratified along genetic lines.¹⁵³

Indeed, as he famously argued in *The Bell Curve*, Herrnstein believed that genetic stratification was already well under way in the United States. Intelligence, he argued, is significantly heritable. But blacks, on average, consistently score lower in IQ tests than whites. Hence, it is likely to be the case that black disadvantage, on a wide range of social indicators, is partly genetic in origin. The existence of a black underclass has many causes, but one of them is the inferior genes of those of African descent.

Herrnstein’s thesis, and many others which attribute different traits to different people on the basis of their “genes,” does not depend upon any particular theory of how heredity functions. It is enough that it works in *some* way. Darwinian theory also demands that traits be heritable, in some manner. Thus, pointing out the limitations of, and the confusions that surround, talk of genes for traits does nothing to circumvent the dangers for social policy stemming from this particular challenge. It is very clear that these dangers are real. *The Bell Curve* makes them explicit. There, Herrnstein and his co-author Charles Murray argue that, since intelligence is largely inherited, improving the quality of education holds little promise for raising the life prospects of the largely black American underclass. Herrnstein and Murray focused particularly on programs like “Head Start,” which aimed to eliminate the social disadvantages suffered by members of low-income households by providing them with access to pre-school education. These programs seemed to offer increases in measured IQ, compared to controls who did not attend them, but these effects were transient. The gains begin to fade shortly after the child leaves the program, and in a few years the effects (on IQ, at least) seem to disappear completely.¹⁵⁴ On the basis of these findings, Herrnstein and Murray argue that pouring resources into remedial education programs for the disadvantaged is a pointless waste of effort. The effects “hardly justify investing billions of dollars in run-of-the-mill Head Start programs.” Similarly, they argue, affirmative action, which is based on the false assumption that ethnic groups are equal in intellectual ability, is dangerous: “Affirmative action, in education and the workplace alike, is leaking a poison into the American soul.”¹⁵⁵ As Herrnstein and Murray make all too apparent, claims for the heritability of intelligence have direct social and political consequence – not, of course, all by

themselves, but when coupled with our other beliefs about society, efficiency, equity, and justice. No matter how many times the naturalistic fallacy is invoked, this remains so.

These are extreme manifestations of the dangers for social policy that lurk in the background – and sometimes in the foreground – of these debates. Less extreme, but perhaps more pervasive, dangers lurk in the inappropriate biologization of human traits. To the extent to which dysfunctional or undesirable characteristics of human beings come to be seen as “in the genes,” in the sense that it would be difficult and costly to alter them, they cease to be felt to be social responsibilities. If depression is caused by an endogenous chemical imbalance, and not by high rates of unemployment; if criminality is caused by a genetic mutation; if low intelligence is inherited; then we must learn to manage, control, or intervene medically in these conditions. We can treat depressives and lock up criminals, but we can’t deal with either problem by building a fairer society. However, if these problems are predominantly social in origin, then we might hope to reduce them by social measures. Depression might be treated not (just) by providing anti-depressants, but by providing a sense of hope for a better future (more concretely, by reducing unemployment and alienation).

Of course, we can’t build a society upon pious hopes. If depression or intelligence are inherited and beyond our (practical) power to alter, then we need to know. If these claims are true, this is essential information, which we must learn to live with. We cannot reject them simply because they are unpalatable. Rather, we must subject them to scrutiny.

Heredity¹⁵⁶

What do we measure, when we measure heredity? We measure the extent to which the *variance* in a trait can be attributed to a variable. Thus, if we want to measure the extent to which a trait is “genetic,” we place organisms in a controlled environment, and measure the variance of that trait. If the environment is exactly the same for all the organisms, but they differ genetically from one another, then the variation is genetic in origin.

There are enormous practical difficulties in making accurate assessments of the genetic contribution to variation. No matter how

hard we try, we cannot control an environment totally; even in the laboratory, there are certain to be minor variations. These problems are magnified many times over in research on human beings, where we cannot conduct controlled experiments at all, for both practical and ethical reasons. For humans, we must rely upon studies of separated identical twins, of identical twins reared together compared to fraternal twins reared together, or of adoptees reared with biological siblings. The limits to the kind of data we can gather pose great problems, problems sufficient to invalidate many of the findings of early studies, which did not put sufficient thought into controlling for these difficulties. But the problems are not insurmountable: careful studies upon very large numbers of people give results that are statistically significant. Hence, I shall not focus on these practical problems but concern myself with the *conceptual* limits upon heredity research.

Herrnstein and Murray, on the basis of their review of many studies on the inheritance of intelligence, estimate that, within the white population, IQ is about sixty percent genetic. That is to say, sixty percent of the *variation* in IQ test scores among whites in the United States seems to be genetic in origin. It is important to understand, however, that the sense of “genetic” here is an artefact of the methodology. To say that a trait is genetic, in this sense, is not to say that it is caused by DNA sequences (or even by DNA sequences plus whatever other cellular and developmental resources combine to build the bodies of organisms). In the sense in which the word is used here, *only significantly variable traits are genetic*. Think, for example, of a trait such as “having two hands.” This is, in all probability, an “entrenched” trait, which is to say that its biological bases are laid down very early in the development of an organism, so that any mutation which tended to disrupt this development would almost certainly also have other, far-reaching, and most likely detrimental, effects on the organism. Having two hands is close to invariant in human beings. But what is the heritability of having two hands? You might think that it is close to one-hundred percent, on the grounds that almost every parent with two hands has a child with two hands. But you’d be wrong. Heritability measures the extent to which the *variation* in a trait has a “genetic” cause; since almost everyone is born with two hands, the heritability is in fact close to zero (indeed, a fraction of one percent). Variation in hand number is almost

entirely due to *environmental* causes – the effects of drugs on fetuses, or accidents – so hand number is a trait that is almost entirely environmentally caused.

So, in considering claims that a trait is partly or largely genetically determined, the first mistake we have to avoid is to confuse “genetic” in the sense in which it is employed here, with “biological.” Hand number is biologically determined, of course, but since it does not vary, it is not genetically caused. Even more bizarrely, using the method of measuring variation to assess the extent to which it is due to genetics counts many traits that are certainly *not* biological in cause as genetic. Consider hair length in the 1950s and 60s, in Western countries. At that time, most women had much longer hair than most men. Thus, variation in hair length was reliably correlated with a real biological difference. Applying the methodology of measuring heritability, we would find that hair length was highly heritable (the female children of mothers with long hair tended to have long hair themselves; the male children of men with short hair tended to have short hair themselves). But the pattern of hair length was entirely explained by fashions and social norms, and its heritability by the fact that these fashions tracked biological features of human beings. It may very well be that a very substantial part of the variance which comes out as genetic on the standard measures is nevertheless rooted in environmental differences, because people who possess certain biological characteristics are systematically treated differently to others who lack these characteristics.

Moreover, as the example of hair length demonstrates, it is a mistake – a most serious mistake, but none the less a very tempting one – to take “genetic” as synonymous with “fixed,” or even “difficult to change.” Traits might be genetic, and yet easily changed. Mightn’t it be objected that though the method of estimating heredity throws up occasional oddities, like the finding that hair length is heritable, this does not undermine the importance of the bulk of its findings? We can see how norms of hair length are maintained, by means that are not biological. Despite the fact that intelligence (for example) is not controlled by social norms, we can see how it can be modified, in certain circumstances, by such norms. If we found that black children scored lower than white, whether they were adopted (by white or black parents) or raised by their biological parents, and that was our only

evidence about the heredity of IQ, then we could speculate that racism, perhaps unconscious, is at work in some manner, leading people to treat these children differently, in a way that leads to them scoring lower in IQ tests. But we have a great deal of evidence upon which the social treatment hypothesis apparently gets no grip. Much of the evidence for the heritability of intelligence compares white children with white children, so we needn’t worry that racism distorts our results.

We can concede the case that the objector presses here, and continue to insist that “genetic” does not mean “unalterable.” Estimates of heritability, it must be recalled, are always *relative to a context*. We measure heredity by controlling the environment, and our estimate is therefore indexed to that environment. It does not follow, from the fact that a trait is (say) sixty percent heritable in one environment, or even across a range of environments, that its heritability will be about the same in another environment we have not yet examined. It might be the same, or higher, or lower. It might even be zero.

It’s useful to construct a graph that represents the extent to which a trait is heritable across a range of environments. Such a graph is known as the *norm of reaction* of that trait. In the following (hypothetical) norm of reaction, the *y*-axis represents the height of genetically identical plants, while the *x*-axis represents an environmental variable (say, the amount of phosphorous in the soil), with all other variables held constant:

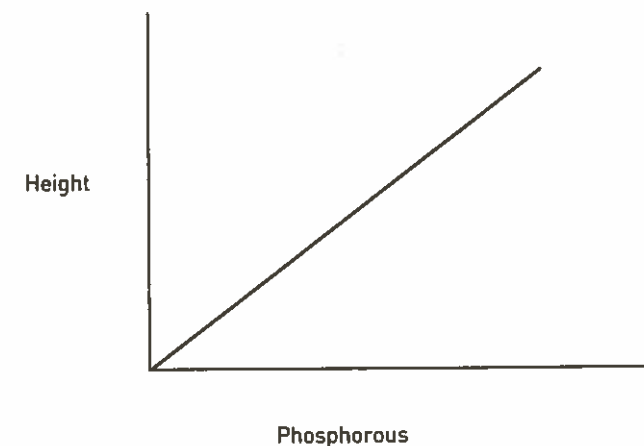


Figure 1: A hypothetical additive norm of reaction

This graph depicts a norm of reaction that is *additive*. That is, the relationship between the variable (in this case, phosphorous) and the phenotype is regular. The more phosphorous we add, the taller the plant. But not all norms of reaction are additive. The norm of reaction sketched above is hypothetical. If we actually tested the relationship between phosphorous and the height of a particular plant, we might discover that the norm of reaction looks something like this:

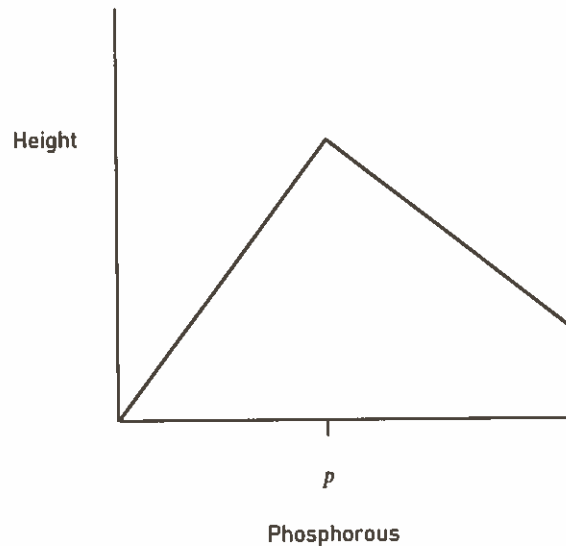


Figure 2: A hypothetical non-additive norm of reaction

In this norm of reaction, increasing phosphorous at first increases plant height. But once we pass a certain point, adding extra phosphorous decreases the height of the plant.

Whether or not a norm of reaction is additive will be, to some extent, an artefact of the range of variables across which it is studied. If we had plotted the norm of reaction in figure 2 only up to point *p*, we would have a graph that was additive. But, as we can see, extending the plot reveals a norm that isn't additive. The lesson here is that it is wrong to assume that the graph will extend at its current angle (or even in the same direction) across new environments. We have to discover the

truth empirically, case by case. Figure 1 illustrated an additive norm, but if the graph represented an actual norm of reaction, we could not simply assume that the slope of the graph would extend to infinity at the same angle.

Why does any of this matter? It matters because in the debate about the heritability of intelligence, character traits, and so on, there are some important implied assumptions about their norms of reaction. Indeed, people who claim that these traits are highly heritable typically assume that their norm of reaction is additive. They assume that the norms look something like this:

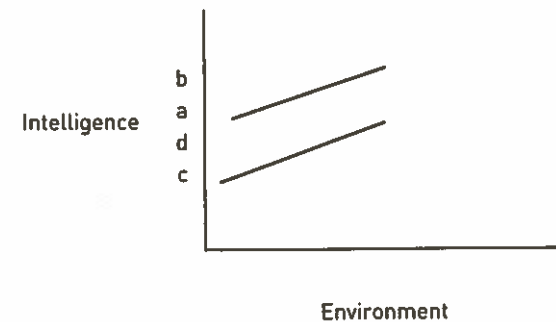


Figure 3: The norm of reaction for intelligence assumed by those who claim that our potential abilities are fixed at birth

Line *a-b* in figure 3 represents the norm of reaction for the intelligence of Highly Intelligent Helen. Helen was lucky; she was born with lots of genes for intelligence. That is not to say that she will be a genius in any environment: no one believes *that*; rather, it is that in any normal environment, she will be clever. The exact nature of the claim becomes clearer when we compare Helen to Rather Dull Roger (represented by line *c-d*). There is *no* environment in which Roger is smarter than Helen. There may be an environment, way off to the left of the graph, in which Roger is *as* smart as Helen. But that would be an extremely undesirable environment indeed, in which Roger is as smart as Helen only because both have been unable to develop their cognitive capacities to anything like normal levels (perhaps both Roger and Helen are brain-damaged in that environment, as a result of severe malnutrition). Even if environmental intervention can increase Roger's

intelligence somewhat (and Herrnstein and Murray's claims about Head Start might lead us to think that they believe that the norm of reaction is even flatter than depicted here), Helen is smarter than Roger across all normal, and all desirable, environments. Genetically speaking, she just *is* cleverer than him.

But if the norm of reaction is not additive, then we cannot speak of the "genetically more intelligent person." Instead, we shall have to talk about the more intelligent person *in an environment*. Consider the following norm of reaction, which depicts the effects of being planted at different altitudes on the height of seven *Achillea* plants:

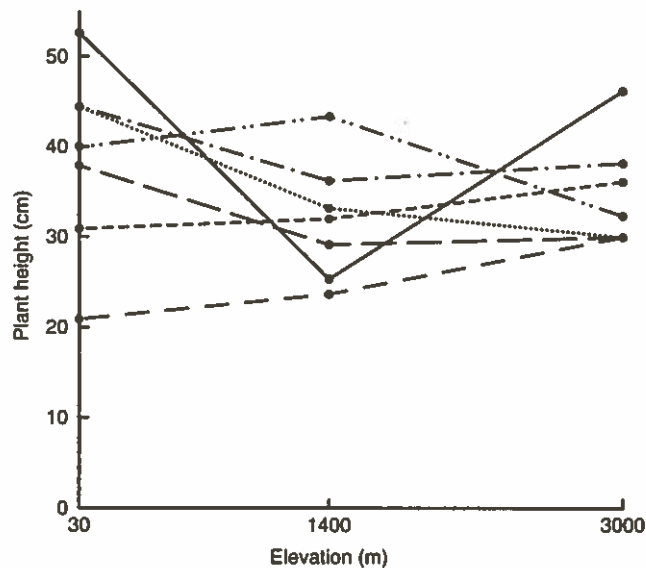


Figure 4: The norm of reaction depicting the height of the plant *Achillea millefolium* as a function of elevation. From David T. Suzuki, et al. *An Introduction to Genetic Analysis* (7th ed.), (New York: W. H. Freeman, 2000), p. 20. Used with permission.

Which of these plants is, genetically speaking, the tallest? Obviously, the question is nonsensical. At different altitudes, different plants are tallest. There is no plant that is tallest, or even relatively tall, across all environments.

Commenting on this example, Francis Fukuyama claims that most human characteristics are not like this.¹⁵⁷ Is he right? What is the shape of the norm of reaction for intelligence? Several studies on laboratory animals have attempted to answer this question. One study examined the effects of impoverished and enriched environments on two strains of rats, one of which had been bred to be dull and the other to be smart (their intelligence was measured by their ability to navigate a maze without making errors). The researchers bred and raised these strains of rats in two environments that differed from that in which their parents had been raised. One was "enriched" with toys and visual stimulation, and the other "restricted"—uniform and monochromatic. Then they tested the rats for maze-running ability. The maze bright rats made relatively few mistakes when they were raised in a normal environment—far fewer than the maze dull rats in the same environment. However, when both were raised in an enriched environment, the "maze dull" rats made nearly as few mistakes as the "maze bright" ones (indeed, the difference between the two was not statistically significant). And when both were raised in restricted environments, the (genetically) maze bright rats made just as many mistakes as the maze dull.¹⁵⁸

A recent study by researchers engaged in the construction of animal models for dysfunctions in human cognition reinforces the point. The research team, led by Professor Colin Blakemore, created transgenic mice (mice into which "foreign" genetic material had been incorporated) to model Huntington's disease—an inherited, and invariably lethal, degenerative neurological condition. The transgenic mice were reared in two separate groups, one in standard laboratory cages, and the other in enriched environments. They found that the onset of the symptoms of brain degeneration was significantly later in the mice reared in the enriched environments, compared to those reared in standard environments. At twenty-two weeks, all the mice in the control group were significantly disabled, but only fifteen percent of the enriched environment mice were experiencing difficulties.¹⁵⁹ Once again, environmental interventions are successful in altering a phenotypic trait that is "in the genes."

The lesson for studies of heritability should be obvious. Even if we assume (for the sake of argument) that findings like those of

Herrnstein and Murray are accurate: certain groups of people are less bright than others *across the environments so far studied*, we can't validly extrapolate from these environments to others as yet unexplored. Heritability is a measure of the extent to which variation is due to genetics *in the environments examined*. In "normal" environments, ability to run mazes is highly heritable, but in enriched and in restricted environments the heritability of the trait drops to near zero. Intelligence *might* be sixty percent heritable in the contemporary United States, but that does not imply that intelligence is fixed unalterably, there or elsewhere. There may be environments, which are accessible to us and not otherwise undesirable, in which its heritability is much lower: perhaps because far more people have a high IQ.

Nor can we assume that, if there are environmental interventions which will be effective in altering phenotypes in desirable ways (making many more humans "maze bright" than is currently the case), these interventions will be difficult to implement or involve restrictions on personal liberties. The story of phenylketonuria (PKU) is instructive here. PKU is a highly heritable disease: sufferers must have inherited a recessive gene from each parent. Until fairly recently, PKU would have been considered an entirely genetic disease, since children of these parents suffered its terrible symptoms (essentially, severe mental retardation) across all known environments. Today, however, we can regard it principally as a product of the environment, because we know how to intervene environmentally to prevent its adverse effects: if sufferers are fed a special diet in the first few years of their life, they escape mental retardation.¹⁶⁰ The heritability of PKU was close to one-hundred percent; now it's close to zero. There is no reason to think that genetic influences – upon behavior or upon physiology – are necessarily harder to alter than environmental influences.

Fukuyama claims that human characteristics are not like this: the norms of reaction for our traits are additive. But there is no evidence for this at all. Norms of reaction are typically not additive,¹⁶¹ so we have good reason to assume the opposite. Twin studies, no matter how carefully conducted, will not come to the aid of the defender of "nature" over "nurture" here. What is at issue is, in principle, something that these studies cannot reveal, for they are, necessarily, concerned only with a narrow range of environments. No matter the degree to which

these studies show intelligence (or any other phenotypic trait) to be heritable in these environments, we cannot extrapolate from them to what might happen in other environments. The only way to discover the effectiveness of environmental interventions of one sort or another is to test them.

It is worth noting, by the way, one other limitation of twin studies. When they discover a pronounced similarity between identical twins reared apart, researchers rightly take this as evidence that innate dispositions play a role in producing phenotypic traits (at least in *this* environment). But we can't conclude that the extent of the similarity is a direct product of "nature," *not even in this environment*. We saw earlier how small asymmetries, which might be due to nature but which might have quite different sources as well, could produce large effects. Something similar is true here. Suppose that a child finds music *slightly* more rewarding than most children – perhaps because she is a slightly better musician than average (people enjoy what they find easier, and get pleasure from the praise which comes from being good at something). It is very likely, other things being equal, that the way she is treated and the way she treats herself will enhance that ability. She enjoys playing her instrument, and therefore practices more. As a result she becomes more proficient, and enjoys playing still more. At the same time, she is praised for her abilities and encouraged to develop them further. Her musical ability increases still more, while other tasks she found more difficult (and therefore less rewarding) are abandoned, and her skills in those areas atrophy.

Now suppose that this girl is one of a pair of identical twins who were separated at birth and reared apart. Her twin sister might well also be slightly better than average at music, and as a result of the same kind of processes her musical abilities have been further enhanced in an ever-turning virtuous circle. When psychologists come along and test them, they will find that they have remarkably similar psychological profiles: they are both very good musicians, but bad at sport. They will take this as evidence that abilities are innate. To some extent, it is. But we go wrong if we take this to be evidence of the unimportance of environment. It is largely because the twins had propensities that encouraged certain kinds of treatment that they are now similar. A slight innate similarity causes a quite dramatic effect.

So, twin studies show us the importance of nurture, as much as of nature. People never have their traits as a direct result of nature, but always also as a result of the way in which they have been treated, and in which they treat themselves. Choices, both ours and those of others, matter. Twins reared together are often *less* similar than those reared apart, because they make an effort to differentiate themselves, and once they do so the virtuous circle begins turning, reinforcing those differences.¹⁶² There is nothing inevitable about innate propensities: they remain responsive to cultural and social factors. Biology is not destiny.

We should also note the way in which my remarks concerning heritability are directly relevant to the dispute between the SSSM and evolutionary psychology. Evolutionary psychology is committed to the hypothesis that human psychological traits are inherited and (at least in many versions of it) that they are therefore, practically speaking, unalterable. But now we see that heritability is always relative to environment, so that we cannot extrapolate estimates of it to further environments. A trait that is sixty percent heritable in one environment might be ninety-five percent, or two percent, heritable in another. Conversely, the extent to which a trait is due to environmental factors is always relative to the amount of genetic variation within the population under study. *All* variation in the phenotypes of genetically identical corn (for example) is environmentally caused.

This allows us to state the dispute between evolutionary psychology and the SSSM much more carefully than before. As Philip Kitcher has pointed out, it is essentially an argument about the shape of the norm of reaction of the traits of interest.¹⁶³ Those who we regard as being on the side of nature hold that the norm is relatively flat *across the range of environments that are practically accessible*. Though (except for the least sophisticated of them) they do not deny that it is possible to alter the traits in question, they hold that is difficult to alter them in ways that are desirable (to bring Roger's intelligence up to the level of Helen's rather than hers down to his). Moreover, to the extent to which we can alter traits in desirable directions, they hold that we can do so only by altering the environment in highly *undesirable* ways: for example, only by indoctrinating people, or massively restricting their freedom.

In contrast, those on the "nurture" side of the equation do not (or should not) deny that biology is an important influence upon

behavior. Instead, they hold that this influence is powerfully mediated by culture, so that we can, at least in principle, alter it in ways of our choosing. They should not deny, either, that sometimes such alterations will be hard to achieve, and that sometimes they will have unexpected costs. But society is not an organism; though it is true that almost all mutations (and by far the overwhelming majority of those that affect entrenched characteristics) are bad for organisms, we have no reason to think the same of societies. Often, if the social changes we make are carefully planned, and informed by all our knowledge of human biology, psychology, and the social sciences, they will bring far more benefits than costs. We can alter society in ways we choose, and in doing so we inevitably alter ourselves. That is the claim made by those who advocate the importance of nurture, and, as we have seen, there is every reason to think it a plausible one.

Remaking ourselves

We saw earlier that genetic engineering arouses a particular horror in many writers. Conservatives and liberals alike see it as threatening to bring about a dystopic "brave new world," as the almost irresistible cliché has it. Genetic engineering has the potential to make the acquired advantages of parents heritable (to make Lamarckism true through technology), and so to create two separate classes of human: one born to rule, and one suited only to be ruled. Moreover, genetic engineering will allow us to choose the characteristics of our children, to transform them from chance "gifts" (perhaps of God) to consciously designed artefacts. In so doing, many fear, it risks stripping human life of its meaning and its dignity. This fear, too, comes in left and right wing forms. Leon Kass, the head of George W. Bush's Council on Bioethics, is one of its better-known conservative defenders:

Any child whose being, character, and capacities exist owing to human design does not stand on the same plane as its makers. As with any product of our making, no matter how excellent, the artificer stands above it, not as an equal but as a superior, transcending it by his will and creative prowess. [...] Such an arrangement is profoundly dehumanizing, no matter how good the product.¹⁶⁴

The very fact that we choose the characteristics our children possess dehumanizes us, as well as them.

It should be clear by now that this fear is misplaced. If genetic engineering of human characteristics proves to be possible and practicable – and we have seen that there are reasons to doubt that it will – it will not endow us with powers that we do not *already* possess. If we fear that it will give the rich access to enhancements that are not available to the poor then we need to recall that such enhancements are already available to, and widely used by, the wealthy – in the superior schooling, nutrition, medical care, and other advantages that they can buy. Consider the current life expectancy of a child born today in Sierra Leone, versus that of a child born in the United States: thirty-eight years for the former, and seventy-seven for the latter. This difference is strongly correlated with income. Sierra Leone has a per capita gross national product of just \$160, whereas the United States has a per capita GNP of \$30,600. Infant mortality figures paint a similarly grim picture.¹⁶⁵ Within countries, the disparities in life expectancy also follow income lines. These stark injustices are not the result of any genetic superiority of Americans over Africans. They are the result of the typical environments in which members of each group are raised. We are already allowing people to use enhancement technologies, and they enhance the only entity that counts: the phenotype.

What goes for life expectancy and for health goes for behavioral traits as well. Recall Terrie Moffit's research upon the genetic bases of violence. Her team found that men who possessed a particular version of one gene *and* were physically or sexually abused as children were far more likely to become violent criminals than were men who possessed the gene, or had a history of abuse, alone. As she pointed out, her research reveals the importance of both elements of the equation, nature *and* nurture: "This is not really the story of a gene that has a risk for anti-social behaviour. It's the story of the interplay between a gene and the experience of maltreatment."¹⁶⁶ Parents already possess the ability to pass on their advantages (or their disadvantages) to their children. So does society at large. We choose our children; design them, at least in rough outline. Better understandings of gene/environment interaction – that is, the construction of more detailed norms of reaction, for more variables and across more environments – will enhance

this control, *whether we use genetic engineering or "mere" environmental manipulation* as our instrument.

It might be objected that despite what I have said, genetics will remain a special focus of concern, because enhancements encoded into the genome will be passed on to offspring, in a way in which purely social advantages can never be. This is the real concern: that genetic enhancement by the wealthy could lead to their splitting off from the rest of humanity, becoming a species unto themselves, with a biologically-based right to rule. Once again, though, this ignores the reality of heritability. Since heritability *is* heritability-within-an-environment, the two inextricably linked, we ought to be concerned as much with the environmental as the genetic side of the equation. At present, parents pass on their environmental (dis)advantages to their children as a matter of course, so their children start from significantly different positions. If it is the inheritance of advantage with which we are concerned – the way in which the privileged are able to accumulate it generation upon generation, in ways that are expressed in higher cognitive ability (for instance) – then we ought to be as much concerned with this kind of inheritance as with germline intervention.

It is morally and scientifically arbitrary, not to mention fundamentally confused, to be concerned with *genetic* justice. A concern for justice must be *phenotypic*: it must be focused on the observable characteristics displayed by people, and not on the question of their genetic base, except in so far as the latter is relevant to the former. If we have the intuition that inherited gross inequality is morally impermissible, we ought to be concerned with current inequalities. In principle, the possibilities opened up by genetic enhancement add nothing new to these concerns.

I have no doubt that many people will remain skeptical. The facts about heritability and what it measures – that "heritable" does not necessarily mean "genetic" *nor* vice versa, or that estimates of heritability are always relative to the environments in which they are measured – have been pointed out over and over again for decades, without any discernible impact upon the propensity of apparently intelligent people to invoke heritability claims to show that biology is destiny. The proponents of this view rarely indicate why they believe they can predict what shape norms of reaction will have in new environments. I

suggest, however, that, underlying their faith in their predictions, must be the same views about history and cultural variation that divide them so sharply from proponents of something closer to the SSSM. They believe that historically and geographically distant societies differ only superficially from our own, and that *therefore* the capacity of human beings to create social worlds is tightly constrained. Proponents of the SSSM tend to deny the first claim, and insist that the world has seen a great diversity of human societies. From this, it follows that such diversity is possible. Even if they turned out to be wrong about this last claim, however, they might still be right that *future* diversity is possible. One final example, then, to sway waverers, and strengthen the conviction that fundamental alterations in human society – with concomitant changes in gender roles, for instance – are indeed possible.

Human beings are, all sides agree, unique in the degree of their behavioral flexibility. I assume, therefore, that if any species is capable of fundamentally altering their social arrangements in desirable ways, that species will be humans. If any other species has succeeded in such a transformation, it would constitute powerful evidence for the claim that human beings are indeed capable of such a revolution. And at least one other species has altered its fundamental social arrangements, arguably for the better: chimpanzees. Placed in a new environment, which fundamentally differs from their ancestral home, chimpanzee behavior changes radically.

In the wild, chimpanzees live in bands consisting of around fifty members. Chimpanzee society is hierarchical and patriarchal. A single alpha male, aided by allies, monopolizes access to females in estrous, and is deferred to by other members of the group. His allies receive sexual and food privileges in return for their support. Females are not entirely powerless, but because they are, on average, smaller than the males, no female or small group of females is able to override the wishes of the dominant males.

In captivity, the situation is quite different. This is not the result of any direct or planned human intervention to equalize the power of males and females. Rather, it seems to be the unintended consequence of providing the chimps with easy access to food. Freed from the need to forage constantly, female chimps are able to form active coalitions, which play an important role in the “political” life of their group.

This coalition-forming behavior, rare in the wild, has been observed repeatedly in captivity, and has the effect of dramatically increasing the power of female chimpanzees:

Captive female allies have been able to control certain behaviours of males who are individually dominant over them, and do so in ways that are striking. They usually manage to play ‘kingmaker’, and they always seem to control which males will be involved with peacemaking [...] a large, well-unified, rank-and-file political coalition is successfully manipulating key behaviours of individuals that are threatening to other individuals.¹⁶⁷

A change in their environment (the ready availability of food), which, from their point of view, is obviously desirable, leads to an important transformation in chimpanzee social life. Captive chimpanzees do not suddenly transcend their biology. They remain, very recognizably, chimpanzees. But they begin to behave in ways that we would never have suspected, on the basis of observation of their behavior in their own EEA. If “mere animals” that are, we believe, far more tightly constrained by innate behaviors, can have their lives transformed by environmental alterations, then we have every reason to believe that the same is true for human beings. We live in environments that are much further away from our own EEA than do these chimps. Those of us who are lucky are also spared the trial of constant seeking for food. Almost none of us hunt for basic sustenance, or live in nomadic groups. Ten thousand years of agriculture has made us sedentary, and allowed us to live in larger and larger urban conglomerations. It would be very surprising indeed if these changes were not reflected in our behavior and our social lives.

Conclusion: humane nature

How constrained is our future by our past? We are *moral* animals, but we are still *animals*. Our brains allow us to engage in sophisticated reasoning, but they have built-in biases and heuristics of which we are largely unaware. We are not angels, fallen or otherwise, but merely jumped-up apes. Does the legacy of evolution, our Stone Age minds, empty our lives of meaning, and our morality of substance? We are material beings, constrained by physics, and we are animals,

constrained by biology. Yet, to a very large extent, indeed to an extent that seems to be ever-growing, we seem able to shape our own futures, to endow our lives with meaning, and to build an ever more adequate morality on the basis of our animal instincts.

We could conclude our story in one of two possible ways: call them the *conservative* and the *radical* views of human nature. The conservative view concedes a great deal to evolutionary psychology: it accepts that a great many of our dispositions are fixed. But it insists – rightly – that nevertheless our future remains open; fixed dispositions do not constrain the possible forms of our social life. The radical view rejects even the claim that there is a large and interesting set of fixed human dispositions. I think the radical view is correct, and I will go on to defend it. But it is important to see that even the conservative view gives us all the freedom and morality we need.

Consider, first, an example that, even upon the conservative view, establishes the extent to which human social life is malleable. The evidence, from a variety of fields, suggests very strongly that human beings are evolved to live in relatively small groups. Robin Dunbar argues convincingly that large brains evolved to enable complex group life. Animals, like monkeys and apes, which live in large and complex societies, with hierarchies that must be respected, need large brains to keep track of group members and to remember the relation in which everyone stands to everyone else. Dunbar has convincingly shown that we can predict the average size of an animal's group just by measuring its relative brain (more specifically, its neocortical) size.

So what size groups are humans evolved for? According to Dunbar, the predicted group size is around 150 members.¹⁶⁸ Further evidence for this figure comes from anthropologists, who tell us that hunter-gatherers (who, we believe, live in conditions quite similar to those prevailing in the EEA) live in groups of around 150, and paleo-anthropologists, who tell us that the archaeological evidence also supports this figure. But of course human beings increasingly live in *much* larger groups, in cities, where populations are measured in the millions. We may have evolved in small groups, and we may still carry around cognitive equipment capable of tracking only a relatively small number of people. But we are not obliged to continue to live in such groups.

Might it be that we are better suited to small group life? Perhaps we would be happier in a small group. Just think of the number of television programs and novels celebrating village life. Doesn't everyone know that life in the city is a rat race, and villages are friendlier places, to which everyone would escape – from the pollution, the noise, the crime, and the pressure – if only they could? Matt Ridley suggests that cities breed crime, because one shot encounters in prisoner's dilemmas encourage the "defect" strategy. If you can melt into the crowd, you can get away with much more.

If cities are so awful, though, why do people continue to move to them, of their own free will? And move they do: around the world big cities are magnets for the surrounding countryside, and the proportion of the world's population that is urbanized is growing ever larger. It may well be true that city living has its costs, but it has its benefits as well. If it can be more alienating, it is also, frequently, wealthier, since industry is usually based in it, and is able to take advantage of the economies of scale that stem from having a large workforce to hand. If it can be isolating, it can also be exciting: it attracts a range and variety of people, together with their traditional food, music and theatre, who would otherwise never mix, and adds the attractions of technology to the mixture. If its anonymity can allow criminals to prosper, it can also allow for a degree of freedom unavailable in the claustrophobic atmosphere of a village. Gay subcultures, artists, intellectuals; all flourish only in cities.

So even if it is true that, in some ways, our Stone Age brains can't adapt to city life, because they can't track social networks bigger than those in which our ancestors lived, and even if that imposes costs on those who choose to live in cities, it still might be rational to make the choice. If there are costs, then there are also benefits, and the latter might outweigh the former. We can transcend our past, which is not to say that we can wish it away, but that we can find ways to work with and even around it, if we so wish. Stone Age minds might be just the tools we need to build our Space Age future.

Even on the conservative view, according to which many of our traits (like the ability to track members of our group) are fixed, and which insists that therefore there are limits to how well we can adapt to new social forms, we can reshape our social lives for the better. We can

turn our in-built dispositions to new tasks and find ways to accommodate what we can't change. Though we might be adapted for small groups, we might nevertheless be happier, freer, and more fulfilled, in large ones.

In the course of this book, however, we have seen that there are many good reasons to think that the conservative view understates the malleability of the human mind. If, as we have seen, we cannot extrapolate from the heritability of traits at present to the heritability of the same traits in a future environment, we have little reason to think that many of our dispositions are as firmly fixed as all that. My guess, for what it's worth, is that the conservative story will turn out to be true for *some* dispositions, but far fewer than evolutionary psychologists generally believe.

According to Matt Ridley, who defends the conservative view, culture adds nothing significant to the human mind: "We human beings would probably be almost as good at playing, plotting and planning if we had never spoken a word or fashioned a tool."¹⁶⁹ Radicals like me reject this view; we think that cultural tools fundamentally change our cognitive capacities.

In the radical view, too many evolutionary psychologists and proponents of the more simplistic varieties of evolutionary ethics simply get human beings wrong. They are right in thinking that we need to understand what kind of animal we are, but they do not see the ways in which human beings are unique: uniquely unique. We are a kind of animal who can – who *must* – rebuild ourselves, using the tools of our culture. We come into the world relatively undeveloped and helpless, and require a lengthy childhood before we can take care of ourselves. This is time in which parents take over the task of forming their children, more or less consciously, and prepare them for a life of continual self-transformation. We are animals who are incomplete, and are therefore made to interface with the cultural tools that finish us off. Our minds are as much outside as inside our skulls.

Andy Clark recounts a wonderful example in his recent *Natural-Born Cyborgs*, of how access to a system of symbols transforms capacities, even of chimpanzees. Experimenters trained a group of chimps to associate particular symbols with pairs of objects that were either identical or different, so that any pair of identical objects (say two bananas)

would be associated with a red triangle, whereas any pair of objects that were different (say a banana and a cup) would be associated with a blue square. The symbol-trained chimps, and only those chimps, were able to go on to solve another problem, that of sorting *pairs of pairs* of objects into the right categories. A pair of pairs is the same if both pairs are the same (for example, two bananas and two cups) *or* if both pairs are different (for example, a banana and a cup, and a shoe and a box). A pair of pairs is different if one of the pairs is the same and the other is different. As you can see, the notion of higher-order sameness (difference) is quite hard to grasp, and proved to be completely beyond the grasp of non-symbol-trained chimps. For symbol-trained chimps, however, it was possible: they had only to compare their symbols to get the right answer. Having symbols available transforms a higher-order categorization task into a first-order task, which is much more tractable. Clark suggests that human beings have the cognitive capacities they do because we have an entire system of symbols – language – available to us, which enables us to transform many tasks. But, as he goes on to argue, if this is true, there is every reason to think that further transformations in our symbolic systems will enhance our cognitive capacities even more.¹⁷⁰

Clark draws the correct moral for our understanding of ourselves in relation to our evolutionary past. Our "cognitive biases are indeed the product of our evolutionary past," but they do not determine the contemporary mind: "Fixed genetic resources [are] one small group of players on a crowded stage. Our self-image as a species should not be that of ancient biological minds in colorful young technological clothes. Instead, ours are chameleon minds, factory-primed to merge with what they find and with what they themselves create."¹⁷¹ Our minds are products of evolution, and they bear the traces of the past upon them. But we have not yet discovered a limit to their plasticity, and it may be that there is no such limit. We are able to transform our minds by transforming the worlds with which we interact. Our minds create the tools that create our minds.

What of the dispositions that form the basis of our moral system? Are they, too, subject to alteration, through the kinds of social transformations that alter our cognitive capacities? We have no reason to think this set of dispositions is any more immune than any other. This

gives us special reason to take extra care in transforming the world and (therefore) ourselves, to ensure that we strengthen those dispositions of which we are justifiably proud and weaken those (equally innate, equally natural) dispositions of which we are ashamed. We have dispositions to care for one another, to empathize and, instinctively, to offer help, and we have dispositions to war, to anger, and to selfishness; we must seek to strengthen the first set and be vigilant against the second. But we have every reason to think that this is a task at which it is at least possible to succeed.

T. H. Huxley, Darwin's bulldog, was right, over a century ago: our moral motives are a product of evolution, but so are our *immoral* motives. We become moral animals not when we obey our evolved dispositions, but when we go to work on them: when we use those which we rightly identify as the most important, the ones which we are obliged to obey, as a template to prune and refine our dispositions. My inclination simply to take what I want, without regard for the feelings of others, is as much a product of evolution as is my inclination to stop and lend a hand. But I am a rational animal (thanks to evolution), and an acculturated one as well (thanks, indirectly, to evolution), and I am able to engage in the ongoing cultural project of distinguishing between evolved dispositions, separating out those which are binding and those to which we ought to accord no weight at all, to work at strengthening some and reducing the power of, perhaps even eliminating, others. Evolution gave us the preconditions of morality, but it is only as a result of the cultural elaboration of this raw material that we come to be moral beings.

Darwin and his followers have been accused of attempting to strip the world, and humanity, of its magnificence, reducing it to no more than a product of blind forces. But the process of evolution, the billions of years long process whereby simple life forms slowly developed into the myriad of animals and plants around us, is awe-inspiring. It allows us to glimpse apparent intelligence in the unexpectedly sophisticated behavior of the simplest microbes, at the same time as it pricks the bubble of pretension of the most complex, showing how they – we – too are the product of blind forces, all too apt to reveal our lowly origins. More magnificent still are the indirect products of evolution: our cathedrals and our philosophical systems, our cities and our

morality. Strange to say, these indirect products react back upon their creators, helping to re-create us, to give us new powers and new capacities. We are animals, and we cannot ever free ourselves of our biological heritage. We have no need: it enables all the flexible, rational, and caring behavior that we could want, and allows us to seek to become ever more moral beings.