Against Maladaptationism: or What's Wrong with Evolutionary Psychology?

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My title may surprise some. One of the common accusations against evolutionary psychology is that it is panadaptationist, or Panglossian, it supposes that everything about us is an adaptation, perfectly shaped for its conditions of life by the all-powerful hand of natural selection. But there is no mystery here, and both criticisms may be correct. The explanation is just that evolutionary psychology, or the variant of it with which I shall be concerned, supposes that we are adapted to the environment of the Stone Age. Thus we are adapted, perhaps Panglossianly adapted, by natural selection, but not to the environment in which we have the misfortune to find ourselves but to one long past. Hence the maladaptation. To take one familiar example, in the Stone Age fat and sugar were rare and excellent sources of energy, so we became adapted to consume them voraciously whenever the chance presents itself. But in a world full of Krispy Kreme donuts and deep-fried Mars bars this trait is highly maladaptive and leads to heart disease, diabetes, and all the other woes of the age of obesity.

The brand of Evolutionary Psychology I'm considering today is the programme developed by Leda Cosmides, John Tooby, David Buss and a few others, and popularised with great effect by Stephen Pinker and, indeed, my commentator today, Robert Wright. It is not the only version, but it is the most prominent. It can be quickly summarised. It holds that our minds consist of a large set of modules, shaped by natural selection to

solve particular problems set in our evolutionary history. These are problems from the period known as the Pleistocene, roughly the one to two million years preceding the emergence of human civilisation, which I shall refer to loosely as the Stone Age. The view that we are adapted to the Stone Age rather than to modern life is the maladaptationism of my title.

It is quite surprising that we should be, in this way, systematically maladapted. We are, after all, probably the most successful large organisms in the history of life, and this success has accelerated as the conditions of our existence have diverged ever further from those of the Stone Age. It is surprising, at least, that this should have happened if we are systematically adapted to a quite different environment from the one in which we appear to have thrived so spectacularly. Fortunately, there is no good reason to accept this maladaptationist thesis. It is based on bad biology: an obsolete view of genetics and a dubious and probably unsupportable view of evolution. There is much else wrong with evolutionary psychology, and its errors have been thoroughly documented by myself and others. I shan't speak today, for instance about the Panglossianism mentioned above, the assumption that any feature of an organism, including the cognitive structure of the human mind is likely to be an optimal response to some conditions at some point in evolutionary history, or the even less defensible obverse assumption, that if something would have been a good idea, it almost certainly evolved. I shan't speak on the controversial issue of the modularity of the mind, the as analogous to a Swiss Army knife. And I won't speak about the endemic evidential weaknesses of the project, the ways in which evolutionary speculations or conveniently hand-picked animal analogies

so often make up for thin and controversial empirical grounding of the claims about what has actually evolved in the human case.

Given the widespread criticisms of Evolutionary Psychology and, perhaps more importantly, the fact that there are other perhaps more credible evolutionary approaches to understanding human behaviour, it may well be wondered why I am spending my time on it. So let me offer some broader context. I think quite generally that the ways in which evolution can illuminate biological questions is often misunderstood. Evolution provides one essential kind of explanation of biological phenomena, but its ability to predict or discover phenomena is limited. Attempts to do so generally involve extremely simplistic evolutionary models, and their apparent outputs can be almost entirely traced to these simplifications. The one important exception to this sceptical suggestion is the extent to which evolution legitimates comparative biology. Detection of homology, the common evolutionary origin of a feature, can provide defeasible but valuable clues about function. Despite the hype about the human genome project, it has been well understood from the start that the most interesting information that might come from genome sequencing technology was comparative, the ability to detect similar and evolutionarily related genomic elements in different biological contexts. An extraordinary difficulty for any form of evolutionary psychology is that there are no relevant species for evolutionary comparison. To the extent that cognitive mechanism evolved, as evolutionary psychologists propose, several million years after the division of the human lineage split from that of the chimpanzees, and given that everyone agrees that all contemporary humans belong to one species, this lack is indisputable.

Evolutionary Psychology proposes to fill this gap by claiming to know when we evolved our distinctive psychology, what were the environmental conditions that our ancestors faced there, and by offering a priori arguments about what would be the best psychological mechanisms to deal with those conditions. Such arguments then provide epistemological depth to thin and controversial evidence about what humans are actually like. I claim that all of these steps are invalid. We don't know when our distinctive psychology evolved and much of it is likely to be well adapted to contemporary conditions. We don't really know a great deal about the conditions of the Pleistocene and even of we did this would provide the most doubtful grounds for inferring anything about our adaptive responses to them. Psychology should be empirical not a priori.

As I have indicated, my main focus will be on the first part of the argument. Much of this, however, is an excuse to discuss some remarkable developments in recent biology which should quite generally invite reconsideration of some common broad assumptions about the mechanism of evolution. Complexities emerging from recent molecular biology point to a much wider range of possible evolutionary mechanisms than have been widely recognised. Though in one way this makes evolutionary theory an increasingly rich and exciting field, it also makes attempts to infer biological fact from evolutionary theory increasingly risky. And while the plurality and complexity of evolutionary mechanisms greatly increases the resources for evolutionary explanation, it correspondingly decreases the possibilities for evolutionary prediction. In fact, and perhaps for this I should apologise, I won't have a lot to say at all directly about evolutionary psychology. I do hope, however, to show what an increasingly implausible project it is becoming in the light of recent biology. As evolutionary thinking begins to

catch up with the revolution in molecular biology, the decades old evolutionary theory on which Evolutionary Psychology has been built can now be seen to be of merely antiquarian interest.

Why are we thought to be adapted to the conditions of the Stone Age? Let me quote Leda Cosmides and John Tooby:

> Our species spent over 99% of its evolutionary history as hunter-gatherers in Pleistocene [Stone Age] environments. Human psychological mechanisms should be adapted to those environments, not necessarily to the twentieth-century industrialised world. The rapid technological advances of the last several thousand years have created many situations, both important an unimportant, that would have been uncommon (or nonexistent) in Pleistocene conditions. Evolutionary theorists ought not to be surprised when evolutionarily unprecedented environmental inputs yield maladaptive behaviour. (Cosmides and Tooby, 1987, p. 280-1).

Here we see not only the explicit maladaptationism but also the implicit panadaptationism: 'human psychological mechanisms should be adapted to those environments'.

Why should we not expect that psychological mechanisms have adapted to more contemporary conditions? It is not enough to say merely that our ancestors have spent more time in the Stone Age. Our ancestors have also spent a great deal more time as single-celled organisms. But this does not show that we are adapted to life in the primordial slime. The answer widely assumed by Evolutionary Psychologists is that there

has not been enough time since the Stone Age for us to have adapted significantly to more recent conditions. (And, of course, that there was enough time for our early human ancestors to adapt to the conditions they encountered, whatever those were.)

So how much time is enough? How fast is evolution? It is still common, and underlies this part of the Evolutionary Psychologists' argument, that evolution consists in change in gene frequency. The whole story goes something like this. Psychological adaptation amounts to the existence of neurological structures in the brain. These structures are built by genes. The necessary genes are acquired by random mutation of existing genetic material and selection of advantageous mutations. Since a random mutation is almost certain to be disastrous unless its consequence is fairly similar to that of the unmutated state, each mutation is assumed to provide only a small change. A series of these small changes, each of which will take a substantial number of generations to reach fixation in the population, can eventually produce complex adapted structures.

Richard Dawkins gives a celebrated illustration of this way of thinking in his discussion of the evolution of the eye in *The Blind Watchmaker*. Provided we can think of 1,000 or 10,000 steps between no eye and fully functional eye, geological time is long enough for each of these steps to have appeared by chance mutation and spread to fixation through the population. Dawkins in fact seems to think that this development is almost inevitable, but we need only assume that it is possible.

I'm not at all sure whether, if this picture is right, a million or two years is long enough for the evolution of the human mind. Our ancestors two million years ago had brains about one third of the size of our present brains, so it is reasonable to assume, as Evolutionary Psychologists generally do, that important contemporary human

neurological structures evolved in those two million years. For evolutionary psychology this amounts to the generation of genetically determined neurological structures, mutable only by thousands of generations of genetic trail and error.

One crucial idea behind this argument, then, is that adaptive traits are carried over from the periods in which they evolved by genes. And the random mutation and selective retention of genes is a process that requires thousands of generations. So let me begin by saying something about genes. For the reason just noted, genes figure prominently in Evolutionary Psychological writing. Although they reasonably enough protest when accused of holding that genes determine behaviour, they do generally hold that genes determine psychological mechanisms[1]. To quote Robert Wright: "They boil down to genes, of course (where else could rules for mental development ultimately reside?)" (1994, p.9).

So what are genes? It is not sufficiently widely known how difficult this question has become to answer. One possible answer goes back to the history of genetics and the Mendelian research programmes, particularly on fruit flies, of Morgan, Mueller, and others. This programme investigated hypothetical factors that were the heritable causes of differences between organisms. It became clear that these causes had something to do with chromosomes, and experiments on linkage, correlations between inherited traits, enabled the mapping of these factors as quasi-spatially related. When Crick and Watson famously published the chemical structure of DNA it was natural to suppose that these hypothetical factors could finally be identified with concrete material objects, parts of chromosomes or, that is, sequences of DNA molecules.

[1] This may not be true in more recent work by Cosmides and Tooby (e.g. 1992), but if not it is quite unclear how they can maintain the Stone Age adaptation story that is at the core of their programme.

This conclusion has turned out to be highly problematic, however. Certainly the phenotypic differences studied by classical geneticists could generally be identified with differences in the DNA sequence somewhere in the genome. Alternative bits of sequence with identifiable phenotypic effects are referred to as alleles, so that for example, we can talk about alleles for blue eyes or brown eyes which, more or less, follow the familiar Mendelian laws. However we should not assume that these alleles are readily identifiable objects. We can see this by looking at what may be the most important upshot of allelic selection, elimination of genomic errors. Medical genetics, because it is concerned precisely with harmful genomic errors, retains a strong connection with the tradition of classical genetics. But, to take one of the best known genetic diseases, cystic fibrosis, there is no object referred to by the expression 'gene for cystic fibrosis'. Cystic fibrosis is caused by a dysfunction in a protein that controls ion transfer across cell membranes. About 100 mutations have been identified in the genomic region that codes for this protein, with different mutations determining varying severity of symptoms in cystic fibrosis patients. The gene for cystic fibrosis, then, is a set of errors. Though in this case it would be correct to say that any of these mutations causes cystic fibrosis, it would be highly misleading to describe the unmutated sequence as a gene for not having cystic fibrosis.

As a matter of fact a very similar story can be told about the gene for blue eyes. Again, this is not a piece of DNA that somehow produces the blueness of eyes, but any of a range of errors in the DNA sequence that subvert the production of brown pigment in the eyes. And again, though there are therefore genes that cause blue eyes, it is at best misleading to think of the functional alleles at blue eye loci as causes of brown eyes. The

complexity of causal paths from bits of DNA to features of organisms makes the project of correlating things of these two kinds largely futile. Many different bits of DNA sequence and much else besides are involved in the normal production of a phenotypic trait. We can confidently assert that a bullet in the head was the cause of death, but it is problematic to suggest, except under very unusual circumstances, that the absence of a hole in the head is the cause of someone staying alive.

One might say that genes for brown eyes are parasitic on genes for blue eyes: if there were no identifiable effects of mutations in the relevant bit of DNA there would be no classical genes for either blue or brown eyes. And this follows merely from the quite uncontroversial point that the Mendelian concept applies only to differences. There is an irony here with some of the more acrimonious debate around Evolutionary Psychology. Critics of EP have suggested that Evolutionary Psychologists are involved in providing genetic explanations for human differences, between males and females or between homosexuals and heterosexuals, for example, and thereby reifying what are in fact superficial and malleable distinctions. Evolutionary Psychologists retort indignantly that their central concern is with the genetic basis for human universals. But in the only really clear sense of the word 'gene' there are no genes for universals. And that by definition, since genes are defined only by the differences they cause.

Am I suggesting that there is no genetic basis for normal development? Only, admittedly, in the pedantic sense that there are no well-defined entities, answering to the concept of genes, involved in normal development. But we can then say that there is a genomic basis for development: parts of the genome are crucially important. So why not

just call those parts of the genome genes, and stop quibbling? To answer this we need to look a bit more closely at the quite different concept of the gene employed in genomics.

When analysts of data from the human genome project report that there are about 30,000 genes therein, this estimate has nothing to do with relations to phenotypic traits. Very roughly speaking, what they mean is a sequence of coding DNA between a signal to start transcribing (that is, generating RNA sequence that may later be translated into amino acid sequence that may become part of a functional protein or enzyme) and a signal to stop transcribing. The fact that estimates of such numbers differ by as much as 10,000 indicates that this is not a simple matter, and a closer look at genomic activity makes this easy to understand.

The number of proteins produced in human cells is a more controversial issue than the number of genes, but typical estimates start at around 100,000 and range up to several times this number. Obviously this indicates that a gene, in the sense used by molecular biologists, can be involved in the production of many proteins. In fact these molecular genes are known usually to consist of alternating segments, known as exons and introns. In the simplest case, after the gene has been transcribed into RNA the introns are edited out and only the exons are translated into a protein. But in many or most cases different sequences of exons are composed by different editing processes, genes are 'altenatively spliced', and the same gene may give rise to many different proteins. In some cases products from parts of other genes, even the introns from other genes, are included in the splicing process. And further modifications to proteins occur after the edited RNA product has been translated into a protein. Cases are known in which several hundred different protein products are derived from the same gene.

Why does all this complexity matter? In the first place it contributes to dislodging a picture of the genome that still informs a good deal of thinking about evolution, not least human evolution, of the genome as some kind of blueprint or programme for the production of an organism. It begins to suggest, instead, something quite different, a repository of informational resources upon which the cell can draw in making a huge range of functional products. I think this is still misleading, because like the blueprint or programme metaphors it aims to replace, it sounds too static. The genome is located in a complex structure and the various forms that this structure adopts in the life of a cell are important to its functioning and its interactions with other components of the cell. A biologist colleague likes to define the genome as 'a space in which genetic events happen'. But for now the important point is to dislodge the metaphors that somehow suggest that the whole organism is somehow encoded in the genome, the idea that Lenny Moss has appropriately characterised as preformationist.

To get beyond this picture we need to look at another bit of biological dogma, the demise of which is perhaps less universally acknowledged, what is appropriately enough known as the Central Dogma of molecular biology. This dogma holds that information flows only from DNA to RNA and finally to amino acid sequence, never in the other direction. This may sound like nothing more than a characterisation of the basic steps in the production of functional proteins, but in fact it is widely used to lend support to the preformationist picture of the genome: since information only flows outwards from the DNA, it must all be contained in the DNA. At any rate, interpreted in anything more than the narrowest sense just indicated, it is completely false. In a way it seems obviously false. For what matters to the functioning of a cell is that the right functional products get

produced at the right time and in the right place and this is certainly effected in part by changes in the chemical environment of the cell. Still, if one held to the view of the genome as a programme, one could think of the cellular environment as something fully controlled by the genome and thereby effecting the appropriate expression of the necessary bits of DNA sequence at that point in the programme. To see that this picture cannot be sustained we need to look at more direct ways in which the central dogma is mistaken.

The most important, or at least the best understood, of these is methylation. This process, which has led some to refer to methylation as the fifth base in the DNA code, is a modification of the DNA structure that suppresses the expression of modified bits of sequence. This is a process that occurs throughout the life of a cell, and is certainly one of the crucial determinants of gene expression. While it was once thought that methylation was removed during the production of gametes, it is becomingly increasingly clear that this is by no means always the case. This leads us to what might well be called the Central Dogma of evolutionary biology, a dogma closely related to the previous Central Dogma, and one that has also become wholly untenable, the assumption that the only thing that is inherited is DNA. This brings us back to evolution.

One of the points of problematising the gene concept is to raise the question what kinds of genomic difference are in fact the important targets of selection. It seems increasingly likely that the importance of selection between alleles has been greatly exaggerated in recent evolutionary theory and it may indeed turn out, as most geneticists believed in the heyday of classical genetics, that this is largely a process of error elimination and not one capable of creating major evolutionary novelty. Getting rid of

the idea of genes for traits, too easily interpreted as objects with the specific function of causing those traits, should at least raise doubts about this idea.

Perhaps more importantly there is increasing awareness of a much greater range of possibilities for genomic changes that may provide far more promising bases for major evolutionary change. We have seen that a DNA sequence comprising a set of exons and introns may provide the basis for production of a large number of distinct products. Which, if any, of these it produces at a particular point of development and in a particular tissue will depend on a wide variety of factors: chemical modification of the genome, as for example in methylation, structural changes such as greater or lesser condensation of the chromatin, and the chemical species present in that cell at that time. There are parts of the genome capable of initiating cascades of developmental changes, and interestingly, these genetic triggers are generally extremely ancient, found in very distantly related organisms. As I shall explain further in a moment, some of these factors, and not merely those consisting of DNA sequence, can also be passed on to offspring.

Genomes themselves evolve in a great diversity of ways. Recombination, the result of random sampling from the genomes of two parents in producing an offspring, is standardly recognised as an important source of variation. But there are many other processes. Whole chromosomes and even genomes can be duplicated. These duplications are thought to be important in providing redundant genetic material in which large changes of organisation or sequence can occur without loss to the organism of essential functions. Smaller parts of genomes can be duplicated within or across chromosomes by inserting copies of themselves into the genome. Retrotransposons, a very important class of such genomic elements, which constitute a substantial proportion

of many genomes, appear to be important in the functional reorganisation of genomes. And, much more commonly than was once supposed, DNA from other organisms can be inserted into genomes.

It is interesting to reflect, in the light of some of these facts, on the surprise that is sometimes still expressed at the claim that human genomes are, say, 99.4% identical to those of chimpanzees. One may well wonder what exactly this means, but without worrying about that, we can certainly wonder why we should care. No doubt if the genome were a blueprint this would be quite surprising. If the blueprints for two ships, say, are 99.4% identical (without again worrying exactly what that means) we might expect two pretty similar vessels. But if we were told that they were made of an almost identical set of raw materials, or that they had identical engines, we would have no such preconceptions. The fact is that even if the genomes were 99.9 or 100% identical, nothing much would follow as to the degree of similarity of the organisms of which they were the genomes. It *is* an interesting and important discovery that parts of genomes are very strongly conserved through very much longer periods of evolutionary time, and substantial proportions of our genomes are almost identical to parts of the genomes of worms and even bacteria. This does not tempt us to wonder whether we are really rather similar to worms or germs, though it does direct us to look for bits of chemical machinery that we may share with very different creatures.

We may here recall some prescient remarks of Francois Jacob in 1977: "Biochemical changes do not seem … to be a main driving force in evolution...What distinguishes a butterfly from a lion, a hen from a fly, or a worm from a whale is much

less a difference in chemical constituents than in the organization and the distribution of these constituents…It is a matter of regulation rather than structure."

In sum, genomic function is a very different matter from genetic sequence and it is genomic function that provides the differences on which natural selection can work. It is likely that creation of entirely new bits of sequence has been greatly overstated as a central process in evolution and that redeployment of existing genomic resources may be much more important in producing large evolutionary changes. At any rate, research in genomics is opening up a wide range of possibilities for thinking about evolutionary change, and we should certainly not be committed to seeing the evolutionary process solely in the terms developed over fifty years ago.

Let me now come at the topic from a rather different direction, the philosophical analysis of evolution itself. Much of this has been focused on the so-called units of selection problem, the question what exactly does natural selection select. Thirty years ago it was fairly uncontroversial that the primary objectsof selection were individual organisms and perhaps also groups of organisms. Then came Richard Dawkins' notoriously successful popularisation of the ideas of G.C.Williams, and a great many people were convinced that ultimately the only possible unit of selection was the gene, understood by Dawkins in a broadly Mendelian way as a difference in the DNA sequence that made a difference to the phenotype. The crucial premise for this move was the claim that only genes were inherited. Whereas organisms invariably perished in an evolutionarily trivial length of time DNA, in Dawkins's colourfully hyperbolic term, was immortal. The structure of DNA was passed on intact from parent to child and hence that

structure was potentially immortal. Indeed, molecular biologists have been able to find large chunks of DNA more or less identical between ourselves and plants or even bacteria, and thus presumably preserved across the aeons of evolutionary time from our distant common ancestors.

But, impressive though this point may seem, the view that only DNA is inherited is quite unsustainable. And this is one of the reasons why most philosophers of biology, and some prominent evolutionists, have never been much convinced by the gene selection theory. It is easy to show that Dawkins' gene selection theory provides an inadequate model of evolutionary processes even if it is conceded that inheritance is solely mediated by DNA, and most philosophers concerned with these issues have accepted a pluralistic answer to the units of selection: selection acts on objects at a range of different scales, including genes, organisms, and very possibly groups of organisms. But we should not concede this view of inheritance. Broadening our understanding of inheritance suggests a much more radical rethinking of the units of selection problem that has been developed under the rubric of Developmental Systems Theory, or DST. DST, I shall suggest, provides a context in which we can understand the significance for evolution of the recent advances in genomics.

The Central Dogma of evolutionary theory stated that the only transgenerational vehicle of inheritance is the genome. The negative phase of DST provides a fundamental critique of this dogma. In very brief summary, it asks the question whether there is anything unique about DNA that justifies its privileged status in evolutionary models, and offers a negative answer. It has been claimed that DNA is unique in its ability to replicate itself. But DNA requires a range of other structures and substances for

replication and, with similar access to other resources, including DNA there is a wide range of structures that successfully replicate themselves in the course of development. The genome has been conceived as a privileged source of information. But it is easy to show that from an informational perspective the status of the genome is symmetrical with other contextual resources through which information is conveyed. Just as the cellular environment provides a channel for conveying information about the genome, the genome provides a channel for conveying information about its environment. Though the issue in not uncontroversial, DST has placed a strong burden of argument on those who wish to show how the genome has a unique status in biological organisation.

The positive claim of DST brings us back to the unit of selection. For DST this is the full life cycle of the organism. DST looks at the whole set of resources that are necessary for the reproduction of the life cycles of organisms, and the means by which parent organisms facilitate the availability of these resources for their offspring. This picture, of course, retains the basic Darwinian idea that evolutionary change is driven by the differential success that organisms have of launching, during their own life cycles, life cycles of organisms similar to themselves. By rejecting the picture of evolution as essentially no more than a sequence of gradually changing gene pools, this move makes room for the reintegration of development into evolutionary models.

It is plain that the restriction of inheritance to the genome cannot be right in the case of human reproduction. For a modern human in a developed modern society to successfully launch and sustain the life cycle of another modern human in that same society many other resources must be provided: maternal care in infancy, schools, hospitals, and much else. And these resources affect the course of development. Despite

debates about the importance of innate underlying cognitive structures, it is impossible to deny that a human developing with access to the full range of such developmental resources will acquire a range of capacities—from reading and writing, to appropriate table manners and locally appropriate dress sense—not available to one denied these resources.

Such cultural developmental resources are not unique to humans. Birds must provide nests, termites must construct mounds, and beavers must build dams if they are to be successful in reproducing their respective kinds. No doubt there are greater or lesser innate dispositions displayed in these acts of provision—lesser for birds than for termites, for instance. But often the experience of exploiting the resource will also provide some of the information necessary for reproducing the resource when they become parents. Many species of birds, for instance, learn by imitation the songs necessary for attracting reproductive partners.

These developmental resources fully external to the bodies of the reproducing and reproduced life cycles are of obvious importance in human evolution as current human reproduction involves a vast infrastructure of resources that are maintained and improved upon by successive generations. What is less obvious is the genome is not a unique bearer even of internal heritable information. A consequence of the critical work of DST has been to dismantle the conceptual firewall that some have tried to construct around the genetic to preserve its privileged place in evolutionary models. In reality, the minimum physical material passed on to an organism in reproduction is a single cell. The female egg contains a vast set of chemical materials. Though the production of these chemicals depends on genomic resources, but as I have stressed, the genome contains resources that

could in principle produce an unimaginably large set of different chemical environments. The transmission of one particular set is potentially a transgenerational transmission of information of a complexity not incomparable with the transmission of the genome itself. And as I have mentioned, functionally important modifications of the genome itself, such as methylation, are also transmitted to an extent that remains unclear and, indicatively of its pivotal ideological role, highly controversial.

An obvious consequence of transmission outside the body is that this sort of inheritance is Lamarckian. By this I refer (with apologies to Lamarck) to the inheritance of acquired characteristics. Schools, for instance, allow the acquisition of characteristics that can be transmitted to future generations. A consequence, or ideological function, of the dogma of only genetic inheritance is to emphasise the intellectual iniquity of Lamarckism. The phenomenon of methylation is one of a range of recent biological insights that threaten to open more fully the Lamarckian Pandora's Box. This is more controversial terrain than I have so far ventured into, but some recent results are suggestive.

It is well established, for instance, that maternal care in rats affects gene expression in the brain of pups, and does so in part through methylation. Rats deprived of maternal care in infancy grow up more fearful and show stronger hormonal response to stress than normally nurtured rats. There is evidence that these changes in methylation are directly heritable, though of course they could surely be indirectly heritable through changes in the maternal behaviour of mother rats themselves deprived of maternal care. There are data showing that low birth weight of children born during the Dutch famine of 1944-5 not only had increased susceptibility to various later life illnesses, but passed this

susceptibility on to their children, and epigenetic effects such as abnormal methylation patterns provide a plausible explanation. One other well documented case is the effect of social status on the production of dopamine receptors in the brain. Higher ranking Macaque monkeys turn out to be less susceptible to cocaine addiction than monkeys that they socially outranked. This difference was traced to the fact that exposure to lower ranking monkeys, but not to higher ranking ones, effected changes in the expression of genes in the monkey's brains, specifically to the production of dopamine D2 receptors. I don't know whether these changes are heritable, but certainly mechanisms exist whereby they could turn out to be.

Less controversial are effects of the environment that are not directed, but affect the rate of evolutionary change. There is work going back to Barbara McClintock that shows that the activity of retrotransposons, genetic elements that replicate themselves throughout the genome, is increased when plants experience stress. This will tend to cause genomic reorganisation that can provide material for rapid evolutionary change. There is, at any rate, considerable evidence that these elements, which constitute a very substantial proportion of most genomes including ours, have important effects on gene expression, and can have decisive effects in early embryogenesis, which is of course the point at which the largest effects on development can be expected.

Perhaps most intriguing of all are the small, non-protein-coding RNAs that are proving to be omnipresent in cells and to have vital, diverse, but very partially understood, functions. They appear able to bind to DNA, inhibiting its expression, they can control the activity of protein coding RNAs, and some can even bind to proteins, altering their behaviour. It would be impossible to begin to describe the intriguing

findings that are beginning to emerge from this incipient research filed, but it seems clear that it represents an entirely new level of cellular control. Small RNAs also have the ability to move between cells, and may prove to have important communicative functions between tissues. And, of course, a set of these RNA fragments is part of what is transmitted with the maternal ovum in reproduction.

A very radical and heretical view of evolution, most forcefully presented by Mary Jane West Eberhard, suggests that adaptation is, in the first instance, a process of organismic response to the environment, facilitated by the developmental plasticity of organisms. Genomic adaptation follows. So far from selection among genes being the primary force behind adaptation, it is largely a consequence of phenotypic adaptation. Perhaps then we are well adapted to modern life, but our genomes are still catching up.

I have done no more than gesture at some of the extraordinary insights that are currently emerging in molecular biology. Why should we care? Recall the basic argument underlying the Stone Age origin of the human mind. Essentially the mind is a product of the genome. Behaviour, to be sure, responds differentially to environmental circumstances, but the basic structure of the mind is laid down in the genes. This is a thoroughly bottom up picture. Genes, as the dogma has it, produce RNA, which produces proteins. Proteins provide the predetermined structure that then interacts in a determinate way to environmental contingencies. We are not, as the Evolutionary Psychologists insist, exactly programmed to be rapists, but given the right set of stimuli in which our Stone Age minds calculate rape as the best reproductive strategy, rapists we become. And finally, the most fundamentally bottom-up part of the picture is the model

of evolution that claims that evolutionary history is, in essence, no more than a sequence of genomes, each slightly modified to improve on its predecessor.

I have tried to indicate that this picture is entirely obsolete and unrelated both to contemporary molecular biology and the most plausible understanding of the evolutionary process. Certainly these bottom-up processes are important, but equally important are simultaneous top-down processes. The environment does not just shape the human mind in the uncontroversial sense of filling in gaps in a pre-existing structure—speaking English rather than French, or knowing which social rules to monitor for cheats. As shown by the high status monkeys who just say No to drugs, social factors can influence the expression of genes in the brain, and basic brain chemistry. Gross morphology can affect the shape of cells which can effect the chemical functions within cells, a process that has been found to be very significant in early cell differentiation. DNA produces RNA, but while some of that RNA contributes, in very complex ways, to the coding for protein sequences, other bits feed back on the function of DNA or on the splicing and translation of coding RNAs. Proteins also feed back on the expression of DNA or contribute to the physical structure of DNA, also an essential determinant of gene expression.

Hence in reproduction it is not just a set of genes that is passed on to descendants, but an exquisitely complex and dynamic chemical system of which the genome is just one vital interacting part. And to the extent that organisms shape the environment in which their offspring are found either purposefully, as is carried to by far the highest level by our own species, or simply as a by-product of their characteristic behaviours, this will also affect the developmental sequence of chemical environments in the

differentiating cell lines. How effective at tracking exogenous changes in the environment such a system will prove to be over evolutionary time is not to be settled by abstract calculations on the trajectories of naked DNA. Certainly there can be maladaptive time lags in evolutionary processes, but these are to be discovered empirically rather than proved a priori.

Let me summarise these conclusions by returning to the question of the universality and diversity of human nature. Evolutionary psychologists respond to the accusation that by seeing human nature in the genes they are reifying differences between people, by insisting that their primary concern is with the common genetic inheritance that we have all inherited from our Flintstone ancestors. Still, where there are evident differences between people, as for example between homosexuals and heterosexuals, these must be located in the genes, and silly stories are made up about Stone Age homosexual shamans providing for their nephews and nieces. It is true that we are an unusually genetically homogeneous species, so perhaps these differences are not so great. An important exception, of course, is the difference between men and women. Since the genetic difference between a human male and a human female exceeds that between a human male and a male chimpanzee it is not surprising that Evolutionary Psychologists have portrayed men and women almost as if they belonged to different species—perhaps even came from different planets.

The picture I have sketched is neutral on the uniformity of human nature, in large part because I am sceptical about the usefulness of this concept. Of course there is a vast amount of human biology common to the human species, some of which we are just

beginning to understand. The problem with the concept of human nature is that it tends to suggest fixity, indeed something like a traditional human essence. Of course we have mostly learned to reject biological essences as incompatible with evolution, but still in the time scales that matter to us, this may seem a pedantic difference. And indeed, though they certainly admit that we have evolved and are probably evolving still, Evolutionary Psychologists perfectly illustrate this effective essentialism with the claim that human nature is, as far as matters to us, stuck in the Stone Age.

I object that there is no reason to suppose that we are stuck in the Stone Age, and indeed that we are very likely quite well adapted to the twenty-first century. And it is possible that we may soon be adapted to something quite different. Is this an assertion of the blank slate view of the human mind so violently denounced by Stephen Pinker? Not at all. Human development is a much more complex process than the crude genetic determinism supposed by Pinker, but its very complexity may make it difficult to change in predictable ways. My point is just that organisms in general, and ourselves in particular are much more subtle and interesting than the antiquated biological picture I have criticised suggests. In evolutionary time there are many ways in which they may respond to changing environments: partly by changing their genomes, though probably the important changes to the genome amount to the redeployment of existing genomic resources, and perhaps more importantly still by changing environmental factors that elicit new employments of existing resources both genomic and more widely biological. Because for our species many changes in this last category can be purposefully effected, it is possible that significant evolution could have happened very rapidly. And the great differences in all except genomes between ourselves and our nearest relatives clearly

point to the conclusion that it has. This evolutionary flexibility is, from a proper perspective, inextricably connected to developmental flexibility. It is not, as the accusation of blank slateism suggests, a trivial matter of producing whatever developmental outcomes that we might like to imagine, but nor is the production of new developmental outcomes something ineluctably barred by genetic fate. Human differences, the diversity, however much there may be, in actual human developmental outcomes is our best clue as to the diversity of outcomes that might be achieved with a will and a better understanding of human development.

No doubt there are many kinds of time lags. Much of our genomic machinery is inherited from simple organisms billions of years ago, though it seems to be rather adaptable to new uses. At the opposite extreme rapid social change produces developmental obsolescence. People of my generation are surely less well-adapted to the age of information technology than will be today's teenagers. And it may be that we have deeply engrained tendencies of behavioural development that stem from exigencies of some part of our evolutionary history. But if so this needs to be empirically demonstrated in detail, not proved by a priori argument. And even if such atavistic defects are demonstrated, there is no reason to suppose that they are somehow immutable.

One message of this talk is a sceptical one. The more we understand of contemporary biology, the more we see how much we don't know. We still understand very little of the development of the simplest organisms, let alone the most complex. How, as our knowledge of molecular biology and ontogeny develop, these will bear on more refined understandings of the process and tempo of evolution, is perhaps even more difficult to discern. The biology underlying Evolutionary Psychology, at least can be

confidently rejected as based on assumptions that have unravelled in the last couple of decades. Conclusions drawn from so rickety a base about a matter as important to us as Human Nature should be rejected not only for the epistemological worthlessness, but because groundless guesses in this area can be extremely dangerous.

Let me end where I began, with the Krispy Kreme donuts. There can of course be no doubt that biological facts about humans, a part even of human nature, are engaged in the attraction of many of us to fat and sugar. These are, after all, good sources of energy that we are physiologically equipped to exploit. But what is interesting about the case is the diversity of human responses to the omnipresence of these resources. Obesity is not an inevitable response to the overabundance of cheap calories. In fact, and unsurprisingly, obesity seems to arise most strongly where overabundance intersects with poverty, that is among poor people in rich countries. Unfortunately such observations do not differentiate between the hypothesis of a fixed psychology responding to varying circumstances and a variable psychology developing in response to varying environments.

So how do we choose between these alternatives? Are we genetically programmed fat-guzzlers sucked inexorably towards the donuts, or are we blank slates, haphazardly imprinted with the culture of Mars bars or a healthy bourgeois love of broccoli? Of course we are neither. The way to break down the dichotomy between these equally hopeless alternatives is to begin to appreciate the intricate hierarchy of upward and downward interactions between objects and structures at all levels of the biological hierarchy. In doing so we dispense with the stultifying dogmas I have mentioned in this talk, and we see the importance of a perspective on evolution that

encompasses the diversity of processes susceptible to selective change. And, finally, we can begin to understand the vast changes in human behaviour that have occurred over the last few thousand years without seeing ourselves either as formless lumps of pscyhoplasm or atavistic relics from the mists of prehistory.