
CA☆ FORUM ON
ANTHROPOLOGY IN PUBLIC

Genes and Cultures

What Creates Our Behavioral Phenome?

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A central theme of the flood of literature in recent years in “evolutionary psychology” and “behavioral genetics” is that much or even most human behavior has been programmed into the human genome by natural selection. We show that this conclusion is without basis. Evolutionary psychology is a series of “just-so” stories rooted in part in the erroneous notion that human beings during the Pleistocene all lived in the same environment of evolutionary adaptation. Behavioral genetics is based on a confusion of the information contained in a technical statistic called “heritability” with the colloquial meaning of the term, exacerbated by oversimplification of statistical models for the behavioral similarity of twins. In fact, information from twin studies, cross-fostering, sexual behavior, and the Human Genome Project makes it abundantly clear that most interesting aspects of the human behavioral phenome are programmed into the brain by the environment. The general confusion created by the genetic determinists has had and will continue to have unfortunate effects on public policy.

The recent publication of the first draft of the human genome (e.g., Venter et al. 2001, Lander et al. 2001) has brought to public attention the relationship between two concepts, genotype and phenotype—a relationship that had previously been discussed largely by academics. The genotype of an organism is encoded in the DNA that is held in chromosomes and other structures inside its cells. The phenotype is what we are able to observe about that organism’s biochemistry, physiology, morphology, and behaviors. We will use the term “phenome” to circumscribe a set of phenotypes whose properties and variability we wish to study. Our focus will be on that part of the human phenome that is defined by behaviors and especially on the behavioral phenome’s connection with the human genome.

Our understanding of human behavioral traits has evolved; explanations of the control of those traits offered 50 years ago differ from those most common today. In prewar decades genetic determinism—the idea that genes are destiny—had enormous influence on public policy in many countries: on American immigration and racial policies, Swedish sterilization programs, and, of course, Nazi laws on racial purity (Ehmann 2001, Ehrlich and Feldman 1977, Fisher 2001). Much of this public policy was built on support from biological, medical, and social scientists (e.g., Brigham 1923, Goddard 1917, Terman 1916), but after Hitler’s genocidal policies it was no longer politically correct to focus on putative hereditary differences. The fading of genetic determinism was an understandable reaction to Nazism and related racial, sexual, and religious prejudices which had long been prevalent in the United States and elsewhere. Thus, after World War II, it became the norm in American academia to consider all of human behavior as originating in the environment—in the way people were raised and the social contexts in which they lived.

Gradually, though, beginning in the 1960s, books like Robert Ardrey’s *Territorial Imperative* (1966) and Desmond Morris’s *The Naked Ape* (1967) began proposing explanations for human behaviors that were biologically reductionist and essentially genetic. Their extreme hereditarian bias may have been stimulated by the rapid progress at that time in understanding of the role of DNA, which spurred interest in genetics in both scientists and the public. But perhaps no publication had broader effect in reestablishing genetic credibility in the behavioral sciences than Arthur Jensen’s (1969) article “How Much Can We Boost IQ?” Although roundly criticized by quantitative geneticists and shown to be based on the fraudulent data of Sir Cyril Burt (Kamin 1974), Jensen’s work established a tradition that attempts to allocate to genetics a considerable portion of the variation in such human behaviors as for whom we vote, how religious we are, how likely we are to take risks, and, of course, measured IQ and school performance. This tradition is alive and well today (e.g., Plomin, Owen and McGuffin 1994, Plomin et al. 1997).

Within the normal range of human phenotypic variation, including commonly occurring diseases, the role

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of genetics remains a matter of controversy even as more is revealed about variation at the level of DNA (Pritchard 2001, Reich and Lander 2001). Here we would like to reexamine the issue of genetics and human behavior in light of the enormous interest in the Human Genome Project, the expansion of behavioral genetics as described above, and the recent proliferation of books emphasizing the genetic programming of every behavior from rape (Thornhill and Palmer 2000) to the learning of grammar (Pinker 1994). The philosopher Helena Cronin and her coeditor, Oliver Curry, tell us in the introduction to Yale University Press's "Darwinism Today" series that "Darwinian ideas . . . are setting today's intellectual agenda" (1999). In the *New York Times*, Nicholas Wade (2000) has written that human genes contain the "behavioral instructions" for "instincts to slaughter or show mercy, the contexts for love and hatred, the taste for obedience or rebellion—they are the determinants of human nature."

Genes, Cultures, and Behavior

It is incontrovertible that human beings are a product of evolution, but with respect to behavior that evolutionary process involves chance, natural selection, and, especially in the case of human beings, transmission and alteration of a body of extragenetic information called "culture." Cultural evolution, a process very different from genetic evolution by natural selection, has played a central role in producing our behaviors (Cavalli-Sforza and Feldman 1973, 1981; Ehrlich 2000; Feldman and Cavalli-Sforza 1976; Feldman and Laland 1996).

This is not to say that genes are uninvolved in human behavior. *Every* aspect of a person's phenome is a product of interaction between genome and environment. An obvious example of genetic involvement in the behavioral phenome is the degree to which most people use vision to orient themselves—in doing everything from hitting a baseball to selecting new clothes for their children. This is because we have evolved genetically to be "sight animals"—our dominant perceptual system is vision, with hearing coming in second. Had we, like dogs, evolved more sophisticated chemical detection, we might behave very differently in response to the toxic chemicals in our environment (Ehrlich 2000). The information in our DNA required to produce the basic morphology and physiology that make sight so important to us has clearly been molded by natural selection. And the physical increase in human brain size, which certainly involved a response to natural selection (although the precise environmental factors causing this selection remain something of a mystery [Allman 1999, Klein 1999]), has allowed us to evolve language, a high level of tool use, the ability to plan for the future, and a wide range of other behaviors not seen in other animals.

Thus at the very least, genetic evolution both biased our ability to perceive the world and gave us the capacity to develop a vast culture. But the long-running nature-versus-nurture debate is not about sight versus smell. It

is about the degree to which differences in today's human behavioral patterns from person to person, group to group, and society to society are influenced by genetic differences, that is, are traceable to differences in human genetic endowments. Do men "naturally" want to mate with as many women as possible while women "naturally" want to be more cautious in choosing their copulatory partners (Bermant 1976, Symons 1979, Birkhead 2000; see also Small 1993: chap. 7)? Is there a "gay gene" (Hamer et al. 1993, Hu et al. 1995, Rice et al. 1999)? Are human beings "innately" aggressive (Ehrlich 2000: 210–13)? Are differences in educational achievement or income "caused" by differences in genes (Bowles and Gintis 2001, Jacoby and Glauber 1995, Lewontin, Rose, and Kamin 1984, Taubman 1976)? And are people of all groups genetically programmed to be selfish (Hamilton 1964, Richerson and Boyd 1978)? A critical social issue to keep in mind throughout our discussion is what the response of our society would be if we knew the answer to these questions. Two related schools of thought take the view that genetic evolution explains much of the human behavioral phenome; they are known as evolutionary psychology and behavioral genetics.

Evolutionary Psychology

Evolutionary psychology claims that many human behaviors became universally fixed as a result of natural selection acting during the environment of evolutionary adaptation (Tooby and Cosmides 1992), essentially the Pleistocene. A shortcoming of this argument, as emphasized by the anthropologist Robert Foley (1995–96), lies in the nonexistence of such an environment. Our ancestors lived in a wide diversity of habitats, and the impacts of the many environmental changes (e.g., glaciations) over the past million years differed geographically among their varied surroundings. Evolutionary psychologists also postulate that natural selection produced modules ("complex structures that are functionally organized for processing information" [Tooby and Cosmides 1992: 33]) in the brain that "tell" us such things as which individuals are likely to cheat, which mates are likely to give us the best or most offspring, and how to form the best coalitions (Kurzban, Tooby, and Cosmides 2001). These brain "modules," which are assumed to be biological entities fixed in humans by evolution, also have other names often bestowed on them by the same writers, such as "computational machines," "decision-making algorithms," "specialized systems," "inference engines," and "reasoning mechanisms" (Duchaine, Cosmides, and Tooby 2001). The research claims of evolutionary psychology have been heavily criticized by, among others, colleagues in psychology (e.g., Bussey and Bandura 1999).

Those critics are correct. There is a general tendency for evolutionary psychologists vastly to overestimate how much of human behavior is primarily traceable to biological universals that are reflected in our genes. One reason for this overestimation is the ease with which a

little evolutionary story can be invented to explain almost any observed pattern of behavior. For example, it seems logical that natural selection would result in the coding of a fear of snakes and spiders into our DNA, as the evolutionary psychologist Steven Pinker thinks (1997: 386–89). But while Pinker may have genes that make him fear snakes, as the evolutionist Jared Diamond points out, such genes are clearly lacking in New Guinea natives. As Diamond says, “If there is any single place in the world where we might expect an innate fear of snakes among native peoples, it would be in New Guinea, where one-third or more of the snake species are poisonous, and certain non-poisonous constrictor snakes are sufficiently big to be dangerous.” Yet there is no sign of innate fear of snakes or spiders among the indigenous people, and children regularly “capture large spiders, singe off the legs and hairs, and eat the bodies. The people there laugh at the idea of an inborn phobia about snakes, and account for the fear in Europeans as a result of their stupidity in being unable to distinguish which snakes might be dangerous” (1993: 265). Furthermore, there is reason to believe that fear of snakes in other primates is largely learned as well (Mineka, Keir, and Price 1981, Mineka and Cook 1993).

Another example is the set of predictions advanced by Bruce Ellis (1992) about the mating behavior that would be found in a previously unknown culture. The first five characteristics that “the average woman in this culture will seek . . . in her ideal mate,” he predicts, are (p. 283):

1. He will be dependable, emotionally stable and mature, and kind/considerate toward her.
2. He will be generous. He may communicate a spirit of caring through a willingness to share time and whatever commodities are valued in this culture with the woman in question.
3. He will be ambitious and perceived by the woman in question as clever or intelligent.
4. He will be genuinely interested in the woman in question, and she in him. He may express his interest through displays of concern for her well-being.
5. He will have a strong social presence and be well liked and respected by others. He will possess a strong sense of efficacy, confidence, and self-respect.

Evolutionary theory does not support such predictions, even if an “average woman” could be defined. First of all, it would be no small developmental trick genetically to program detailed, different, and *independent* reproductive strategies into modules in male and female brains. Those brains, after all are minor variants of the same incredibly complex structures, and, furthermore, the degree to which they are organized into modules is far from clear (Ehrlich 2000: 115–19). If the women in the unknown culture actually chose mates meeting Ellis’s criteria, a quite sufficient alternative evolutionary explanation would be that women (simultaneously with men) have evolved big brains, are not stupid, and respond to the norms of their cultures. Scientifically, the notion that the detailed attributes of desirable mates must be

engraved in our genetic makeup is without basis, especially in light of the enormous cultural differences in sexual preferences.

For any culture, Ellis’s evolutionary arguments would require that in past populations of women there were DNA-based differences that made some more likely to choose in those ways and others more likely to seek mates with other characteristics. And those that chose as Ellis predicts would have to have borne and raised more children that survived to reproduce than those with other preferences. Might, for example, a woman who married a stingy male who kept her barefoot and pregnant out-reproduce the wife of a generous and considerate mate? That is the way genetic evolution changes the characteristics of populations over time: by some genetic variants’ out-reproducing others. When that happens, we say that natural selection has occurred. But, unfortunately, there are no data that speak to whether there is (or was) genetic variation in human mate preferences—variation in, say, ability to evaluate specifically whether a potential mate is “ambitious”—upon which selection could be based. And there are no data for any population showing that women who seek those characteristics in their sexual partners are more successful reproductively—are represented by more children in the subsequent generation—than women who seek husbands with other characteristics. Ellis is simply confusing the preferences of women he knows in his society with evolutionary fitness.

Behavioral Genetics and Heritability

Another reason laypersons tend to overestimate how much of our behavior is genetically determined derives from the claims of some scientists that the variation between individuals in behaviors is due to their genetic differences. Often these same scientists look to advances in molecular genetics as a kind of justification. The following quote from a recent book by the biochemists Dean Hamer and Peter Copeland (1998) reflects the attitude of many behavioral geneticists: “The emerging science of molecular biology has made startling discoveries that show beyond a doubt that genes are the single most important factor that distinguishes one person from another. We come in large part ready-made from the factory. We accept that we look like our parents and other blood relatives; we have a harder time with the idea that we *act* like them” (p. 11).

Hamer and Copeland’s view has a long history. For example, in his influential monograph, Arthur Jensen (1969) claimed that the high heritability (a statistical value) of IQ made it unlikely that environmental intervention could succeed in improving the educational performance of disadvantaged children; the fault lay in their genes. The next three decades saw the growth of behavioral genetics, a part of psychology built around the statistical comparison of identical and fraternal twins. Most of these studies follow Jensen’s example and produce a high heritability, which is wrongly interpreted as a mea-

sure of how important genes are in determining differences between individuals in the behavior under study. For instance, the law professor Kingsley Browne (1998: 27) tells us that "evidence from behavioral genetics indicates that many personality traits are highly heritable; that is, much of their individual variation is attributable to genetic differences among individuals." We often see headlines in major newspapers that summarize the claims of behavioral geneticists with "Gene for Happiness Found" or "We Vote with Our Genes." But the text is not really about genes but about the behavioral geneticists' interpretation of their own estimates of heritability computed from twin studies.

The behavioral genetics literature is based on studies of identical and fraternal twins combined with a set of statistical assumptions about genetic and environmental contributions that are used to extract estimates of how important genes are in determining behaviors (e.g., Plomin et al. 1997). We shall examine this heritability paradigm in some detail and then see what new knowledge about the human genome can tell us about it.

Heritability was originally introduced in the 1930s as an index of amenability to selective breeding of agricultural animals and plants (e.g., Lush 1945; Falconer and Mackay 1996: chap. 10). Under carefully controlled environmental conditions it measures the fraction of genetic variation that would respond to selection by the breeder on a trait such as fat content in milk or egg production in chickens. An accurate measure of heritability requires that parents and offspring be raised in identical environments. This original narrow definition was predictive—it told the experimenter what had to be done to move the desired trait in a given direction by a given amount. The definition of heritability was later modified, broadened in fact, to include genetic variation that was unresponsive to selection and to accommodate the fact that genotypes and environments might interact in a way that could not be estimated or controlled, especially in the case of human behaviors² (Falconer and Mackay 1996: 123).

2. The usual model in behavioral genetics takes the phenotype to be a linear combination of genetic and environmental effects: $P = hG + eE$. In this statistical representation, the square of h is the broad-sense heritability, a number between 0 and 1.0 (or 100%), e is the corresponding fraction of the phenotype due to the (nontransmitted) environment E , and G and E operate independently. It is in the quantity G that the action of the DNA is summarized. For a single gene, the contributions to an individual's phenotype that come from maternal and paternal contributions (alleles) may be independent and summed, in which case the action of that gene is purely *additive*, or they may interact in some way that is measured by genetic *dominance*. When two or more genes interact to produce their contributions to the phenotype, we call the genes "epistatic," and the part of their contribution to the variability of the phenotype that is not the sum of their individual contributions is called *epistasis*. These are all statistical notions about variance that are very difficult to translate into genetic structural or regulatory phenomena. The fraction of the variance of P that is due to variation in the additive contributions to G is called the "narrow-sense" heritability. The fraction of the variance of P due to G and to possible interactions between G and E is called the "broad-sense" heritability. The latter is what is most often referred to as *the* heritability in behavioral genetics.

This broad-sense heritability has no predictive value and indeed cannot be legitimately used in the human behavioral context to predict anything. It has, however, been widely misinterpreted as diagnostic of the underlying causes of variation. Thus, in a recent perspective in the widely read magazine *Science*, the behavioral geneticists P. McGuffin, B. Riley, and R. Plomin (2001) infer that "DNA variations are responsible for the ubiquitous genetic influence in behavior" from the claim that "the most solid genetic findings about individual differences in human behavior come from quantitative genetic research such as twin and adoption studies that consistently converge on the conclusion that genetic variation makes a substantial contribution to phenotypic variation for all behavioral domains." In other words, they claim that the statistical measure of broad-sense heritability is telling us about the causes of the behavioral differences, in particular how "genetic" they are.

The kind of statistical reasoning that underlies this imputed connection between the information coded into DNA and heritability relies on a particular model of how that information causes behaviors, a model that is not verifiable because we have no idea about how the complex interactions between genes, regulation of genes, protein structures, protein concentrations, and environments would be manifest in a measurable trait or behavior. Scientists don't know what model to use to compute the degree of "genetic causation." In one class of such models, for example, Robert Cloninger and colleagues (1979) showed that heritabilities were made very high by using the doubtful assumption that identical and fraternal twins had the same degree of similarity in their environments. In another analysis, Devlin, Daniels, and Roeder (1997) showed that omission of a contribution from the shared prenatal environment of twins also leads to elevated estimates of heritability. In fact, calculated heritabilities give us *no* information concerning the *causes* of our actions. The basic reason is that it is impossible to distinguish human behavioral phenomes that are shared because of genetic similarities from those caused by shared environments. We might act like our parents because they gave us our genes; however, as Richard Lewontin pointed out, "In the United States, the highest correlations between parent and offspring for any social traits are for religious sect and political party. Only the most vulgar hereditarian would suggest that Episcopalianism and Republicanism are directly coded for in the genes" (Lewontin, Rose, and Kamin 1984: 256).

Much has been recently made by behavioral geneticists of heritability estimates for behavioral traits based on data compiled in two twin studies: the Minnesota Study of Twins Reared Apart (MISTRA) and the Swedish Adoption-Twin Study of Aging (SATSA). Results from these studies are widely cited in textbooks on the genetics of human behaviors (e.g., Plomin et al. 1997), but it is only recently that the statistical assumptions underlying these analyses and the inconsistencies in the reporting of estimates have come under careful scrutiny (Devlin, Daniels, and Roeder 1997, Feldman and Otto 1997, Goldberger and Kamin 2002). Goldberger and Ka-

min point out that “the only genetical theory involved in their analysis are the numbers 1, 1/2, and 1/4 representing³ the genotypic correlations for identical twins and the additive and non-additive genotype correlations for fraternal twins.” Not only do these authors find the conclusions from MISTRA and SATSA unconvincing but they raise the important question, ignored in the now large literature on behavioral correlations among relatives, “What conceivable purpose is served by the flood of heritability estimates generated by these studies?”

Policy Implications of Heritability

Perhaps most important, degree of heritability carries no message about how easily a characteristic can be changed, and, normally, knowledge of it will have few if any policy implications. Heritable diseases are routinely treated (e.g., phenylketonuria), as are diseases believed to have little relationship to the victim’s genetic endowment (e.g., endocarditis). Similarly, even if a behavior had a high degree of heritability in one environment, a small environmental alteration could totally change that behavior. The literature on quantitative traits in plants, insects, and animals is replete with experiments that show the sensitivity of measured heritability to changes in the environment.

Furthermore, it would be foolish to make social policy designed to alter behavior on the basis of group averages in characteristics, regardless of the reasons for the differences in those characteristics. Consider a thought experiment on the frequently promoted (with no evidence) view that there are differences between populations in genes influencing intelligence. Suppose that, counter to everything geneticists know, there were something that could be called “genetic IQ” and some way were discovered to assess it—some sort of cognitive litmus paper on which, when placed on the forehead, a number miraculously appeared, faultlessly indexing the “genetic IQ” of that individual. Suppose further that average “genetic IQ” litmus-test scores tended to be somewhat higher in the black population, even though many whites scored much higher than many blacks, some at the “gen-

ius” level. Would it then be good policy to give remedial aid to all whites and none to any blacks? Or would it be wiser to give additional help to those who had low scores regardless of skin color? What, in fact, would be the reason for even bothering to calculate the group average IQ scores? Would we calculate them for populations differentiated on the basis of other characteristics, such as blood groups? In fact, the usual physical and/or cultural criteria used to define ethnic groups may have little to do with the genetic classification of such groups (Wilson et al. 2001).

It is only because people live in socially stratified societies and have a fascination with skin color (or height, or nose shape—after all, we are sight animals) that differences between certain groups are singled out for investigation via heritabilities. If average differences in IQ test scores are correlated with skin color in our society, should we try to reduce the incidence of low test scores by treating skin-color groups differently? Of course not, any more than we would attempt to lower the incidence of skin cancer (to which lighter-skinned people are more susceptible) by doling out sunscreen on the basis of IQ test scores. Smart social policy would be to aid individual students with low scores regardless of skin color and regardless of what role genes played in determining individual IQs.

Jensen’s proposed heritability of 80% for IQ should never have been used to blame the failure in school of some groups of students on their genes. Nor should Herrnstein and Murray have used their value of 60% (which they feel “may err on the low side”) to underpin their claim that “chances of success in life are increasingly affected by genes” (1994: 109–10) and their reiteration 25 years later of Jensen’s claim that environmental intervention in the lives of the disadvantaged in the United States was doomed to failure (pp. 551–52).

It has been fascinating and disturbing for us as biologists to watch the legacy of Jensen’s 1969 opus unfold in psychology. Thus, one of the leading proponents of the use of heritability, Robert Plomin, writing in the *The Psychologist*, claims that “during the 1980s and especially the 1990s psychology became much more accepting of genetic influence, as can be seen in the increasing number of behavioral genetic articles in mainstream psychology journals and in research grants” (Plomin 2001). He goes on to describe this change in psychology as a “wave of acceptance of genetic influence in psychology.” This acceptance is entirely due to the widespread acceptance of the statistical methodology that leads to the reporting of broad-sense heritability and its misinterpretation as an index of genetic causality, not to any neurogenetic advances that have tied human behavioral differences to variation in DNA. In a similar vein, the psychologist M. McGue (1997) claims, for example, that “the IQ debate now centers on whether IQ is 50% to 70% heritable.”

Our point is that the assumptions used to build the statistical models that produce these estimates do not permit us to infer from such heritability estimates the actual extent of “genetic influences” on IQ. Further,

3. Referring to the model of n. 1, $P = hG + eE$, for any two individuals labeled 1 and 2, we can write $P_1 = hG_1 + eE_1$ and $P_2 = hG_2 + eE_2$ where the genetic and environmental contributions to the phenotypes P_1 and P_2 of the two individuals are G_1, G_2 and E_1, E_2 , respectively. Now, for identical or monozygous (MZ) twins G_1 and G_2 are the same because their complete complement of genes is the same; the correlation between G_1 and G_2 is 1. If individuals 1 and 2 are sibs, then it can be shown that the correlation between the additive contributions to P_1 and P_2 contained in G_1 and G_2 is 1/2 while that between the dominance contributions is 1/4 (see, e.g., Falconer and Mackay 1996 for more details). Almost all behavioral genetic studies assume that the correlation between environments E_1 and E_2 when individuals 1 and 2 are MZ twins is the same as when they are fraternal or dizygous (DZ) twins. Yet when there are enough data for these correlations to be compared, for example, for IQ, the MZ value is larger than the DZ value. If this difference in environmental correlations is ignored, a higher estimate for heritability is reported (Cloninger, Rice, and Reich 1979, Feldman and Otto 1997).

these estimates do not inform potential strategies for determining the nature of such genetic influences, if they exist. Applications of broad-sense heritability to predictive situations are, we repeat, biologically and statistically erroneous (Feldman and Lewontin 1975). Evolutionary psychologists and behavioral geneticists persist in confounding a technically defined statistic named “heritability” with the colloquial use of that term. The concept of overall heritability should be restricted in its employment to plant and animal breeding, where it can be better measured and the results put to some practical use—such as in applying selection to increase the rate of growth of beef cattle or the weight of swine.

What Does Determine the Behavioral Phenome?

Geneticists know that a large portion of the behavioral phenome must be programmed into the brain by factors in the environment, including the internal environment in which the fetus develops and, most important, the cultural environment in which human beings spend their entire lives. Behavioral scientists know, for instance, that many dramatic personality differences *must* be traced to environmental influences. Perhaps the most important reason to doubt that genetic variation accounts for a substantial portion of observed differences in human behavior is simply that we lack an extensive enough hereditary apparatus to do the job—that we have a “gene shortage” (Ehrlich 2000). To what extent could genes control the production of these differences?

It is important to remember that behaviors are the results of charge changes that occur in our network of neurons, the specialized cells that make up our nervous system. Behaviors are ultimately under some degree of control in the brain. Neuron networks are the locus of the memories that are also important to our behavior. That genes can control some general patterns is unquestioned; they are obviously involved in the construction of our brains. They might therefore also build in the potential for experience to affect a large part of the details involved in the neural circuitry. But they cannot be controlling our individual behavioral choices.

Human beings have only three times as many genes as have fruit flies (many of those genes appear to be duplicates of those in the flies, and the biochemistry of fly nerve cells seems quite close to ours) (Zigmond et al. 1999: figs. 9.8, 9.9). But in addition to having sex and eating (what flies mostly do) we get married, establish charities, build hydrogen bombs, commit genocide, compose sonatas, and publish books on evolution. It is a little hard to credit all this to the determining action of those few additional genes (Ehrlich 2000: 124–26). Those genes are, however, likely to have contributed to the increased brain size and complexity that support the vast cultural superstructure created by the interaction of our neurons and their environments. They may also contribute to the wonderful flexibility and plasticity of human behav-

ior—the very attributes that make our behavior less rather than more genetically determined. But to understand the development of and variation in specific human behaviors such as creating charities and cheesecakes, we must invoke culture, its evolution, and its potential interaction with biology.

It might be argued that since a relative handful of genes can control our basic body plan—one’s height depends on millions of the body’s cells’ being stacked precisely—a handful could also determine our behavioral phenome. Genes initiate a process of development that might be analogized with the way a mountain stream entering a floodplain can initiate the development of a complex delta. Why, then, couldn’t just a few genes have evolved to program millions of our behaviors? In theory they might have, but in that case human behavior would be very stereotyped. Consider the problem of evolving human behavioral flexibility under such circumstances of genetic determination. Changing just one behavioral pattern—say, making women more desirous of mating with affluent men—would be somewhat analogous to changing the course of one distributary (branch in the delta) without altering the braided pattern of the rest of the delta. It would be difficult to do by just changing the flow of the mountain stream (equivalent to changing the genes) but easily accomplished by throwing big rocks in the distributary (changing the environment).

This partial analogy seems particularly apt in that it is apparently difficult for evolution to accomplish just one thing at a time. There are two principal reasons for this. The first is the complexity of interactions among alleles and phenotypic traits, especially pleiotropy and epistasis. Because there are relatively so few of them, most genes must be involved in more than one process (pleiotropy). Then if a mutation leads to better functioning of one process, it may not be selected for because the change might degrade the functioning of another process. And changes in one gene can modify the influence of another in very complex ways (epistasis). Second, because they are physically coupled to other genes on the same chromosome, the fates of genes are not independent. Selection that increases the frequency of one allele in a population will often, because of linkage, necessarily increase the frequency of another. Selection favoring a gene that made one prefer tall mates might also result in the increase of a nearby gene that produced greater susceptibility to a childhood cancer.

The Mysteries of Environmental Control

Behavioral scientists are still, unhappily, generally unable to determine the key environmental factors that influence the behavioral phenome. For instance, in the case of the Dionne quintuplets, quite subtle environmental differences—perhaps initiated by different positions in the womb or chance interactions among young quints, their parents, and their observers (Blatz 1938)—clearly led to substantially different behavioral and health outcomes in five children with identical ge-

nomes. As their story shows, we really know very little about what environmental factors can modify behavior. For example, some virtually undetectable differences in environments may be greatly amplified as developing individuals change their own environments and those of their siblings. Equally, subtle and undetected environmental factors may put individuals with the same genetic endowments on similar life courses even if they are reared apart, perhaps explaining anecdotes about the similarities of some reunited identical twins.

We also know too little about the routes through which genes may influence behavior, where again changes may be behaviorally amplified. Suppose that a study shows that identical twins, separated at birth, nonetheless show a high correlation of personality type—both members of twin pairs tend to be either introverted or extraverted. This is interpreted as a high heritability of introversion and extraversion. What really is heavily influenced by genetics, however, could be height, and tall people in that society (as in many societies) may be better treated by their peers and thus more likely to become extraverted (Buss 1994: 39–40). Genes in this case will clearly be involved in personality type but by such an indirect route as to make talk of “genes for introversion or extraversion” essentially meaningless.

And, of course, scientists *do* know that what appears to be “genetic” is often simply a function of the environment. An example suggested by the philosopher Elliott Sober (personal communication) illustrates this. In England before the 18th century, evolutionary psychologists (had there been any) would have assumed that males had a genetic proclivity for knitting. The knitting gene would have been assumed to reside on the Y chromosome. But by the 19th century, evolutionary psychologists would have claimed that women had that genetic proclivity, with the knitting gene on the X chromosome. With historical perspective, we can see that the change was purely culture-driven, not due to a genetic change. As it did with knitting, the environment, especially the cultural environment, seems to do a good job of fine-tuning our behavior. A major challenge for science today is to elucidate how that fine-tuning occurs.

Would Selection Generally Favor Genetic Control of Behavior?

Would we be better off if we had more than enough genes to play a controlling role in every one of our choices and actions and those genes could operate independently? Probably not. One could imagine a Hobbesian battle in which genes would compete with each other to improve the performance of the reproducing individuals that possessed them—genes for caution being favored in one environment one day and genes for impulsiveness in another environment the next (“Look before you leap,” “He who hesitates is lost”). It is difficult to imagine how *any* organism could make the grade evolutionarily if its be-

havior were completely genetically determined and interactions between its genes and its environments did not exist. Even single-celled organisms respond to changes in their surroundings. Without substantial environmental inputs, evolution would not occur and life could not exist.

Biological evolution has avoided that problem by allowing our behavior to be deeply influenced by the environments in which genes operate. In normal human environments, genes are heavily involved in creating a basic brain with an enormous capacity for learning—taking in information from the environment and incorporating that information into the brain’s structure. It is learning that proceeds after birth as an infant’s brain uses inputs such as patterns of light from the eyes to wire up the brain so that it can see, patterns of sound that wire up the brain so that it can speak one or more languages, and so on. As the brain scientist John Allman put it, “the brain is unique among the organs of the body in requiring a great deal of feedback from experience to develop its full capacities” (1999: 177). And the situation is not so different for height. There aren’t enough genes to control a child’s growth rate from day to day—adding cells rapidly in favorable (e.g., food-rich) situations and slowly or not at all under starvation. And there aren’t enough genes to govern the growth of each column of cells, some to regulate those in each column on the right side of the spine, some for each in the left. Instead, all growth patterns depend on environmental feedback.

Does Cortical Mapping Change This View?

But hasn’t all the above been shown to be incorrect by recent mapping studies (Thompson et al. 2001) of cortical structures in the brains of monozygous or identical (MZ) and dizygous or fraternal (DZ) twins? This has been the interpretation of those studies by the popular press (Mottuk 2001). Thompson and his colleagues computed differences in the quantity of gray matter of MZ and DZ twins and unrelated individuals for various regions of the cortex. Not only did they claim to have demonstrated that genetic factors significantly influence a number of structural regions of the brain but they argued that their gene maps revealed how genes *determine* individual brain differences. These are indeed strong claims.

Thompson et al.’s data analysis of the brain maps suffers from many of the defects mentioned above as permeating the behavioral genetic literature. Environmental contributions are ignored: “Because non-genetic familial effects contribute to the resemblance between relatives, such effects were accommodated, if not entirely eliminated, by assuming the same common environmental variance for MZ and DZ pairs” (Thompson et al. 2001: 1257). Then, a squared correlation greater than 0.8 having been claimed for volumes of cortical structures between MZ twins, the squared correlation for DZ twins in the same areas varied from 0.6 to 0.89. There is an obvious difficulty here in that such large DZ correlations suggest important environmental contri-

butions.⁴ The relationship between the MZ and DZ correlations certainly does not suggest “strong genetic control of brain structure” (p. 1254) or even “tight coupling of brain structure and genetics” (p. 1256) as claimed.

Strangely, in the analysis by Thompson et al. of the relationship between cortical gray matter and Spearman’s g , a quantity often used as a measure of cognitive performance (from subtests of the Wechsler Adult Intelligence Scale), unrelated individuals were omitted and only the 40 twins received the cognitive tests. A highly significant relationship between frontal matter volume and g is reported. It is not clear how the correlations between the twins were controlled here, especially in light of the very high DZ correlations in the brain images and the well-established effects of gender on some contributors to g , especially the greater variability of males’ scores than females’ (e.g., Jensen 1998:537). In sum, Thompson et al. cannot be regarded as having demonstrated a gene-brain relationship, nor do their “genetic brain maps” contribute to our understanding of how genes influence cognition.

Conclusions

What the recent evidence from the Human Genome Project tells us is that the interaction between genes, between the separate components of genes, and between controlling elements of these separate components must be much more complex than we ever realized. Simple additive models of gene action or of the relationship between genes and environments must be revised. They have formed the basis for our interpretation of phenotype-genotype relationships for 84 years, ever since R. A. Fisher’s famous paper (1918) that for the first time related Mendelian genes to measurable phenotypes. New models and paradigms are needed to go from the genome to the phenome in any quantitative way. The simplistic approach of behavioral genetics cannot do the job. We must dig deeper into the environmental and especially cultural factors that contribute to the phenome. The ascendancy of molecular biology has, unintentionally, militated against progress in studies of cultural evolution.

Theories of culture and its evolution in the 20th century, from Boas’s insistence on the particularity of cul-

tural identities to the debates between material and cultural determinism described by Sahlins (e.g., 1976), were proudly nonquantitative. Recent discussions on the ideational or symbolic nature of the subjects of cultural evolution (e.g., Durham 1991), while critical of attempts to construct dynamical models of cultural evolution based on individual-to-individual cultural transmission, nevertheless acknowledge the centrality of cultural evolution to human behavioral analysis. Thus, although the quantitative paradigms used in behavioral genetics do not inform evolutionary analysis, this does not mean that we cannot or should not take an evolutionary approach to the understanding and modification of human behavior. Genetically evolved features such as the dominance of our visual sense should always be kept in mind, but an evolutionary approach to changing behavior in our species must primarily focus on *cultural* evolution. In the last 40,000 years or so, the scale of that cultural evolution has produced a volume of information that dwarfs what is coded into our genes. Just consider what is now stored in human memories, libraries, photographs, films, video tapes, the Worldwide Web, blueprints, and computer data banks—in addition to what is inherent in other artifacts and human-made structures. Although there have been preliminary investigations by Cavalli-Sforza and Feldman (1981) and Boyd and Richerson (1985), scientists have barely begun to investigate the basic processes by which that body of information changes (or remains constant for long periods)—a task that social scientists have been taking up piecemeal and largely qualitatively for a very long time (e.g., Bischof 1978; Cronk 1999; Durham 1991; Ehrlich 2002; Jacobs and Campbell 1961; Johnson and Earle 2000; Kotler and Zaltman 1971; Murdock 1956; Pirages and Ehrlich 1974; Rogers 1995; Stark 1996, 1999). Developing a unified quantitative theory of cultural change is one of the great challenges for evolutionary and social science in the 21st century.

Identifying the basic mechanisms by which our culture evolves will be difficult; the most recent attempts using a “meme” approach (Blackmore 1999, Dawkins 1989 [1976]) appear to be a dead end. Learning how to influence that evolution is likely to be more difficult still and fraught with pitfalls. No sensible geneticist envisions a eugenic future in which people are selected to show certain behavioral traits, and most thinking people are aware of the ethical (if not technical and social) problems of trying to change our behavior by altering our genetic endowments. Society has long been mucking around in cultural evolution, despite warnings of the potential abuses of doing so (e.g., Huxley 1932). Nazi eugenic policies and Soviet, Cambodian, Chinese, and other social engineering experiments stand as monuments to the ethical dangers that must be guarded against when trying systematically to alter either genetic or cultural evolution.

Nevertheless, we are today all involved in carrying out or (with our taxes) supporting experiments designed to change behavior. This is attested to by the advertising business, Head Start programs, and the existence of in-

4. Referring to n. 2, if individuals 1 and 2 are MZ twins and if they are raised in identical environments (so that $E_1 = E_2$), then their correlation should be 1. If, on the other hand, they are DZ twins, all the genetic contributions to the quantity of gray matter are additive, and E_1 and E_2 are not correlated, their correlation should be 0.5 (which would make the squared correlation 0.25). Dominance effects will increase this, but in most cases the increase will not be sufficient to bring the DZ correlation into the ballpark of the MZ correlation. For these DZ twins, from the simple model of nn. 1 and 2 the only other possible explanation for the high DZ twin correlation would be a correlation between environments E_1 and E_2 . The study included five pairs each of female and male MZ and DZ twins. Despite acknowledging that gender affects volumes of brain structures, images for the sexes were pooled within each zygosity. It is very difficult to invoke statistical contributions from genes to explain the apparently close MZ and DZ values reported.

stitutions such as Sing Sing Prison and Stanford University. The data used by evolutionary psychologists to infer the biological antecedents of human behavior, while not telling us anything about genetic evolution, may actually be helpful in improving our grasp of cultural evolution. What seems clear today, however, is that evolutionary psychology and behavioral genetics are promoting a vast overemphasis on the part played by genetic factors (and a serious underestimation of the role of cultural evolution) in shaping our behavioral phenomes.

Comments

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Ehrlich and Feldman advance serious and valid criticisms of the methods used by evolutionary psychologists and behavioral geneticists and identify factual errors frequently made by them. While in conceptual agreement with them, I propose here to look at some other aspects of the problem.

Cosmides and Tooby's new version of evolutionary psychology (1987, 1995; Tooby and Cosmides 2000) combines teleological, adaptationist, and rigid formalist interpretations of biological evolution with the view of the mind as a sort of computer program or information processor. The adaptationist program considers every evolutionary novelty as a feature that favours survival and/or reproduction (Gould 2002, Kirmayer and Young 1999). Adaptationists regard each aspect of the organism's morphology, physiology, and behaviour as a specific adaptation of the entire organism. For them the problem of evolutionary science is finding out what an adaptation is for, when in fact the first question should be whether it exists (Fodor 2000; Mahner and Bunge 1997:423).

It is extremely difficult to trace traits back in time, and any hypothesis regarding the history of a trait must be based on probability (Northcutt 1999). Few traits have been examined in sufficient detail in enough species in different radiations to allow a meaningful evaluation of them. The problem of identifying traits is compounded by the frequency with which some psychologists and psychiatrists arbitrarily qualify the condition of the state or trait for various phenotype components (Paris 1998).

Questionable concepts such as "brain design in response to environmental demands" (Kirmayer and Young 1999) and concepts difficult to verify such as "adaptive evolution of traits" (Buss 1999, Paris 1998) lack heuristic value, and there is no evidence whatsoever for an instructive component in the appearance of evolutionary novelties (Dover 2000, Gould 2002, Lewontin 2000).

Adaptationists fail to recognize other factors besides natural selection as causally associated with evolution. While it is not a criterion of truth, there is a measure of

consensus that phenomena such as exaptation (Gould 2002), accident as an agent of direction of change itself rather than only a source of variation (Kimura 1983), and molecular drive (Dover 2000) are all causal mechanisms of evolutionary novelties (Goodwin 2002).

Assigning adaptive significance to an organ or behaviour pattern presumes that a problem exists to which it is a solution (Dubrovsky 2002). However, organisms not only solve problems in the environment but create them. As Waddington (1976:18) has put it, "A surprisingly large amount of the environment which affects natural selection outcomes on animals is the more or less direct result of the animal's own behavior." Considering these facts, Lewontin (2000) has suggested that a more faithful description of the organism-environment interaction is "construction" rather than "adaptation." In human evolution, the usual relationship between organism and environment has become virtually reversed in adaptation. Cultural invention has replaced genetic change as the effective source of variation. Consciousness allows people to analyze and make deliberate alterations as situations require, with the result that adaptation of environment to organism has become the dominant mode (Dubrovsky 2002).

Oskar Kempthorne (1978) has criticized the exclusive use of observational data in the debate about inherited and environmental factors contributing to intelligence. Observational data are used by behavioural geneticists (e.g., Plomin et al. 1997) notwithstanding the recognition since the beginning of modern science that only experimentation can test the validity of rival causal hypotheses (Bunge 1967). Kempthorne and later Jacquard (1983) have criticized the use of analysis of variance of one feature in a population to check for causality, arguing that variance only measures dispersion of data around the value of the mean; it is a measure of diversity, sometimes inappropriately referred to as "variability." What is important in considerations of causality, however, is the magnitude of an effect which can be attributed to variation or change in one or more independent variables. Variance cannot point to any causal factor. The parallel with statistical correlation is clear. The latter measures the degree of association of two variables (e.g., size and population of a country) neither of which causes the other but variation in one of which can induce changes in the other. It has never been demonstrated that IQs (Jacquard 1983) are determined by the genome, since causal relations are valid only for events and not for attributes.

Moreover, linear additive models in the absence of a theory of interaction are invalid (Lewontin 2000). That both genes and environment produce a given outcome is a truism, but we are seriously mistaken when we presume that we can best express this principle by assigning percentages and stating, for example, that behaviour A is 40% genetic and 60% environmental. Such reductionist expressions go beyond simple mistakes to enter the domain of the meaningless. Genetics and environment do interact to build a totality, but we need to un-

derstand why the resulting wholes are irreducible to separate components.

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Because Ehrlich and Feldman fail to provide them, I sketch evolutionary psychology's basics here.

It has long been recognized (e.g., Galen, Paley) that organisms consist of functional mechanisms—hearts, lungs, livers, bones, intestines, prostates, uteruses, etc.—but before 1859 their origin was unknown. Darwin and Wallace proposed that these mechanisms—termed adaptations—evolved by natural selection and, thus, *necessarily* were designed to promote reproduction.

Psychologists have demonstrated that cognitive processes, like the body's other mechanisms, have functional structure. Evolutionary psychologists propose that this structure evolved by natural selection to serve reproduction. Given that the brain mechanisms underpinning vision, hearing, motor control, pain, memory, etc., have obvious reproductive utility, this proposition is compelling. Further, these examples suggest that the brain is made up of many functionally specialized parts.

An adaptation is rarely discovered or described by identifying the specific genes that directed its construction or by documenting heritable variation and differential reproduction in ancestral populations. Rather, adaptations are recognized by the close functional fit between an adaptive problem and some aspect of phenotypic structure; it is their *evidence of design*, not genes, that assures us that hearts, lungs, and livers are adaptations.

Although Ehrlich and Feldman believe that the environment of evolutionary adaptedness refers to a fixed time or place, it actually refers to the recurring aspects of the environment that were necessary for the evolution, development, functioning of a particular adaptation (Tooby and Cosmides 1990a). The environment of evolutionary adaptedness of the lung, for example, includes an oxygen atmosphere. Recurring aspects of ancestral environments that had an impact on reproduction include interactions with the opposite sex, children, parents, kin, nonkin, plants, animals, predators, and prey and the need to avoid toxins, pathogens, and injuries. Much evolutionary psychological research has been based on the certainty that in the environment of evolutionary adaptedness women got pregnant and men did not.

To say "There is reason to believe that fear of snakes in other primates is largely learned" implies that learning and psychological adaptations are opposing hypotheses. But, as Pinker (1997:33) and virtually every other evolutionary psychologist repeatedly emphasize:

Yes, every part of human intelligence involves culture and learning. But learning is not a surrounding gas or force field, and it does not happen by magic.

It is made possible by innate machinery designed to do the learning. The claim that there are several innate modules is a claim that there are several innate learning machines, each of which learns according to a particular logic.

Further, the New Guineans' opinion that "the fear in Europeans [is] a result of their stupidity in being unable to distinguish which snakes might be dangerous" supports rather than refutes Pinker (1997:388):

The world is a dangerous place, but our ancestors could not have spent their lives cowering in caves; there was food to gather and mates to win. They had to calibrate their fears of typical dangers against actual dangers in the local environment (after all, not *all* spiders are poisonous) and against their own ability to neutralize the danger: their know-how [etc.]. . . . Between the ages of three and five, children become fearful of all the standard phobic objects—spiders, the dark, deep water, and so on—and then master them one by one. Most adult phobias are childhood fears that never went away. That is why it is city-dwellers who most fear snakes.

Finally, Mineka and colleagues' research, which Ehrlich and Feldman apparently believe undermines evolutionary psychological hypotheses of specialized fear learning, actually strongly supports them. Cook and Mineka showed that lab-raised monkeys readily learned to fear toy snakes but not toy rabbits or flowers, suggesting that there is an innate predisposition to learn fears of evolutionarily salient dangers, such as snakes. Öhman and Mineka (2001) synthesize 30 years of research on fear in humans and other primates in an article subtitled "Toward an Evolved Module of Fear and Fear Learning."

Against hypothesized sex differences in mating psychology, Ehrlich and Feldman claim that "it would be no small developmental trick genetically to program detailed, different, and *independent* reproductive strategies into modules in male and female brains." Well, natural selection somehow "programmed" uteruses in females but not males. Evolutionary psychology argues that men's and women's brains, like the rest of their bodies, are probably identical in most ways but profoundly different in some.

Whereas behavioral genetics focuses on individual differences, evolutionary psychology focuses almost exclusively on human universals (age and sex excepted). Adaptations are grounded in the vast majority of genes that are identical (or nearly identical) in all humans. Important individual differences arise not from minor genetic differences but "from exposing the same human nature to different environmental inputs" (Tooby and Cosmides 1990b:23).

Accused of spinning "just-so stories," evolutionary psychologists have, in fact, tested their hypotheses in hundreds of studies with many thousands of subjects in scores of different cultures and have published their results in the world's top science journals. Empirical re-

search, not armchair criticism, will determine whether these hypotheses stand or fail.

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Ehrlich and Feldman argue that evolutionary psychology and behavioral genetics are based on faulty thinking about genetics, evolutionary theory, and culture. Our concern, as behavioral ecologists interested in the evolution of primate behavior and cognition, is that they have set up a straw man carrying a basket of red herrings. It is easy to criticize a field. It is more challenging to rise above what is bad and see how to address the important problems posed by human psychological evolution.

First, Ehrlich and Feldman claim that certain questions raised by evolutionary psychology and behavioral genetics are impossible to answer, a critique that is surely too pessimistic. Here are two examples: "It is impossible to distinguish human behavioral phenomes that are shared because of genetic similarities from those caused by shared environments" and "Overall heritability should be restricted in its employment to plant and animal breeding where it can be better measured and the results put to some practical use." Concerning the first, they surely cannot mean "impossible." The evidence from comparing identical twins reared apart in different environments with identical twins reared together is surely a point in favor of teasing apart genetic and environmental influences. Why is this any different from a study of corn? Granted, our genome is more complicated, as is our environment, but this amounts to difficulty not impossibility. Claims of heritability are similarly confused. Why restrict analyses to plant and animal breeding? We see no reason that Ehrlich and Feldman need to commit themselves to such an extreme position.

Second, in discussing how evolutionary psychologists make inferences about the role of genetics, Ehrlich and Feldman imagine a nativist for whom the environment is irrelevant. But those who adhere to a nativist position are no different from those biologists who are interested in the relationship between evolution and developmental constraints, a field that we assume Ehrlich and Feldman would support. For example, in a study of language acquisition the idea is that there are learnability constraints set by the genome. Given these constraints, the views espoused are not nearly as naïve as Ehrlich and Feldman suggest. As the Chomskyan revolution demonstrated, the interesting questions are what pieces of the language faculty are universally expressed and thus a reflection of a common biological mechanism and how this universal mechanism constrains the range of variation—the phenome for linguistic expression. Focusing attention in this way does not detract from cultural variation; rather, it asks how variable the system can be.

More specifically, the Chomskyan view suggests that our human nature sets up constraints on the range of cultural variation and therefore certain languages, though imaginable, would not be stable and are therefore considered impossible. The arguments for beauty are no different. Given, for example, the observation that in dozens of species female mate choice is mediated by a mechanism that attends to symmetrical traits and that such a mechanism evolved because of selection for males with good genes, why isn't the most likely explanation of human parallels that we are also equipped with a mechanism for symmetry detection? The possibility that some cultures may fail to apply this mechanism does not invalidate it. This reasoning error shows up in Ehrlich and Feldman's treatment of snake fears. What Mineka's work shows is not an innate fear response to snakes but a disposition to respond with fear to snakes once having seen conspecifics responding with fear.

Third, Ehrlich and Feldman fail to address how they would explain the large number of cases that cannot be understood in terms of environmental input. For example, when children produce grammatical constructions that no adult in their culture has ever produced, given the lack of relevant environmental input the most plausible explanation is that they are equipped with innate grammatical competences that the linguistic environment fine-tunes. Other cases include the universal emotions and the developmental timing and universal acquisition of a theory of mind across cultures.

Fourth, rather than suggest how evolutionary and psychological studies might work hand in hand, Ehrlich and Feldman set up straw men and then argue that we should be "primarily" looking at how culture shapes human nature. "It is difficult," they say, "to imagine how any organism could make the grade evolutionarily if its behavior were completely genetically determined and interactions between its genes and its environment did not exist." No one argues for complete genetic determinism. No one argues against interactions. And no one denies that cultural evolution is important. But saying that cultural evolution is important is not saying much. Rather, we must ask other kinds of questions: What allows humans to have the kinds of cultures they have? Why is it that we have such vast and complicated cultures and other animals don't? Why is it that certain cultural differences are relatively trivial while others lead countries to go to war? If culture is unconstrained by biology, then presumably any kind of cultural drift is possible. We don't believe this is true, and we would be surprised if Ehrlich and Feldman thought differently.

In sum, Ehrlich and Feldman have voiced unoriginal criticisms of evolutionary psychology and behavioral genetics. Worse, they have failed to make suggestions for how these disciplines might improve. It is time to move beyond fears of excessive nativism. The difficult questions about gene-culture interaction cannot be ignored.

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Critics all too often hold their own assumptions uncritically, turning seemingly good criticisms into refutations of those assumptions. Researchers in evolutionary psychology or in behavioral genetics should clarify how Ehrlich and Feldman's criticisms make assumptions that misinterpret these paradigms (see Holcomb 2001). Consider their abstract: Basic logic teaches us to specify a domain before "all" or "some" can be said sensibly. Their term "much human behavior" is meaningless unless one first states whether the phenomena are facts about phylogeny, the focus of evolutionary psychology, or facts about ontogeny. The role of culture in human ontogeny is not evolutionary psychology's subject matter, except as Ehrlich and Feldman and others use it to restrict evolutionary psychology's limited domain unduly by confusing levels of analysis. Not to give a cultural explanation does not imply denying one; it is scientific specialization. The authors use a false dichotomy of "genetic determinism" versus their view that general capacities (such as sight) evolved but behavioral specifics did not. No such line can be drawn because general capacities did not promote fitness unless they led to specific behavioral patterns; no generalist species has "evolved general traits" but not corresponding "evolved specific traits," which are subject to variation ("differences"). Evolutionary psychology now uses concepts of the evolutionary environment of adaptation that are more subtle than the one they attack. Basic behavioral genetics teaches us not to confuse the colloquial "inherited" with the technical term "heritable." So, each claim in Ehrlich and Feldman's abstract is false. I plead that critics try to show that their criticisms are good rather than bad ones by anticipating likely objections to their assumptions and rebutting them.

A good criticism should use a charitable interpretation. Behavioral geneticists know that by definition behaviors with zero heritability may be under any degree of genetic control (a group with all brown eyes or else all Catholics). So heritability is irrelevant to genetic determinism as a view of the roles of genes and environment in individual development and, consequently, as a view of differences among individuals or groups arising as a statistical aggregate of degree of genetic control. Many specific methods in behavioral genetics employ a distinction between a *description* of variation, in which typically $.4 \pm .2$ percent of the variation is proportioned among variation among people of different kinship (genetic) relations and remaining variation, and a *causal explanation* of variation, in which either half of the variation so described exists because the people share or do not share certain genes, psychological mechanisms, social interactions, or cultural contexts.

A good criticism should be informed. Ehrlich and Feldman do not engage the lengthy debates in evolu-

tionary psychology over how to make the concept of the evolutionary environment of adaptation precise (a relevant one is roughly the history of selective forces relevant to the evolution of a trait, not confined to the Pleistocene or to a common environment for all humans) between evolutionary psychologists and Darwinian anthropologists over what simplifying assumptions to make about such an environment or between Darwinian anthropologists and cultural anthropologists over coevolutionary theories of gene-culture evolution. By ignoring the issues, they don't advance the issues. When multiple paradigms can explain the same data, to call an evolutionary psychological story or the authors' purely cultural story a "just-so story" is divisive name calling.

A good criticism should advance disciplinary cooperation. What should the interface between evolutionary psychology and behavioral genetics be, given their separation because evolutionary psychology focuses on human universals and behavioral genetics focuses on individual differences? Kinship is key: the family-based methods of behavioral genetics utilize kinship to describe genetic and environmental variation that evolutionary psychology then explains using inclusive-fitness theory (Mealey 2001). Proximate mechanisms for individual phenotypic differences include genetic polymorphism (phenotypic differences caused by genotypic differences), phenotypic plasticity (conditional strategies triggered by environmental cues), and ontogenetic shifts (change during the life cycle). Natural selection favors various combinations of these to yield within-species differences, as biologists find in animals. Evolutionary theory shows that when genes are maintained in a balanced polymorphism by frequency-dependent forces within a population or by frequency-independent forces in different directions over time and space, heritable variation exists for the trait. Thus biological theory, its results in animals, animal-human comparisons, and results of behavioral genetics research designs jointly imply that genetic differences involving natural selection are often causes of human heritabilities.

A good criticism should recognize how the initially large number of possible theoretical hypotheses is narrowed by adding statements of the circumstances to explanations of concrete cases. Consider Ehrlich and Feldman's misattribution to evolutionary psychology of the just-so story of "an inborn phobia about snakes in all humans." Evolutionary psychologists distinguish obligatory from facultative traits; the latter are flexible within limits and are expressed contingent on immediate physical or social environment or previous individual development or both. As facultative traits, learning mechanisms prepare us to learn some things more easily than others; Americans aren't born with a fear of snakes, but under certain circumstances we learn it more easily than fears of dangerous evolutionarily novel things such as electric outlets. Other circumstances obtain in New Guinea; an initial hypothesis to be tested is that those people's minds, which transmit their culture to offspring, have adapted to intense re-

curing site-specific selective pressures imposed by living closely with snakes.

The preceding points function negatively to imply the falsity of the assumptions behind Ehrlich and Feldman's criticisms and positively to set standards for criticism that will make debate more fruitful.

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Ehrlich and Feldman argue that evolutionary psychologists and behavioral geneticists, adopting what I will call the "genetic position," greatly overstate the importance of genetic contributions to human behavior. Their article has two main themes: that the genetic position is often based on data about the heritability of behavioral traits rather than about their development and that the development of behavior is more strongly influenced by the environment than the genetic position recognizes. Heritability measures the relative importance of genetic and environmental variability in determining variability in the phenome and has nothing to do with development (Lewontin 1974). As Ehrlich and Feldman point out, heritability is a very local measure, and its value for any trait can change substantially with both the genetic makeup of a population and its environment. Much of the confusion in debates about genetic contributions to human behavior could be avoided if this relatively simple point were more widely understood.

Their second theme, that genetic influences play a less important role in the development of human behavior than the genetic position supposes, is far less helpful. In choosing to debate this question, Ehrlich and Feldman implicitly (and sometimes explicitly) support two ideas that are based on fundamental misconceptions about development. The first is that it is possible to quantify genetic and environmental contributions to development; the second is that genes can *in principle* control or program behavior although they do not *in fact* control as many aspects of human behavior as the genetic position maintains. Developmentalists since Carmichael (1925) and Anastasi (1958) have pointed out that debating the relative amounts of genetic and environmental influence on behavior is futile. One might as well ask, to use a well-worn pedagogical device, how much the length and the breadth of a rectangle contribute to its area. However, the misconception involved in the idea that genes *might* completely control a behavior is far more insidious.

Developmentalists have argued for a long time that partitioning elements of the phenome into those specified by the genes and those specified by the environment simply will not work (see Gottlieb 1996, Gray 1992, Griffiths and Gray 1994, Johnston 1987, 1988, Oyama 2000a, b). The main conclusion of this argument is that both genes and environment make es-

sential developmental contributions to all aspects of our behavior: It makes no more sense to deny genetic contributions to (for example) patterns of mate choice in humans than it does to deny a cultural contribution to those patterns. This is not the same as saying that *differences* in patterns of mate choice among societies depend on genetic (or cultural) differences: the question under discussion is what creates behavior, not differences in behavior (cf. Lewontin 1974). The ways in which a particular man or woman makes decisions about sexual attractiveness, marital fidelity, and other matters that define patterns of mate choice depend on interactions between his or her neural circuitry and the various opportunities for mating available in the environment. That neural circuitry in turn depends on a developmental history in which genes and environment have played essential roles that are very hard (though not impossible) to analyze.

The ways in which behavioral and social scientists generally think about the roles of genes in development are poorly adapted to a real understanding of the issue (Johnston 1987). A point that often goes unappreciated is that genes are molecules, and not very active molecules at that. Genes cannot, in principle, specify a behavior—all they do is provide a template for the synthesis of a protein or other biologically active molecule through the intermediate steps of transcription (of a messenger RNA molecule) and translation (of a protein). In order for protein synthesis to take place, some other molecule must activate the gene—a process called induction—so that transcription (also known as gene expression) can occur. Very often, gene expression depends on behavior (Gottlieb 1998, Johnston and Edwards 2002). For example, gene expression in regions of the brain known to be important for the regulation of maternal behavior in rats depends on sensory stimulation provided by the pups (Fleming et al. 1994). This and other findings imply that far from gene expression's controlling behavior, behavior usually controls gene expression.

When I discuss results like these and the rethinking they imply about genetic contributions to behavior with social scientists, the response is often something like, "I'm not a molecular biologist, I'm an anthropologist (or sociologist, or psychologist), and I don't have the time or training to understand these molecular details." Unfortunately, it is down among the molecules that genes do their work, and if we want to speak about the ubiquitous genetic contributions to behavior then we will have to master enough of the molecular details to understand what they mean for behavioral and social processes. Communicating that understanding to social scientists will be a difficult process (see Lewontin 2000, Morange 2001, Moore 2001, Johnston and Edwards 2002), but it is essential if we are truly to understand what creates the behavioral phenome.

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Ehrlich and Feldman are clearly unhappy with careless and misinformed explanations for human behaviour that allocate too great an explanatory role to genes, and they provide a strong case against the extremes of behavioural genetics and evolutionary psychology. While they group these two fields of research together, closer inspection reveals that human behavioural genetics and evolutionary psychology provide two very different kinds of explanation for human characteristics. In fact, their basic premises appear to render these two viewpoints mutually incompatible. Behavioural geneticists commonly argue that differences between humans reflect underlying genetic variation, while most evolutionary psychologists suggest that human beings exhibit universal behaviour patterns as a result of evolved psychological mechanisms that are reflections of our genetic makeup. Human behavioural genetics and evolutionary psychology cannot both be right, although they could both be wrong.

Ehrlich and Feldman's objection to human behavioural genetics is clear: An overreliance on twin data and oversimple statistical analyses have led to inflated estimates of heritabilities, with disturbing ramifications for social policy. We wholeheartedly endorse this criticism and agree that comparisons of identical and fraternal twins provide a fundamentally misleading basis for explaining the differences between people. However, the real problem here is methodological, lying in the inadequate empirical and theoretical tools that most behavioural geneticists are content to use. In the absence of genuinely reliable analyses, the claim that there are *some* heritable genetic differences among people that influence behaviour remains plausible, although generally unsubstantiated.

Similarly, Ehrlich and Feldman's disapproval of evolutionary psychology appears to rest largely on their unhappiness with its methods. Clearly, weak speculations based on untestable assertions about the characteristics of unknown ancestral environments, combined with oversimplified adaptationist reasoning and hypermodularity, do not make for good science. Of course, not all evolutionary psychologists are adherents of the dominant modular-adaptationist school, and a significant minority are critical of just-so storytelling. Many evolutionary psychologists will feel that Ehrlich and Feldman have dismissed a straw-man version of their field. However, even if all of the work emanating from evolutionary psychology were widely considered worthless because of its poor methodology, it would remain possible that there are human universals and that genes constrain the delineate developmental pathways in meaningful ways. Our intuitions, like Ehrlich and Feldman's, are that psychological states are better

regarded as by-products of our extraordinary adaptability than as adaptations, but intuitions count for nothing.

The real problem with behavioural genetics and evolutionary psychology is not that they potentially misrepresent the role of genes but that they commonly make unsupported and perhaps dangerous claims for genes on the basis of weak science. Yet understanding the biological bases of human behaviour and the roles that genes and evolution play in that explanation is a fundamental and taxing challenge. Are there clearly established ways of addressing these challenges more rigorously? Ehrlich and Feldman are advocates of the methods of cultural evolution and gene-culture coevolution, and we too are supporters of these approaches. However, the number of researchers involved in them remains small. This school is regarded by friendly critics as "theoretically rich but empirically poor" (Smith 2000) and by most other potentially interested parties as mathematical hieroglyphics. In contrast, evolutionary psychology is a much larger and thriving discipline, with the number of advocates growing exponentially. Human behavioural ecology is smaller but empirically and theoretically rich and progressive. The field of me-metics, which Ehrlich and Feldman characterize as a "dead end," is the subject of intense interest and is beginning to produce scholarly works (Aunger 2000, 2002). Perhaps the reality is that researchers are attracted to evolutionary psychology and behavioural genetics because their methods are relatively easy to use in comparison with the mathematical models of gene-culture coevolution.

However, could there be an empirical science of cultural evolution? Cavalli-Sforza and Feldman (1981) have pioneered this theoretical tradition by adapting population genetics models on the assumption that there may be useful parallels between biological and cultural evolution. If these parallels are genuine, then the empirical methods that evolutionary biologists currently employ to detect natural selection in the wild (Endler 1986) or similar ones should be applicable to the study of cultural selection. For example, researchers interested in cultural evolution could investigate whether there is evidence for convergent evolution of cultural traits or evidence for character displacement in cultural activities (these and other methods are discussed further in Laland and Brown 2002). We recognize that a small number of empirical studies have emanated from the cultural evolution school (Cavalli-Sforza et al. 1982, Soltis, Boyd, and Richerson 1995, Guglielmino et al. 1995, Henrich et al. 2001), and there are promising recent developments (also reviewed in Laland and Brown 2002), but Ehrlich and Feldman must accept that its experimental side is not yet well-established. Until critics devise a straightforward empirical program of research based on more rigorous methodologies, behavioural genetics and evolutionary psychology are likely to remain popular among those interested in exploring the factors that shape our behavioural phenome.

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Ehrlich and Feldman disdain attempts to understand human behavior from an evolutionary biological perspective, even while acknowledging that genetic evolution has meaningfully influenced behavior. Fundamentally, it makes no sense to argue that human behavior was not subject to biological evolution. The question is really just how to characterize the relationship between genes and behavior. Their suggestion that there aren't enough genes to code for all possible behavioral responses is irrelevant; the argument has never been that there is total genetic control of every aspect of behavior. This kind of slippery mischaracterization of explanations that include genetic influences is quite common (Dawkins 1982) and suggests that humans generally have trouble understanding the concept of multiple causality. The focus by evolutionary psychologists on genetic influences does not constitute evidence that they believe culture is irrelevant any more than the converse is true for those emphasizing cultural influences.

Ehrlich and Feldman state that "geneticists know that a large portion of the behavioral phenome must be programmed into the brain by factors in the environment," but geneticists also know that if some environmental factor has a specific behavioral effect it is only by virtue of its interaction with biology. If rape has a specific psychological effect that is qualitatively different from that of other equally brutal forms of assault, it is because our brains are biologically biased towards this (unless we want to argue that the psychological trauma of rape is also just a cultural response).

Ehrlich and Feldman repeat the criticism that evolutionary psychology consists solely of "just-so" stories even though such stories are just as rampant among social scientists espousing environmental explanations. The reason they object so strongly specifically to genetic evolutionary hypotheses must be that they consider social policy driven by social science "just-so" stories inherently less dangerous. This is a common but fundamental mistake. In fact, the belief that there is no biological basis of behavior that defines "human nature" is a very dangerous position. As Robin Fox (1973: 13) pointed out,

If there is no human nature, any social system is as good as any other, since there is no base line of human needs by which to judge them. If, indeed, everything is learned, then surely men can be taught to live in any kind of society. Man is at the mercy of all the tyrants—be they fascists or liberals—who think they know what is best for him. And how can he plead that they are being inhuman if he doesn't know what being human is in the first place?

Ehrlich and Feldman themselves allude to the horrible atrocities committed in the name of the infinite malleability of human behavior. Removing hypotheses about the genetic influences on behavior from serious consideration simply will not inoculate policy makers against doing harm to us all.

That said, there certainly are cases in which the degree of genetic specificity has likely been overstated, one example being in discussions of "grammar genes" (Pinker 1995), for which simpler models with less specificity have been proposed (e.g., Bates and Goodman 1999, Deacon 1997, Kirby 2000, Schoenemann 1999, Schoenemann and Wang 1996). It is also true that Thompson et al.'s (2001) data suggest much less genetic "control" of brain structure than a few of their comments imply. Furthermore, the brain/g correlation had previously been found to be very small within families (which suggests between-family confounds [Schoenemann et al. 2000]).

However, any single influence (whether it is genetic or environmental) can have wide-ranging, complicated effects (e.g., think of socioeconomic status). A claim that such effects are the result of either environmental or genetic influences is not a claim that the underlying causal forces must be highly specific. Ehrlich and Feldman confuse "These behaviors have a genetic influence" with "Each of these behaviors is individually specified by different genes."

Ehrlich and Feldman acknowledge that "we really know very little about what environmental factors can modify behavior." While they disparage behavioral genetics, in fact it provides the best method for demonstrating that environment likely plays an important role. Just showing that culture *could* have some effect does not prove that it actually does. Neither does simply showing that some environmental influence is correlated with some behavioral trait. A great deal of effort has in fact been expended by behavioral geneticists on testing environmental influences (Plomin et al. 1997).

Ehrlich and Feldman correctly point out that evolutionary genetic explanations have no obvious policy implications. If Thornhill and Palmer (2000) are right that rape is an evolved strategy, for example, this says nothing about whether rape should be accepted behavior. It is crucial that evolutionary psychologists take pains to discredit naturalistic fallacies deriving from their work, and by and large they have. However, it certainly must make a difference in dealing with social problems whether or not there are evolved biases in behavior. Rather than dismissing certain types of casual explanations out of hand, accepting that a variety of causal influences (genetic, environmental, and their complex interactions) are possible is the first step in thinking clearly about how to develop policies that would improve the lives of all people.

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Ehrlich and Feldman are clearly right to be disturbed not only by worries about the inherent reliability of reductionist approaches to human behavioral evolution but by the policy implications of the assumption that human beings are genetically programmed to favor certain forms of behavioral response. Any attempt to engineer the conduct of business in human societies that oversimplifies the often unfathomable complexity of interacting *Homo sapiens* is likely to lead to major distortion and misery. Yet the press in particular appears to be entranced by the simplistic reductionisms of “evolutionary” or “Darwinian” psychology—possibly because, whatever else it may be, *H. sapiens* is a storytelling creature, and such accounts certainly make tidy stories.

But wasn't it Charles Darwin's close colleague Thomas Henry Huxley who said that the social philosopher Herbert Spencer's idea of tragedy was “a beautiful theory [story] slain by an ugly fact?” And wasn't Darwin himself appalled during his lifetime by the extravagant claims made in his name by Spencer and others? The travesties since committed in the name of Darwin—who, perhaps more than anyone else in his time, knew what a mysterious and complex thing Nature is—are legion. And the claims of the evolutionary psychologists arise essentially from a fundamentalist interpretation of Darwinism as transmitted through the Evolutionary Synthesis, which became the dominant paradigm of evolutionary thought in the latter half of the 20th century. According to the synthesis, virtually all evolutionary phenomena can be boiled down to the gradual generation-by-generation operation of natural selection on population gene pools. This encouraged a transformational view of evolution that focused on a sort of slow fine-tuning of individual characteristics over vast periods of time and totally ignored the role of taxa and other elements in the evolutionary process.

It was not until the 1970s that the realization began to dawn that evolution is a multilevel process in which numerous influences operate, often simultaneously. It is now widely recognized by biologists in general that, while the synthesis played an essential role in clearing away a great deal of mythology and contradiction from views of the evolutionary process, it represents only a partial framework for understanding evolution. Nonetheless, it lingers on in its most simplistic, “hardened” form in many quarters, including anthropology and the allied behavioral sciences—as witness the aggressive promotion over the past couple of decades of evolutionary psychology as a means of understanding, or at least of explaining, human behaviors. The equation of structure with adaptation has, it appears, a seductive appeal to members of a species that seems to fear its own complexity.

But consider the essential Darwinian mechanism, nat-

ural selection, and how it has to work: It is an intra-population instrument of pruning among variable individuals (notice: individuals, not characteristics). In promoting or inhibiting the reproductive success of individuals, natural selection can vote up or down only on whole individuals, not individually on any of the components into which we might find it convenient to decompose them. Thus, however much we may wish to speak of the evolution of this or that characteristic, whether physical or behavioral, if we do so we are not only ignoring the complexities of the evolutionary process but distorting the concept of natural selection itself. Any organism is a staggeringly complex assortment of phenomic characteristics, most of which are polygenically controlled by genes that are pleiotropic. And we ignore the integration of the genotype at our peril.

One must thus applaud Ehrlich and Feldman for taking the evolutionary psychologists, behavioral geneticists, and their like to task, for in a species in which one can find individuals who are describable by any pair of antitheses one can imagine, reductionist zealotry can potentially do a great deal of damage.

Reply

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A recurring theme in the comments is the inseparability of genetic and environmental contributions to the human phenome. Johnston cites the valuable analogy we often use in our own teaching about genes and environments. Neither the length nor the width of a rectangle controls the area but rather an interaction between the two, just as we say that “every aspect of a person's phenome is a product of interaction between genome and environment.” Dubrovsky makes the same point—that phenomes are whole and “irreducible to separate components.” We agree with Johnston and Dubrovsky; in fact, one of our main criticisms of heritability is that the linear statistical model on which it is based cannot subsume the molecular complexities and environmental interactions that they stress.

We do not, of course, as Schoenemann acknowledges, argue that “human behavior has not been subject to the biological evolution.” We state that “genetic evolution both biased our ability to perceive the world and gave us the capacity to develop a vast culture.” We agree with Johnston, however, that in order to understand the meaning of biological evolution and “the ubiquitous genetic contributions to behavior” social scientists “will have to master enough of the molecular details to understand what they mean for behavioral and social processes.” Some understanding of quantitative genetics would also be helpful. We wonder what, for example, we are to make of Holcomb's statement

Many specific methods in behavioral genetics employ a distinction between a *description* of variation, in which typically $.4 \pm .2$ percent of the variation is proportioned among variation among people of different kinship (genetic relations) and remaining variation, and a *causal explanation* of variation, in which either half of the variation so described exists because the people share or do not share certain genes, psychological mechanisms, social interactions, or cultural contexts.

Perhaps he means that there is a difference between the use of heritability as a descriptive tool and its use as a causal explanation of variation. The behavior genetics literature seems not to make this distinction.

Schoenemann claims that behavioral geneticists such as Plomin are bent on testing environmental influences, and as evidence he cites Plomin et al. (1997). This is the same Plomin who in 1993, in an influential psychology journal, reported on the heritabilities of 23 “change” and “continuity” traits from children aged 14 to 20 months. For change, 2 of the 23 heritabilities were greater than 40% and 17 were less than 15%, but the abstract describes this as “evidence for genetic change.” For continuity, 3 of the 23 heritabilities were greater than 40% and 15 were less than 33%, but the abstract claims that “genetic factors are largely responsible for continuity.” The article in question begins with a quote from Sir Francis Galton on the importance of “qualities inherited at birth.” Thus we do not share Schoenemann’s faith that behavior geneticists, of whom Plomin is a recognized leader, have a genuine interest in understanding environmental influences.

Our reply to Hauser and Wrangham’s question why one can estimate heritabilities in corn and not people is straightforward. It would be unethical to raise groups of people in “identical” environments and then do one-generation selection experiments to estimate heritabilities. And the results would be totally uninformative, since they would only speak to heritability in that artificial environment. Similarly, approaching the question through twins alone is impossible because, as we say, we cannot estimate the environmental differences and similarities in MZ and DZ pairs from their correlations alone. Other problems with twin studies are discussed extensively in our article. “Impossible” may be a little strong, since someday someone might invent a magic lifetime-environment-integrating meter, but we’ll stick with it.

There is some disagreement among the commentators as to the equating of evolutionary psychology’s brain modules with organismic structures such as limbs or uteruses. Tattersall raises the issue of adaptationism in criticizing this equation of structure and adaptation. This is a battle that we evolutionary biologists have fought in many guises over the past 75 years. Evolutionary psychology gives the argument a new twist, however. It claims that these imagined structures—biological brain modules or decision-making algorithms—are the adaptations that became universally fixed in humans in

response to the environment of evolutionary adaptation and that they determine human nature. Patrick Bateson (2002:2212), in his critique of Pinker’s (2002) *The Blank Slate*, makes the point succinctly: “What Pinker happily calls human nature is in reality individual nature and depends critically on the circumstances of that person’s life.”

Hagen begins with the proposition that the structures in organisms are adaptations, exactly the position against which Tattersall argues. Along with many evolutionary psychologists, he goes even farther with his claim that cognitive processes also have functional structure and therefore must be adaptations. It is this behavioral pan-adaptationism that we argue against in our article. As Bateson (2002:2212) writes, evolution is helpful in studies of behavior, but “it does not follow that all examples of present-day behavior that clearly benefit the individual in the modern world are products of evolution.”

Hagen repeats another error that permeates evolutionary psychology, the idea that evolution, including that of human behaviors, works by design. Pinker’s (2002: 52) version of this position is “Signs of engineering in the human mind go all the way up, and this is why psychology has always been evolutionary.” This is where the mainstream science of biological evolution and evolutionary psychology part: evolution is a matter of contingency and tinkering as well as natural selection. For humans in particular, we must also incorporate cultural contingencies, feedbacks to and from the environment as well as cultural transmission.

Hagen considers the existence of uteruses (organismic structures) in females but not males and differences between the sexes in reproductive strategies as due to “genes that are identical (or nearly identical) in all humans.” He then goes on to quote Tooby and Cosmides (1990b) to the effect that “important human differences arise not from minor genetic differences but ‘from exposing the same human nature to different environmental inputs.’” We agree with this last statement, but it is exactly why one cannot equate behaviors and organs; uteruses could not appear in human males in any of the environments commonly experienced by humans or our ancestors over the past few million years. He also fails to understand how much easier it would be for changes in genes controlling the timing and quantity of hormone production over many tens of millions of years to modify developmental pathways so that a uterus would be produced in individuals carrying two X chromosomes than for changes in the intricate neuronal connections in the brain to produce an extensive suite of different behaviors triggered by XX or XY genomes in perhaps a million years or less.

We also think it possible that fear of snakes is more readily elicited in the human brain than fear of flowers, just as we think that responses to visual stimuli are more likely to dominate over chemical ones. But the key point is that the behavioral differences in this response among individuals and cultures are clearly determined environmentally, as is suggested by the lack of fear among New Guineans and many others. And it is precisely the pu-

tative explanatory value of genetic differences in eliciting such everyday behaviors that is the only significant claim of evolutionary psychologists and behavioral geneticists.

Holcomb suggests that we use the quantitative theory of natural selection to establish an interface between evolutionary psychology and behavioral genetics. He argues that this can be accomplished by invoking balanced polymorphisms induced by natural selection. This just won't work. On the one hand, balanced polymorphisms (which are very rare in humans) cannot explain biological universals, and, on the other, the properties of the heritability statistic, the basis of behavioral genetics, are violated in the presence of natural selection. This is a very confusing interface indeed.

Holcomb admonishes us to distinguish obligatory from facultative traits. In the absence or ethical impossibility of the necessary genetic experiments, it becomes a matter of speculation whether variation in, say, ability to detect cheaters is due to different learning experiences or merely irrelevant environmental and random perturbation on a genetically fixed module in the brain (obligatory). Stories about the environment of evolutionary adaptedness do not help to resolve this issue scientifically. Holcomb claims that we should engage with evolutionary psychologists to make the concept of such an environment "precise." He says a "relevant environment of evolutionary adaptedness is roughly the history of selective forces relevant to the evolution of a trait, not confined to the Pleistocene or to a common environment for all humans." Hagen enlightens us by explaining that the environment of evolutionary adaptedness "of the lung . . . includes an oxygen atmosphere." An attempt to refine this empty "concept" would be a total waste of time, especially since it has been incredibly difficult to document even a few selective forces on genotypes in living natural populations (e.g., Burton and Feldman 1983, Curtsinger and Feldman 1980, Ehrlich and Camin 1960, Ehrlich and Holm 1963, Endler 1986).

Hauser and Wrangham support the evolutionary psychologists' claims for the existence of universal (biological) brain modules in humans; they call them "mechanisms." But in discussing mate choice they allow variation among cultures. The existence in some humans of a preference for specific phenotypes in the opposite sex does not imply that there is a mechanism for it or genes for it or that it is innate. It is this postulation that there is a mechanism, which entails innateness, that is dangerous, because it is often elevated to the status of *result* from evolutionary psychology. They claim that we are attacking a "straw man carrying a basket of red herrings." Consider, however, the list offered to the public by Nicholas Wade (*New York Times*, September 17, 2002) of what Steven Pinker views as innate human behavior and abilities. These include reciprocity, ethnocentrism, variation in intelligence, a moral sense, and intuitions about physics, biology, probability, engineering, psychology, and economics. Robert Richards (*New York Times*, October 13, 2002) describes this view of human nature as "largely inscribed by indelible genes," a "ge-

netically fixed human nature" that has evolved even to determine our artistic preferences. Although Hauser and Wrangham may argue against complete genetic determinism or for the importance of interactions and of cultural evolution, it is clear that very visible evolutionary psychologists are not so careful in their public positions. The questions that Hauser and Wrangham conclude with are indeed interesting and important. We just don't agree that evolutionary psychology, as currently practiced, is the framework in which to address them.

Laland and Brown raise the interesting possibility that evolutionary psychology may be more attractive than methodologically more rigorous disciplines such as human behavioral ecology or formal studies of cultural evolution. Rigorous studies of the role that genes play in producing human (and other species') phenotypes are, as Laland and Brown point out, much more challenging than the "unsupported and dangerous claims for genes" that constitute much of behavioral genetics and most of evolutionary psychology.

We do not dismiss evolutionary thinking about humans as having no value. On the contrary, we advocate careful, rigorous empirical and theoretical studies that address the role of evolution in the human sciences and, as Laland and Brown point out with reference to cultural evolution, where potentially fruitful research avenues might be. We agree with them and with Aunger (2000) that artifacts created by humans can have a major subsequent impact on the human environment as an ecological inheritance. Dubrovsky is correct in emphasizing Waddington's view that the organism's environment which affects natural selection is the "more or less direct result of the animal's own behavior." Niche construction can be a major force in evolution, especially human evolution, in which the cultural component is so strong.

However, this is a deeper line of analysis than advocated by most proponents of memetics, who treat memes as bits of culture that have a strategy to maximize their replication, like "selfish genes." Our view of cultural evolution as a center of the human sciences incorporates individuals' preferences, historical and economic contingency, and, in some cases, interactions with genes that cannot be resolved by linear statistical models and a heritability number.

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