Comment by Paul Ehrlich & Marcus W. Feldman

Genes, environments & behaviors

Our large brains are surely at the center of our humanity. But it is equally certain that few organs are the subject of more misinformation in scientific and public discourse – especially in the widespread notion that most behaviors controlled by our marvelous brain are somehow programmed into it genetically. A typical treatment in the popular press is this overexcited claim by columnist Nicholas Wade in the *New York Times*: "When . . . [the human genome] . . . is fully translated, it will prove the ultimate thriller – the indisputable guide to the graces and horrors of human nature, the creations and cruelties of the human mind, the unbearable light and darkness of being."[1]

Wade may get a pass for being a journalist, but some scientists are equally confused. Molecular biologist Dean Hamer wrote: "People are different because they have different genes that created different brains that formed different personalities," and "[u]nderstanding the genetic roots of personality will help you 'find yourself' and relate better to others." As distinguished a neurobiologist as Michael Gazzaniga is guilty of

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1 The authors thank Richard Lewontin, Deborah Rogers, Robert Sapolsky, and Michael Soulé for their comments on earlier versions of the manuscript. N. Wade, "Ideas and Trends: The Story of Us; The Other Secrets of the Genome," *New York Times*, February 18, 2001, sec. 4, 3.

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the misleading claim that "all behavioral traits are heritable";[2] and molecular evolutionists Roderick Page and Edward Holmes have asserted that "genes control 62% of our cognitive ability."[3] In fact, an entire neo-field labeled evolutionary psychology has sprung up based on the misconception that genes are somehow determining our everyday behavior and our personalities. It is a field that believes there are genetic evolutionary answers to such questions as why a man driving an expensive car is more attractive than one driving a cheap car.[4]

So even well-educated and thoughtful observers have been persuaded by the language of heritability. With expressions such as 'genes are responsible for 50 percent of,' or 'genes contribute 50 percent of,' a behavior, this language gives the impression that genetic and environmental contributions to human behaviors are actually separable. They are not.

Heritability was originally introduced in the 1930s in the context of agriculture. It is an index of amenability to selective breeding under environmental conditions that the breeder could control. This index, now often termed 'narrow-sense heritability,' is the fraction of all variation in a trait that can be ascribed only to genes that act independently of one another and whose joint effect is the sum of their individual effects. One easy-to-understand way of measuring heritability is through a one-generation selection experiment. Individuals with extreme values of a trait are bred to one another – for example, the heaviest individuals from a hog population. The offspring are then raised in the same environment, and their average weight calculated. If the average weight of the offspring doesn't increase over that of the entire population (not just of the heavy parents) in the previous generation, the heritability is zero. On the other hand, if the average weight of the offspring equals that of their heavy parents, the heritability is 100 percent.

In the 1960s, the term 'heritability' was adopted by some students of human behaviors who wanted to know what fraction of the variation in these behaviors was primarily attributable to genetic differences and what percentage to environmental differences. Because controlling the environments of human subjects is not possible, however, this fraction – now called 'broad-sense heritability' – includes variation from interactions between genes and environments. That fraction of variation is nevertheless interpreted as determined by genes, thus inflating the heritability.

In other words, this new heritability statistic assumes no relationship between genetic transmission and environment, e.g., that the IQ scores of parents cannot affect those parts of the environment that might interact with genes to influence a child's IQ. The amount of stimulation parents provide their young children, the nature of dinner-table conversations, and the number of books in the home are thus taken to be independent of any genetic influences on children's IQ. When this independence assumption is violated, there is gene-environment correlation – exactly the correlation that agricultural experiments to estimate narrow-sense heritability eliminated by holding environments constant. But with human behav-

2 M. S. Gazzaniga, *The Ethical Brain* (New York: Dana Press, 2005), 44.

3 R. D. M. Page and E. C. Holmes, *Molecular Evolution: A Phylogenetic Approach* (Oxford: Blackwell, 1998), 119.

4 D. M. Buss, *The Evolution of Human Desire* (New York: BasicBooks, 1994), 99 – 100.

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iors such designs are impossible, and the correlation between parental IQ and the offspring's environment may contribute to the heritability.

Many of the high estimates for heritability, and the resulting interpretation that human behavioral traits are heavily influenced by genes, have been derived from comparisons of identical twins (who originate from a single fertilized egg) and fraternal twins (from two eggs). These estimates are based on the fact that identical twins share exactly the same hereditary endowment, while fraternal twins, on average, share only 50 percent of their genes.

But many assumptions about twins inflate twin-based estimates of broad-sense heritability. One is the 'equal environments' assumption, that variation in environments created by parents to which identical twin pairs are exposed is the same as those to which fraternal pairs are exposed – i.e., that there is no difference between the way parents treat identical and fraternal twins. Statistical estimates of the differences in the environmental exposure of identical and fraternal twins *outside of the parental contribution*, however, are not usually made. Some studies have found that the correlation between IQ and the environments *not* transmitted by the parents of identical twins is much higher than that of fraternal twins.[5] Thus, factors in the nonfamilial environment of identical twins are often more similar than those of fraternal twins, but this difference between identical and fraternal twins is usually ignored.

It might be thought that some of the problems with twin studies may be overcome if the identical twins under study were reared apart, that is, in different families. In a perfect experiment of this kind, all observed differences between the twins should be environmental, and high levels of similarity of the pair should be due to their identical genes. It turns out not to be so simple. First, separated twin pairs are rare, and the reasons for the separation are not usually known. Second, the twins share the prenatal environment of the ovary, fallopian tube, and uterus, which could be very influential in producing similar developmental pathways. Third, the separation is frequently carried out well after birth so some shared early postnatal environmental effects could mistakenly be interpreted as genetic. Fourth, twins have often been placed in separate homes that are similar in aspects that may be important for the traits under study, for example, in homes of relatives of their parents. The environments are thus not a random sample of all possible environments. Kamin and Goldberger documented these problems with the well-publicized Minnesota study of twins reared apart.[6] All of these effects add to that component of variation that is interpreted as genetic, with the result that estimates of genetic heritability based on identical twins raised separately are biased upward.

At first glance, some of the stories of the similarities of identical twins raised separately seem extraordinary examples of the power of genetic identity. Two men separated near birth grow up to be beer-drinking firefighters and grasp the beer cans in the same unusual way, hold-

5 C. R. Cloninger, J. Rice, and T. Reich, "Multifactorial Inheritance with Cultural Transmission and Assortative Mating," *American Journal of Human Genetics* 31 (1979): 176 – 198; M. W. Feldman and S. Otto, "Twin Studies, Heritability and Intelligence," *Science* 278 (1997): 1383 – 1384.

6 L. J. Kamin and A. S. Goldberger, "Twin Studies in Behavioral Research: A Skeptical View," *Theoretical Population Biology* 61 (2002): 83 – 95.

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ing the little finger under the can.[7] But they were raised in similar lower middle-class Jewish homes in New Jersey. Being a firefighter is an ambition of many males, and firefighters are not notorious for being addicted to wine. Furthermore, it is well known that physical attributes of people greatly influence how other people treat them. Individuals with identical genomes are usually strikingly alike in appearance, and within the same culture they will be treated more similarly than randomly selected individuals of the same gender from the same occupational and age groups. Resemblance in body structure (strong in identical twins) would probably also make it comfortable to hold containers in the same manner, and we doubt if even the most dedicated hereditarian would seek a gene for use of the pinky in beer drinking.

Ever since narrow-sense heritability was first used, it has been well understood by geneticists that an estimate of the genetic influence on a trait's variability depends on the particular population and the particular environment in which the trait was measured. Furthermore, even a very high heritability measured in a population cannot be used to infer something about any single member of that population. Suppose a population is known to have higher than average blood pressure. Would a physician treating one individual patient from that population prescribe an antihypertensive drug on the basis of the population statistic? Of course not – a doctor would use detailed history and laboratory workup to decide on the appropriate treatment for that particular patient. The patient's diet or stress level (the environment) would be critical to the medical recommendation and, in most cases, likely to overwhelm any genetic effect inferred from population studies. The logic of using the heritability of some trait in a population to predict something about a member of that population would be foolish.

Recent studies of intelligence in samples of twins of different socioeconomic status strongly reinforce these restrictions on the generalization of heritability. For example, the estimated heritability of IQ in individuals from advantaged backgrounds is significantly higher than in those from disadvantaged backgrounds.[8] That is because better environments allow more variance in IQ to be expressed: potential geniuses have trouble developing into Einsteins in slums without schools. Likewise, the heritability of height in a normal human population would be greater than that in a starved one, where everyone's growth is stunted and the variance in height thereby reduced.

Individuals with Down syndrome, caused by an entire additional chromosome 21 (trisomy), develop as severely mentally handicapped if given no special treatment. But it turns out that the degree of handicap is extremely labile to the environment of rearing.[9] In fact, the day may come when an environment can be provided in which their development will be entirely normal. Moreover, not even evolutionary psychologists

7 N. L. Segal, *Indivisible by Two: Lives of Extraordinary Twins* (Cambridge, Mass.: Harvard University Press, 2005).

8 E. Turkheimer, A. Haley, M. Waldron, B. D'Onofrio, and I. I. Gottesman, "Socioeconomic Status Modifies Heritability of IQ in Young Children," *Psychological Science* 14 (2003): 623–628.

9 R. I. Brown, "Down Syndrome and Quality of Life: Some Challenges for Future Practice," *Down Syndrome Research and Practice* 2 (1994): 19–30; N. J. Roizen and D. Patterson, "Down's Syndrome," *The Lancet* 361 (2003): 1281–1289.

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have proposed that chromosome 21 is the locus of 'the intelligence gene.'

Such important gene-environment interactions preclude the partition of variation in traits like trisomy, IQ, or height into genetic and nongenetic influences. It is especially inappropriate to talk about genetic 'contributions' to such complex traits when in some environments genetic variation is not even detectable. It is equally incorrect to say, 'characteristic A is more influenced by nature than nurture,' as it is to say, 'the area of a rectangle is more influenced by its length than its width.' (Note that the area of a rectangle one hundred miles long and one inch wide is halved by reducing its length by fifty miles or by reducing the width by half an inch.)

None of this should be taken to mean that genes do not affect behavior. In fact, in a sense, they influence all behavior, at least by laying out how human capabilities differ from those of other primates. If genes did not, in the course of development, interact with pre- and postnatal environments to generate the brain – some of the major patterns of its organization, and its principal modes of interaction with hormonal systems – the human behaviors that interest us would not occur at all. Genomic disparities between species doubtless influence differences in the general configuration of the systems that control behavior.

But it is clear from the long pre- and (especially) postnatal environmental programming that these systems must undergo to produce a behaviorally 'normal' person that genes are not responsible for embedding detailed instructions on how to act, or even 'tendencies' toward certain kinds of behavior. Environmental inputs are so extensive that the cortex of the brain is not fully developed until the mid-twenties. In view of this, it's not surprising that nothing indicates that genes favored by selection while our ancestors were hunter-gatherers significantly influence such contemporary individual behavioral characteristics as choice of beers or marriage partners.

For many behavioral traits, especially serious psychiatric disorders, some individual genes have been shown to play a role in some environments but not in others. Consider research by psychologist Avshalom Caspi and his colleagues on the effects of having different forms of a gene involved in the transport of serotonin, a compound that is involved in transmitting signals along certain nerve pathways. Which form an individual possesses apparently influences whether stressful events will produce depression. Having the 'wrong' gene, however, only makes a difference if an individual is exposed to a stressful environment early in adult life – a beautiful example of gene-environment interaction.[10]

Many other cases illuminate the failure of genes to 'control' behavior. The original Siamese twins, Chang and Eng, were joined for life by a narrow band of tissue connecting their chests. Despite their identical genomes, they had very different personalities. One was an alcoholic, the other sober; one was dominant, the other submissive. Equally fascinating is the story of the Dionne quintuplets, five genetically identical little girls who, in the 1930s, were essentially raised in a laboratory under the supervision of a psychologist. When the girls were only five, the psychologist wrote a book that expressed his astonishment at how *different* the little girls were – something confirmed by their very different

10 M. Rutter, *Genes and Behavior: Nature-Nurture Interplay Explained* (Oxford: Blackwell, 2005).

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life trajectories. One had epilepsy, the others did not; some died young, the others old; some married, others remained single; and so on. Similarly, the identical Marks triplets grew up with different sexual orientations, two straight and one gay; one of the two identical Perez girls chose to change her sex with hormones and surgery and married a woman, while the other twin remained female and married a man.[11]

But one does not even have to look at such extreme cases to see that genes are not controlling human actions; evidence that common behaviors are not genetically determined is superabundant. Perhaps the most impressive comes from thousands of cross-cultural 'experiments' in which children from one culture are raised from an early age by adoptive parents from another. Invariably, the children mature with the language and attitudes of the adoptive culture.

Also impressive is the ease with which culture overrides the only 'commandment' we can be sure is contained in everyone's DNA: ensure that your genes are maximally represented in the next generation, either by having more children or by helping your relatives (who tend to have the same genes) to reproduce. Differential reproduction of genetically different individuals (not explicable by chance) *is* natural selection, the creative force in evolution. We wouldn't be here if our ancestors hadn't been effective reproducers of their genes, if they hadn't had high 'fitness.' But culture (part of the environment) has led human beings to limit their reproduction as far back in history as we can trace, all the way to the ancient Egyptians who used crocodile-dung suppositories as contraceptives (which we are convinced were very effective!).[12] Indeed, although evolutionary psychologists like to imagine that rapists are programmed to assault women in order to reproduce themselves – that is, to increase their fitness – over half of all rapes occur in circumstances (e.g., victims too old or too young, no ejaculation into the vagina) where fertilization is impossible, and in more than a fifth of cases more force is used than would be required to achieve the supposed reproductive goal.[13]

Most definitive, though, is the problem of gene shortage.[14] Our roughly twenty-five thousand genes can't possibly code all of our separate everyday behaviors into the human genome. After all, we have less than twice as many as required to make a fruit fly, and just a few more than those that lay out the ground plan of a simple roundworm. Even if the human brain had not evolved for flexibility but instead were programmed for stereotypic behavior, our genes couldn't store enough information to accomplish it. Genes are not little beads with instructions like 'grow up gay' engraved on them. They are instructions that, in a very complex mechanism, can be translated into a sequence of amino acid residues in a protein. It is near miraculous that these proteins – interacting with each other, function-

11 Segal, *Indivisible by Two: Lives of Extraordinary Twins*.

12 L. Manniche, *Sexual Life in Ancient Egypt* (New York: Kegan Paul, 1997).

13 J. Coyne, "Of Vice and Men: A Case Study in Evolutionary Psychology," in *Evolution, Gender, and Rape*, ed. C. Travis (Cambridge, Mass.: MIT Press, 2003), 171–189.

14 P. R. Ehrlich, *Human Natures: Genes, Cultures, and the Human Prospect* (Washington, D.C.: Island Press, 2000); P. R. Ehrlich and M. W. Feldman, "Genes and Cultures: What Creates Our Behavioral Phenome?" *Current Anthropology* 44 (2003): 87–107.

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ing in different physical, physiological, and social environments, and helping to control the production of other proteins – are able to produce an entire human body and the basic scaffolding for a brain with a trillion or so nerve cells (neurons) connected to each other by tens of trillions of intricate junctions (synapses). On average, each gene must influence many characteristics. There are obviously enough genes, interacting with each other and with diverse environments at all scales, to provide a brain that can generate all observed human behaviors. But this has confused some observers into thinking that because one gene normally affects many functions there is no gene shortage.

That fact is actually the *basis* of calling it gene shortage. It means that natural selection altering the genome to encode one behavior would inevitably change other aspects of the genome as well – so that selection increasing, say, the speed of contraction of muscle fibers would quite possibly modify the connections between some neurons that, say, transmit visual information from the retina to the brain. Because of the small number of genes in the human genome and the ubiquity of interactions between proteins and between proteins and environments, natural selection must ordinarily entrain a multiplicity of changes. It must operate on a genome enormously 'amplified' in development by the multiple uses of the proteins produced by single genes, by the alternative ways the proteins are assembled, by the small RNA molecules that often control the expression of multiple genes, and by the epigenetic phenomena that may have differing effects even on identical genotypes.[15]

This may be why it has been so difficult to demonstrate that natural selection has changed more than a tiny fraction of genes during the transition from chimpanzee to modern human being. Changing just a few genes can have effects that totally transform an entire organism. Thus, most population geneticists – remembering linkage, pleiotropy, epistasis, and developmental complexity – reject evolutionary psychology as a theoretical paradigm: its predictions ignore how difficult gene-gene and gene-environment interactions make it for selection to operate on just one phenotypic attribute. If we had trillions of largely independent genes, then it might be possible for selection (were it strong enough and time available long enough) to program us to rape, be honest, detect cheaters, excel at calculus, or vote Republican. But the number of *independent* genes is much smaller than twenty-five thousand.

Perhaps the most interesting thing about all the attention paid to whether nature or nurture controls behaviors is not that individuals with identical genomes often behave very differently, but that those same individuals exposed to extremely similar environments also turn out to behave quite differently. This has been clearly demonstrated in mice, where genetically uniform strains exposed to laboratory environments made as identical as possible still behaved differently.[16] Indeed, nonidentical human siblings, who share half of their genes, the same parents, and apparently very similar environments, often seem more

15 M. F. Fraga et al., "Epigenetic Differences Arise During the Lifetime of Monozygotic Twins," *Proceedings of the National Academy of Sciences USA* 102 (2005): 10604 – 10609.

16 J. C. Crabbe, D. Wahlsten, and B. C. Dudek, "Genetics of Mouse Behavior: Interactions with Laboratory Environment," *Science* 284 (1999): 1670 – 1672.

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unalike than unrelated people drawn from the same population. Think of all the 'isn't it weird that Johnny and Sammy Smith are so different' anecdotes – many more, it seems to us, than 'isn't it weird that Johnny and Sammy Smith are so similar.'

If genes don't 'determine' our behavior, how can it be that obvious aspects of our (or mice's) environments don't either? We don't know for sure, but we can make some guesses. One is that researchers have not yet identified key environmental variables that are subtle to them but central to a behaving organism – be it a mouse with a genome that makes it love alcohol or Johnny trying to get along with Sammy. Another is that prenatal influences may put genetically similar (or identical) individuals on quite different behavioral trajectories. There is a tendency to think, first there's fertilization, and then some nine months later a baby pops out. But, of course, an incredibly complex series of events takes place during those nine months: cell-cell, tissue-tissue, and organ-organ interactions; pulses of hormones; responses to pleasant and unpleasant stimuli such as voices heard through the uterine wall; and in some cases interactions with another fetus in the womb. Studies have already shown what dramatic effects prenatal environments can have. For instance, young female fetuses whose mothers had minimal diets during the Dutch famine of World War II grew up into women who were more obese than those whose mothers were well fed; they also had higher levels of 'bad' cholesterol. As more is learned about environmental influences in the womb it seems likely that many of the differences between siblings could be discovered to have prenatal origins.

Could there be another source of the sometimes dramatic differences among siblings, including identical twins? We hypothesize that there may be a 'sibling bifurcation' phenomenon, in which individuals having close relationships with others early in life, either pre- or postnatal, often seek different life courses. This could be related to such things as a kin-recognition/inbreeding avoidance system; attempts by parents, siblings, teachers, and peers to distinguish related individuals; genetic differences (between fraternal twins); birth-order effects; and so on.

We now know more than enough about the human genome and human development to see that the notion of 'genes for behaviors' is misguided. For complex traits such as normal behaviors, few cases have been found where a specific gene, or even many genes, greatly influences variation in the trait. It is clear that when genes influence traits, including behaviors, they only do so in ways that are affected by environments. Thus environments during any phase of life might alter the way in which an individual's genes function in those environments. This is, of course, a tribute to the marvelous plasticity of the human brain, which neurobiologists know changes in response to external and internal environments throughout life. It also makes ridiculous the claim that genes program our behaviors or, indeed, that genes are responsible for some specified fraction of any human behavior.

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