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Genetic Differences and Human Identities

On Why Talking about Behavioral Genetics Is Important and Difficult

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On the cover: *Awakening Woman,* by Paul Klee. ©ARS, NY Photo: © Bildarchiv Preussischer Kulturbesitz/Art Resource

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Genetic Differences and Human Identities

On Why Talking about Behavioral Genetics Is Important and Difficult

BY ERIK PARENS

A *New York Times* headline announces, "First Gene for Social Behavior Identified in Whiskery Mice."[1] "Attention-Deficit Gene Is Located," asserts a headline in the *Wall Street Journal*.[2] A Case Western Reserve University press release declares, "Researchers Discover Anxiety and Aggression Gene in Mice."[3] Myriad Genetics Inc. proclaims that the company "has discovered a novel gene that causes human obesity."[4]

Some of this "gene-for" language is run-of-the-mill hype. The language is intended to attract attention, and ultimately dollars.[5] Some of it, however, is shorthand to communicate new findings that researchers believe may help explain why people behave the way they do.[6] One aim of this report is to help the reader get beyond the extravagant claims to begin to appreciate what behavioral geneticists hope to find and what they have—and have not—found.

Because behavioral genetics aspires to illuminate human *behavior*, it raises questions about human *freedom*. What does knowledge about the influence of genes on behaviors mean for my belief that I am free to choose particular actions? Am I free to choose the qualities of my temperament, like how sunny or empathic or outgoing I am? Should increasing knowledge of genetics affect a criminal court's proceedings? And so forth.

Because behavioral genetics investigates genetic *differences*, because it aspires to understand how differences at the level of the gene are related to differences in traits, it also raises questions about human *equality*. Would it affect our understanding of moral equality if we learned that genetic differences help to explain why we behave and appear differently, if we learned, for example, that some individuals are genetically predisposed to antisociality or hypergenerosity, alcoholism or teetotaling, low intelligence or high?

Questions about human freedom and equality are ultimately questions about our self-conceptions or "identities." Thus we need to understand not only the facts that behavioral geneticists present to us, but also what those facts mean for our self-conceptions.

With a generous grant from the Ethical, Legal, and Social Implications program at the National Human Genome Research Institute, The Hastings Center and the American Association for the Advancement of Science undertook a three-year project called "Crafting Tools for Public Conversation about Behavioral Genetics." Over the course of the three years, the project's working group came together five times for meetings spanning several days. We also spent countless hours in e-mail conversations. The working group was made up of people who do the science for a living, people who think about the history, sociology, and ethics of the science, and a variety of others. Nobody assumed that molecular biologists or doctors would learn the finer points of law or philosophy or that the lawyers or philosophers would learn the finer points of molecular biology. All we assumed was that everyone brought to the table a commitment to engage in an open and respectful conversation.

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Our group's highly interdisciplinary work was facilitated by the work of many others.[7] But in spite of all the excellent work already done and the good will brought to the table, our conversation was sometimes difficult. We set out to create tools for public conversation, but underestimated how difficult it would be to conduct a fruitful conversation among ourselves. Much of this report explores why it can be difficult to talk about what behavioral genetics has so far found and what those findings mean.

Altogether unsurprisingly, one difficulty was that working group members came from different disciplinary backgrounds. Those of us from the humanities were sometimes intimidated by the languages of statistics and genetics. Sometimes the behavioral geneticists were frustrated that those of us in the humanities and social sciences did not take more time to learn the science. The behavioral geneticists also sometimes seemed (to this "humanist," anyway) impatient with the languages and concerns of scholars in the humanities and social sciences.

A second and more surprising difficulty was that there was not always agreement about the facts (not all of which are in, of course). For example, behavioral geneticists try to distinguish between two classes of environmental effects: those that make siblings in the same family alike, called "C," and those that make siblings different, called "E." Besides debating the relative magnitudes of C and E, behavioral geneticists disagree about the nature of the environmental processes that constitute these broad classes.

The problem is particularly acute for E. This class is defined as comprising everything that might make a pair of identical twins raised in the same family different from each other. But behavioral geneticists offer two different accounts of what E is like. Some argue that it will turn out to be orderly: despite being raised in the same family, the twins' environments differ in systematic ways, leading to systematic differences in their behavior. Their parents treat them differently, for example, or they go to different schools. Others argue that the critical environmental differences are unsystematic and hard to predict.

These two accounts suggest very different prospects for scientific investigation. If systematic differences are key, it should be possible to measure those differences and track their effects.[8] But if the important differences are unsystematic, it will be possible to observe their total effect but difficult to identify the individual processes.[9] As I will suggest later, there is a parallel and equally important debate among behavioral geneticists about the likelihood of identifying the systematic effects of individual genes on behaviors.

Disagreements like these are an inevitable product of intellectual integrity and can be a source of scientific progress. They can also, however, make assessing the scientific claims especially difficult for nonscientists.

Finally, our working group conversation was difficult because, of course, nobody comes to the table without feelings about what the facts *should* be. Nor can these feelings be divided neatly between scientists and humanists. During our meetings, it seemed that some from both sides of the disciplinary divide fervently wanted genetic differences to go a long way toward explaining behavioral differences, and some from both sides equally fervently wanted genetics to be of rather little use. If a given genetic finding might suggest that genetic differences can play an important role in explaining variation in intelligence, or novelty seeking, or sociality, then that finding seemed to gladden some people as it simultaneously depressed others.

Why these intense feelings? Because in some cases, I think, no less is at stake than human identities and the proper organization of societies. To what extent are whatever privileges people enjoy the consequence of natural gifts instead of luck and "gifts of nurture"? Does our current social order reflect the way things are "by nature" meant to be? Or does it reflect the contingent effects of power-seeking animals? Is it an inexorable fact of nature, or a contestable product of human choices? Ultimately, to what extent are current forms of inequality rooted in natural differences, and to what extent in human intentions? Few issues inspire as much feeling.

Given that no less than how we understand ourselves and how we think we ought to organize our society seem to be riding on how we interpret the findings of behavioral genetics, it is not surprising that the conversation about those findings is sometimes difficult. But it is a conversation we would benefit from sharing in. I hope this report, aimed at readers with a passing knowledge of high school genetics but no knowledge of behavioral genetics, will make a small contribution to others as they embark upon their own conversations.

In part 1, I introduce the reader to the sorts of questions that contemporary behavioral geneticists aim to answer. I emphasize that, whereas many kinds of scientists seek to understand what is *normal* or *typical* for a member of a species, behavioral geneticists seek to understand why individuals are *different* from each other with respect to some trait. I then give some historical background, suggesting that efforts to *explain* human differences have all too often accompanied efforts to *justify* why some individuals (or groups) enjoy more social power than others.

In part 2, I introduce the basic methods behavioral geneticists use to try to understand how genetic differences are related to observable or *phenotypic* differences. I introduce the "classical methods" of behavioral genetics, which begin with twins to try to determine how much of the phenotypic variation in a population is due to genetic variation; I take considerable time to show how the key "classical" concept of *heritability* has been misunderstood and abused. I also introduce the newer "molecular methods," which aim to identify particular genes that influence particular traits. I emphasize that behavioral geneticists themselves increasingly attend to the fundamental fact that observable traits emerge out of the staggeringly complex and constantly changing interactions among genes and envi-

ronmental factors. I also emphasize that thus far, efforts to identify specific genes that help to explain specific observable traits or phenotypes have not yielded nearly as much as the newspaper headlines have promised or the researchers have hoped.

In part 3 I explore how inquiries into behavioral genetics might affect our ideas about freedom and equality and ultimately our self-conceptions or "identities." I say why I think genetics does not offer information that should threaten our experience of ourselves as free. And I suggest that inquiries into genetic differences can be misappropriated by those who want to justify inequalities in the distribution of social power. Unfortunately, such misappropriation is a constant risk.

In part 4 I identify some of the obstacles that will face others who try to converse about what behavioral geneticists believe are the facts and what those facts mean.

In sum, I try to give a fair account of what behavioral geneticists have and have not discovered. I try to make clear how the facts they aspire to discover can be put to the pernicious purpose of justifying the status quo. And I try to make clear how the individual differences perspective of behavioral genetics can be put to the salutary purpose of affirming human genetic variation. Ultimately, this report is no more nor less than an invitation to others to share in a conversation about what the findings of behavioral genetics mean.

Part 1. CONTEXT

The "Individual Differences Perspective"

Behavioral genetics versus medical genetics

The field of behavioral genetics studies the genetic influence on complex human traits, which as Ken Schaffner puts it include "virtually any described or measured feature of an organism, including behaviors."[10] But the field of medical genetics also studies the genetic influence on complex traits. Roughly, medical genetics studies the genetic influence on those traits or behaviors that traditionally have come within the purview of medical doctors—cancer, diabetes, hypertension. Behavioral genetics studies the genetic influence on those traits or behaviors that traditionally have come within the purview of psychologists (and psychiatrists). Those traits include both "mental disorders," like bipolar disorder and schizophrenia, and various cognitive disabilities, including those associated with reading and speech. Psychologists have also traditionally studied behaviors and traits like intelligence and aggressiveness, and so do behavioral geneticists.

The border between medicine and psychology is fuzzy and changing, however. Depression was once squarely within the purview of psychologists and psychiatrists but increasingly is within the purview of internists as well. Many researchers in medical genetics study Alzheimer's, but now so do researchers in behavioral genetics. While the departmental names may be different, when it comes to the newer molecular approaches, the research and methodological questions are largely the same.

Classical versus molecular approaches to understanding genetic influences

Again, many behavioral geneticists today use the same "molecular methods" employed by medical geneticists. That is, both groups use DNA-based science and technology in their efforts to identify the genes that are associated with a variety of traits and disorders. But it is important to understand that before the molecular methods became available, behavioral geneticists used (and still use) a wholly different set of methods, called "classical" or "quantitative" or "epidemiological." I think it's easiest to remember the classical-molecular formulation of the distinction, so that's what I'll use. (In the literature it's more common to distinguish between *quantitative* and molecular methods).

The most basic of these classical methods use identical and fraternal twins raised together or apart to try to understand the extent to which differences at the level of the gene help to explain differences at the level of the phenotype. For reasons I will explain shortly, behavioral geneticists use the classical methods to investigate statistical correlations between the phenotypes of twins. So whereas the molecular behavioral geneticists tend to use the same DNA-based methods as their brethren in medical genetics, the classical behavioral geneticists tend to use the same statistical and psychometric methods as their brethren in psychology.[11] Thus the answer to the question, Who is a behavioral geneticist? is rather like the answer to, Who is a bioethicist? Both fields include practitioners with very different disciplinary backgrounds, and with very different temperaments and political views, who study a heterogeneous range of issues.

The species-typicality perspective versus the individual-differences perspective

According to Robert Plomin and his colleagues, many sciences tend to look at human beings from a "species-typicality perspective."[12] That is, researchers in many fields seek to discover the "typical" or "normal" form of a trait for a member of the human species. In neuroscience, for

example, researchers seek to understand what constitutes typical human cognition.

Behavioral geneticists, however, study human traits and behaviors from a very different although often complementary perspective. Instead of asking what is typical or normal for a species, they ask why members of a population are *different* with respect to some trait. Specifically, they ask how genetic differences help to explain why individuals appear different, or are "phenotypically different."

Instead of asking, What is cognition? (as does the scientist working out of the species-typicality perspective), the behavioral geneticist working out of the individual-differences perspective asks questions like, Why do human beings score differently on IQ tests? This is true for classical behavioral genetics, which focuses on "naturally occurring genetic variation," and it is true for the newer molecular strategies, which aim at "the identification of the DNA sequences that make us different from each other."[13]

Notice that the individual-differences perspective begins from the fact of human variation, the fact that most human traits are distributed in a continuous fashion, which can visually be represented by a bell-shaped curve. The rare exception to the rule are "binary" traits, such as Huntington's, which one either has or does not have. According to the individual-differences perspective, complex traits like depression, even schizophrenia, cannot adequately be understood in binary terms.[14]

Insofar as the individual-differences perspective proceeds from the premise that we should always expect variation with respect to most traits—insofar as it proceeds from the premise that variation is "natural"—it could be a wonderful ally to those who would criticize views that privilege "the normal" or "typical." Admittedly, there is no logical connection between the (statistical) *fact* that traits are continuously distributed and the social or ethical *commitment* to affirm variation as good.[15] But appreciating the prevalence of variation might help to make us more comfortable with, or accepting of, difference. Unfortunately, although the individual-differences perspective is surely compatible in principle with an affirming attitude toward human variation, it has been used for very different purposes in the past.

Some History

Explaining versus justifying differences: distinct but all-too-often convergent desires

The discipline of statistics was born in the middle of the nineteenth century, when L.A.J. Quetelet introduced the bell-shaped curve to describe the variation in heights among French soldiers. Later in that century, investigators like Francis Galton began to try to understand how inherited differences could help to explain what seemed to be a naturally and predictably occurring range of phenotypic differences among humans.[16]

The recognition that human traits were distributed in a way that could be represented by the bell-shaped curve, just like animal and plant traits, was part of the larger effort, then gaining steam, to understand human beings as part of the natural world. Indeed, behavioral genetics can be understood as an expression of that same marvelous, radical, naturalistic desire that is most famously associated with Galton's cousin, Charles Darwin: the desire to understand human beings as part of the natural world, to give a natural scientific explanation for why human beings appear and behave as they do.

So one reason behavioral geneticists desire knowledge of the genetic influence on human behavior is that such knowledge is inherently interesting. They also desire it because they believe that, eventually, it will reduce human suffering. But while behavioral geneticists have long been interested in the nature and treatment of mental disorders, their work has not yet produced knowledge that can actually reduce the suffering of individuals afflicted with diseases like schizophrenia and autism. To the extent that it has been able to demonstrate, however, that bad parenting is not the cause of such diseases, it has been able to reduce the suffering of those parents (especially mothers) who once were made to believe that their parenting style caused their child's disease.

In a classic 1966 paper, Leonard Heston compared foster children who were separated at birth from their schizophrenic biological mothers and foster children who were separated at birth from their psychologically healthy biological mothers.[17] Schizophrenia appeared only in a subset (about 16 percent) of the children whose biological mothers had schizophrenia, a finding which suggested that rather than bad parenting, genetics plays a powerful although only partial role in the emergence of schizophrenia. (About 84 percent of the biological children of schizophrenic mothers did *not* exhibit schizophrenia.)

But almost from the beginning, the marvelous, radical, naturalistic desire to integrate the study of humans into the study of the rest of nature, the desire to *explain* why human beings appear and behave differently, has converged with, or has been co-opted by, one of the ugliest and meanest of human desires—the desire to *justify* the status quo, to give a naturalistic account of why those who have, have, and why those who lack, lack. Efforts to explain human differences scientifically certainly do not logically entail efforts to justify hierarchical forms of social organization. Still, the history of how the sciences of human differences have been used is long and sordid.[18]

In 1869, ten years after Darwin published *The Origin of Species*, his cousin, Francis Galton, published *Hereditary Genius*. That book advanced the idea that intellect and character were "natural abilities" that could be bred into future generations, and he envisioned a meritocracy in which intelligence and character—not money—would determine one's place in the social hierarchy.

The idea of using tests to determine an individual's natural "intelligence" was created by the French psychologist Alfred Binet in the beginning of the twentieth century. Binet's original and innocent intention was to use such tests to identify children with cognitive disabilities so that they could receive the special attention they needed. But the American H.H. Goddard took Binet's tests and put them to less innocent uses. Goddard's research around the time of World War I convinced him that, among the newly arrived Jews, Hungarians, Italians, and Russians, a huge percentage were "feeble-minded"—or to use his technical term, "morons." Given his view that intelligence was a simple trait transmitted from generation to generation in the same simple pattern that color was transmitted in Mendel's pea plants,[19] and given his concern for the future of his country, Goddard recommended that "the feeble-minded be identified and kept from breeding."[20]

The Nazis, of course, also had ideas about natural differences and how to breed a better race of humans. In the wake of the Shoah, however, claims about breeding in general and about natural differences among "races" with respect to intelligence took a break for a couple of decades. In 1969, however, Arthur Jensen published a paper which insinuated that, on average, "whites" score better than "blacks" on IQ tests because of a natural or "genetic" difference between the "races."[21] In 1994, Richard Herrnstein and Charles Murray revisited Jensen's claim. They spent much of their book criticizing social explanations for the black-white test gap and marshalling evidence for the genetic influence on intelligence. They warned against squandering scarce resources on social approaches to narrowing the gap. And, like Jensen, they never explicitly said that blacks are genetically inferior to whites with respect to intelligence. Indeed, they say they are "agnostic" about whether "either the genetic or environmental explanation has won out to the exclusion of the other."[22] It is easy to see how their insinuation, however, can be used to justify whites' social power.

Does the future of behavioral genetics look brighter?

In the past, those who professed to investigate the genetics of complex behaviors fell prey to deeply mistaken scientific claims, such as that traits like "intelligence" or "mental deficiency" are transmitted in the same simple pattern that color was transmitted in Mendel's peas, and this work was used, even in the recent past, to justify unjust forms of social organization. In contrast, much new behavioral genetics work appears to paint a very complex picture of gene-environment interactions,[23] and it can be put to good uses. For example, it may be of use in explaining why some people suffer more than others when exposed to environmental toxins like child abuse and stress.

Two of the most vivid recent examples of behavioral geneticists paying attention to the extraordinary complexity of gene-environment interactions have been published in the journal *Science* by Avshalom Caspi, Terrie Moffitt, and their colleagues. The most recent paper, from the summer of 2003, studied 847 members of a group in New Zealand from the age of three through their twenties.[24] The paper considers the role of a gene for a molecule (5-HTT) that affects the transmission of serotonin. The 5-HTT gene has two common versions: one is long and the other short. Earlier animal studies had suggested that those with two long alleles handled stress better than animals with two short alleles. Caspi and colleagues determined whether members of their subject population had two short alleles, two long alleles, or one of each, and then asked how many stressful events (involving employment, finance, health, housing, relationships) each member of the group experienced between the ages of twenty-one and twenty-six.[25] It turned out that those who experienced significant stress and had two short alleles were about twice as likely to become depressed as those who experienced significant stress but had two long alleles. That is, this study indicates how behavioral geneticists are increasingly doing what many of them have said they want to do: they are looking at the *interaction* between genotypes and environmental variables.

Caspi, Moffitt, and colleagues had published a paper in the summer of 2002 that made a similar finding regarding the interaction of childhood abuse and a gene for an enzyme (MAOA) involved in the regulation of several neurotransmitters, including serotonin.[26] The 2002 paper studied the approximately 500 white boys in the same group of New Zealanders. It reported that the likelihood of a boy becoming antisocial as an adult was affected jointly by his genotype and his early childhood environment. Boys who were abused as children and whose genotype predisposed them to produce low levels of that enzyme (MAOA) involved in neurotransmitter regulation were twice as likely to become antisocial as were those boys who were abused but whose genotype predisposed them to produce high levels of that enzyme. This study proceeded from a plausible hypothesis about how, physiologically, a genotype might affect a phenotype, and it found that people with one genotype were more vulnerable to environmental toxins than were those with a different genotype.

That behavioral geneticists are studying such interactions is good news. The bad news is that the MAOA study was the subject of a piece in *Popular Mechanics* titled "Criminal Genes."[27] The piece in *Time* about the MAOA study was titled, "The Search for a Murder Gene."[28] Although both reporters told subtler stories than the titles announced, the idea that some kids are simply born bad persists. Even though the Caspi and Moffitt MAOA study is about the interaction between genes and child abuse, the story titles in the popular press suggest that genes "cause" criminality. The "criminal genes" language is dangerous at best. When Americans talk about criminality, they often have in mind strung-out poor kids sticking up local bodegas rather than greedy rich guys sticking up

their company's pension fund. Thus loose talk about criminal genes risks reinforcing hateful stereotypes about the difference between "those poor wretches on the bottom" and "us worthy, talented ones on top." It risks being coopted by those who want to justify why some groups enjoy more social power than others. That danger was around in Galton's time, and it's around in ours.

The need for public conversation—and for respecting a special obligation

Since research aimed at understanding genetic influences on complex human traits is inherently interesting, since such research promises to contribute to the reduction of human suffering, and given our country's deep commitment to freedom of scientific inquiry, behavioral genetics is not going away. Nor is the desire for "scientific" justifications of why some people possess social power likely to disappear any time soon.

Given the promise of the research and the persistent danger that attends it, our only real option is to learn to talk together about it. We need to learn to distinguish between real and hyped-up findings, real and hyped-up benefits, and real and hyped-up dangers. Doing that will not be easy. As David Wasserman and his colleagues at the University of Maryland discovered in 1995 when they convened scientists and critics of the science to discuss the genetic contribution to criminality, talking together about these matters can be exceedingly difficult. In spite of Wasserman's Herculean efforts to balance the voices of enthusiasts and critics, the conference was interrupted by protestors who were angry that anyone would dignify with a conference the question, What if any role do genes play in influencing criminal behavior?[29] It is not clear, however, what would be achieved by remaining silent about a line of research that is going forward and will go forward whether or not it is publicly discussed.

I see no alternative to a public conversation about the findings and ethical and social implications of behavioral genetics, but I need to be clear about what I take to be a special obligation of all who would participate in such a conversation. Given that those at greatest risk for being hurt are those who already hurt as a result of the current organization of our society, there is a special obligation to guard against allowing research aimed at increasing knowledge and reducing suffering from being hijacked by the desire to justify the status quo. That obligation is incumbent upon all who engage in the conversation, be they journalists, bioethicists, patient advocates, lawyers, or anyone else. Perhaps first of all that obligation is incumbent upon the researchers.

To their credit, some researchers in behavioral genetics have long recognized that obligation and acted accordingly. In the wake of Jensen's inflammatory article, behavioral geneticist Irving Gottesman (who also served on our working group) was invited in February 1972 to testify before Walter Mondale's Senate Select Committee on Equal Educational Opportunity. Gottesman takes seriously the genetic influence on intelligence and thinks that IQ tests can be a useful measure of an individual's cognitive ability. He agrees with Jensen that IQ tests are like a thermometer: you don't throw it away because it tells you that you have a fever and you don't want one. We should not simply ignore the fact that, on average, black kids are scoring worse on IQ tests than white kids. But Gottesman painted a more complex picture:

> [T]here are at least two situations I can imagine where you would not take action as a result of the thermometer reading. If, unknown to the examiner, a child had been sucking on ice cubes or drinking hot tea before testing, you would be obtaining accurate but misleading information. I would suggest to you with respect to the IQ testing of many disadvantaged children, that the readings reflect an intellectual diet of ice cubes between the time of conception and entrance to elementary school.[30]

In 2003, thirty-one years after Gottesman's testimony before the Mondale committee, he, Eric Turkheimer, and colleagues published a study that addresses the influence of socioeconomic status (SES) on IQ scores.[31] The new study, based on data from a project that Paul Nichols and Elving Anderson had examined earlier,[32] finds that the importance of genetic differences in explaining differences in IQ scores depends on the SES of the persons taking the tests. Genes appear to help explain the differences in test scores among *high* SES children, but not among low SES children. The implication is that the low SES children had been sucking on ice. This study is an example of how behavioral genetics can be wielded in support of progressive social interventions. It provides scientific support for programs like Head Start, which aim to put all kids on a level playing field. (For complexities surrounding what a level playing field means in the wake of genetic research, please see Part 3 below.)

The differing commitments and interpretations of behavioral geneticists are reflected in the history of the Behavioral Genetics Association. In his 1995 presidential address, "Ideology and Censorship in Behavior Genetics,"[33] the outgoing president, Glayde Whitney made unquotably ugly remarks about the genetic explanation for the difference in the rates at which blacks and whites commit murder in the United States. Yet many members of the association—including the incoming president, Pierre Roubertoux, walked out of the lecture in protest.[34] Roubertoux resigned the presidency, although he remained a member of the association.

Many behavioral geneticists, including members of our working group, have spoken against gene hype in general and against claims that could be used to justify a system of racial hierarchy. As our public conversation about behavioral genetics moves forward, such people will be indispensable interlocutors. But for that conversation to move

forward, people who are not behavioral geneticists need to try to grasp some of the scientific basics. If we're clueless about what behavioral geneticists do, we won't adequately appreciate either the potential benefits or the abuses of their work.

Part 2. AN INTRODUCTION TO SOME OF THE FACTS

Classical Approaches: How Much Do Genetic Differences Matter?

The classical (as opposed to molecular) approaches of behavioral genetics employ what are referred to as twin, adoption, and family studies. These studies seek to determine how much influence genes have on a trait—in a particular population, in a particular environment, at a particular time—in comparison to the environment.

It is a fascinating question: To what extent do we appear and act differently as the result of genetics and to what extent are the differences the result of environment? Or in the language of behavioral genetics: To what extent is phenotypic variation a function of genetic variation and to what extent is it a function of environmental variation?

As we will see below, it is crucial to understand that the question addressed by the classical approaches is about *variation*, not *causation*.[35] Perhaps it is surprising to learn, but the widely discussed classical studies cannot teach us anything about which genes cause or influence behavior, nor about how they do it. They can only suggest the extent to which genetic variation helps to explain why people in a particular population in a particular environment at a particular time look and appear different from each other.

To try to explore this extraordinarily complex issue, behavioral geneticists have long begun by taking advantage of what they often call "nature's great experiment": twins. First, behavioral geneticists posit the fact that identical twins are 100 percent genetically similar and fraternal twins are on average only 50 percent genetically similar. Second, they make the crucial assumption that the environmental conditions for identical twins in the same home are as similar as they are for fraternal twins raised in the same home. Given that fact and assumption, they infer that the extent to which identical twins appear more similar than fraternal twins with respect to some trait indicates the magnitude of genetic influence on that trait (see figure 1).

Assume that Albert and Allen are identical twins and thus are 100 percent genetically alike. They are raised by their parents in the same apartment and are subjected to the same environmental conditions: schooling, recreation, diet, and so on. Assume that down the street live Zach and Zeke, who are fraternal twins and thus about 50 percent genetically alike. Assume that in their home, Zach and Zeke are also subjected to conditions as similar as the conditions to which Albert and Allen are subjected in theirs.

Now imagine that Albert and Allen are almost identical heights, whereas Zach and Zeke are separated by two inches. If Albert and Allen were indeed exposed to the same environment and Zach and Zeke were also exposed to the same environment, and given that Albert and Allen are genetically the same while Zach and Zeke are genetically different, we have reason to believe that genetics helps to explain the observed difference in height between Zach and Zeke.

If we studied many pairs of identical and fraternal twins and found that most of the identical twins were more similar than the fraternal ones with respect to height, then we might become more confident about the role of genetic factors. We would not have learned anything about *which* genes are involved or *how* they work, however.

		Twin correlations	
Ability	*Number of studies*	*Identical twins*	*Fraternal twins*
Verbal comprehension	27	.78	.59
Verbal fluency	12	.67	.52
Reasoning	16	.74	.50
Spatial visualization	31	.64	.41
Perceptual speed	15	.70	.47
Memory	16	.52	.36

Figure 1. *Behavioral geneticists infer that if the environmental conditions for identical twins are as similar as they are for fraternal twins, then the closer average correlation of traits for identical twins is due to the genetic influence on those traits.* (Found in R. Plomin et al., *Behavioral Genetics*, fourth ed. [New York: Worth Publishers, 2001], 187.)

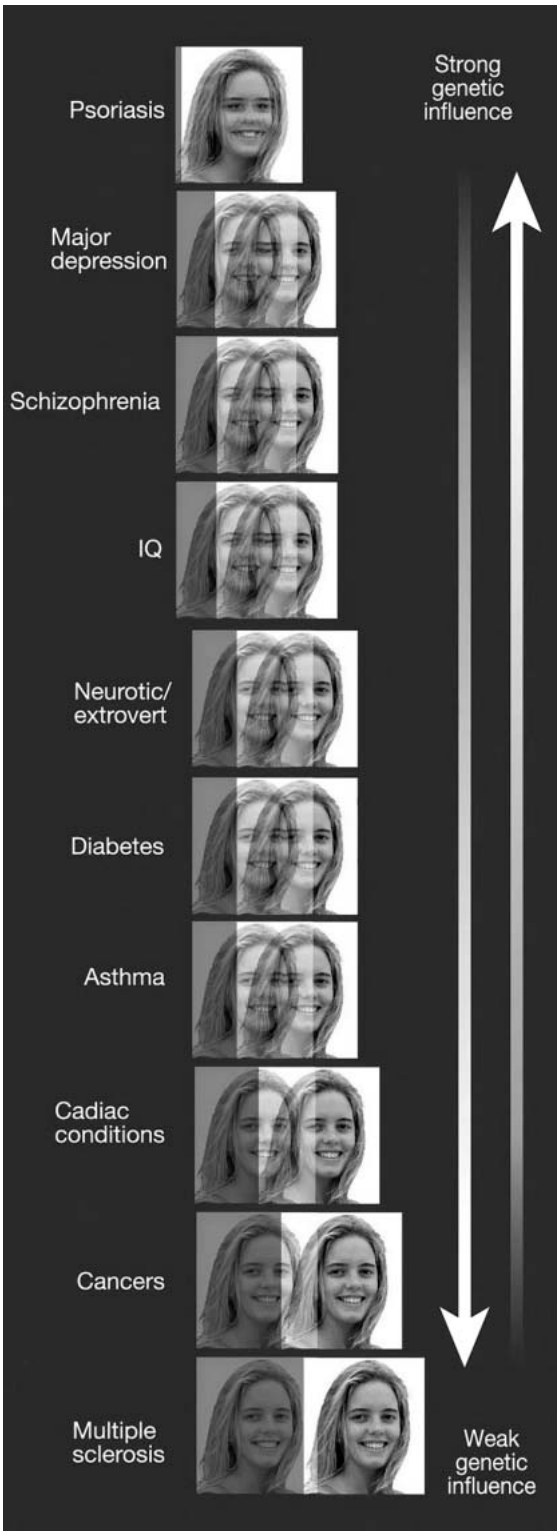

Figure 2. *Studies on identical twins suggest that some traits are much likelier than others to be shared by both twins, suggesting that genetic differences vary in their usefulness for explaining phenotypic differences.* (Reprinted by permission from A. Chakravarti and P. Little, "Nature, Nurture and Human Disease," *Nature* 421 (2003). http://www.nature.com/ Copyright (2003) Macmillan Publishers Ltd.)

Since the first twin study of intelligence in 1924, researchers have found that identical twins are more similar than fraternal twins on a wide range of traits, from height to intelligence to schizophrenia.[36] Almost since the first twin study, too, some critics have questioned the crucial assumption that the environments of identical and fraternal twin pairs are in fact equal. They suggest that the environments of identical twins raised together might be quite similar, but the environments of fraternal twins might not be. If the "equal environments assumption" is violated, then we are on much shakier ground when we infer that genetics explains why identical twins appear more similar than fraternal twins. After all, identical twins could appear more similar because their environments are more similar.

Another of several further ways of testing the hypothesis that genetics makes identical twins similar with respect to some trait is to compare identical twins reared together with those reared apart. If, for example, identical twins raised in different environments appear to be as similar with respect to some trait as identical twins raised in the same environment, then researchers infer that the similarity is due to genetic, not environmental, influences.

The twins-reared apart approach has also suggested that genetic variation helps to explain much phenotypic variation. Notice, however, that in these studies, another crucial assumption is at work, namely, that the twins are raised in importantly *different* environments. Critics question that assumption by pointing to evidence that identical twins tend to be adopted into fairly *similar* environments. The jury is still out on this dispute.[37]

What heritability estimates are and what they tell us

Again, studies that compare identical and fraternal twins raised together or apart, as well as studies that compare twins with other sibling pairs and parents, aim to discover the magnitude of the genetic influence on a particular trait. More precisely, they aim to understand the extent to which observable, phenotypic differences among individuals in a population are a function of genetic differences.

The magnitude of that influence—the extent to which phenotypic variation is thought to be a function of genetic variation—is expressed as a number called a *heritability estimate*. To say that in a given population the heritability of height is 0.9 is to say that 90 percent of the variation in height in that population can be explained by genetic variation. Heritability estimates for most complex human traits, however, are not 0.9. Even the most enthusiastic of behavioral geneticists tend to say that the heritability estimates for most complex traits are in the neighborhood of 0.4-0.6. Of course, if 40 to 60 percent of the phenotypic variation is due to genetic variation, then 60 to 40 percent is due to environmental variation.

Environmental Determinist?

Commentators who are optimistic about the explanatory power of genetic differences sometimes suggest that an "environmental determinism" dominated the study of human behavior in the twentieth century. But pure environmental determinists are hard to find. Freud is sometimes thought to have been one, but consider this passage:

> I take this opportunity of defending myself against the mistaken charge of having denied the importance of innate (constitutional) factors because I have stressed that of infantile impressions. A charge such as this arises from the restricted nature of what men look for in the field of causation: In contrast to what ordinarily holds good in the real world, people prefer to be satisfied with a single causative factor. Psychoanalysis has talked a lot about the accidental factors in aetiology and little about the constitutional ones; but that is only because it was able to contribute something fresh to the former, while, to begin with, it knew no more than was commonly known about the latter. We refuse to posit any contrast in principle between the two sets of aetiological factors; on the contrary, we assume that the two sets regularly act jointly in bringing about the observed result. Δαίμων καὶ τύχη [Endowment and Chance] determine a man's fate—rarely or never one of these powers alone. The amount of aetiological effectiveness to be attributed to each of them can only be arrived at in every individual case separately. These cases may be arranged in a series according to the varying proportion in which the two factors are present, and this series will no doubt have its extreme cases. We shall estimate the share taken by constitution or experience differently in individual cases according to the stage reached by our knowledge; and we shall retain the right to modify our judgment along with changes in our understanding.
>
> (From S. Freud, "The Dynamics of Transference," found in *Standard Edition of the Complete Psychological Works of Sigmund Freud*, vol. 12, ed. J. Strachey [London: Hogarth Press, 1958]: 98-108, at 99 fn. 2.)

Findings of relatively large heritability estimates for traits such as personality or intelligence or psychopathology can be illuminating insofar as they indicate that such traits are less "culturally determined" than arch environmentalists like J.B. Watson or B.F. Skinner would have had us believe. In fact, behavioral geneticist Eric Turkheimer has suggested that the most important contribution that behavioral genetics has made has been to demonstrate that "relations among biologically related family members cannot be unambiguously interpreted as environmental pathways."[38] Behavioral genetics shows what many of us today think is patently obvious: human behavioral differences do not result from environmental influences alone.

Steven Pinker suggests that political liberals have been snookered by "environmental determinism" and the idea of the "blank slate."[39] In fact, it is not entirely clear how many people ever thought that environment alone matters. Freud, for example, certainly never held that view (see "Environmental Determinist?"). He explicitly acknowledged that "biological endowment" would one day be extremely important in understanding many forms of psychopathology, and he said he would try to help where he thought he could: in understanding the role of what he called "accidental" (or what we would call "environmental") variables.[40]

What heritability estimates do not tell us

As behavioral geneticist Michael Rutter bluntly put it, "Knowing that a trait is genetically influenced . . . is of zero use on its own in understanding causal mechanisms."[41] (Likewise, to say that a trait is environmentally influenced is of zero use on its own for understanding causal mechanisms.) Heritability estimates do not—cannot—tell us anything about which genes are directly involved in the emergence of a given trait; they do not tell us anything about, for example, which genes play a role in which physiological pathways. And needless to say, heritability estimates cannot tell us anything about how genetic differences interact with environmental variables to produce phenotypic differences. (As Christopher Jencks pointed out long ago, a trait like skin color, which is heavily influenced by genes, may contribute to various phenotypic traits mainly by eliciting adverse social responses like racial prejudice.[42] If so, genetic differences may play a "causal" role in other phenotypic differences, such as performance on IQ tests, but not in the way that is too often assumed.)

Thus heritability estimates give us only a weak understanding of the genetic influence on phenotype: they can suggest a causal role, but they cannot tell us what it is. Of course, it does not follow from the fact that our understanding of the genetic influence is weak that the influence itself is weak: in principle, the *influence* could be strong despite the weakness of our *understanding* (a point taken up again below, in the discussion headed, "Weak versus strong senses of 'It's genetic'").

Heritability estimates also do not tell us anything about how hard or easy it will be to change a given trait. Specifically, high heritability estimates do not mean that a trait is impossible to change—and low heritability estimates do not mean that a trait will be easy to change.

The mistaken assumption that if the main source of variation is not genetic, it will be fairly easy to make environmental interventions

Some people are more committed than others to the vision of an egalitarian society, where social resources are used to reduce the gap between the haves and have-nots. If an egalitarian thinks that low heritability estimates mean that environmental variables are more important than genetic variables for explaining differing social outcomes, and if she thinks that environmental interventions will reduce those differences, then she might think that a low heritability estimate means that the chances of using environmental interventions to reduce the gap are high.

Unfortunately, it is not true that traits heavily influenced by the environment are easy to change. We know that social and economic deprivation has deleterious effects on many behaviors,[43] yet our society has had little success in changing those influences. Indeed, we have long known that childhood deprivation greatly increases the likelihood that someone will be antisocial as an adult, but we have had enormous difficulty intervening in those environmental variables.[44]

The mistaken assumption that if the primary source of variation is genetic, environmental interventions will be useless

Some people, call them *libertarians*, are not distressed by the unequal distribution of social resources. On this libertarian view, unequal distribution reflects unequally distributed natural gifts, and while the unequal distribution of gifts may in some sense be unfortunate, it is not unfair. Moreover, the libertarians believe that even if more equally distributed resources were a laudable ideal, the gap cannot be closed with environmental interventions. This variety of libertarian assumes that if a trait is highly heritable, then environmental interventions will be of little use.

But again, it is simply not true that if a trait is highly heritable, then it will be impossible to change environmentally. Eyeglasses are the classic example of an environmental intervention that helps to ameliorate highly heritable vision problems. Another classic example is phenylketonuria, a disease that is genetic in the straightforward sense that whether one one gets it depends on whether that person has mutations of both alleles at a single locus. The most effective response to this disease is *environmental*: a strict diet, reducing the essential amino acid that a person with phenylketonuria cannot process, prevents the outcomes associated with the disease.[45]

The mistaken assumption that heritability estimates tell us something about why groups are different with respect to some trait

It is a logical mistake to assume that high heritability estimates mean environmental interventions will not work or that low estimates mean they should be easy. It would be a political and ethical mistake to think that noticing the logical mistake will alone deprive heritability estimates of their potential for mischief.

Before we talk about people, let's talk about cows. Imagine that there is a population of white cows in Montana and a population of black cows in Vermont. Imagine that several studies have shown that the heritability of the capacity to convert grass to milk is high. Let's say it's 0.8. And imagine there's a test to measure how efficiently cows convert grass to milk. Finally, imagine that the cows in Montana on average do better than the cows in Vermont on the grass-to-milk conversion test. Given the high heritability estimate and the phenotypic difference (the different scores on the test), can we infer that the cows in Montana are "genetically superior" at converting grass to milk?

Absolutely not, but the reason why is not intuitive. The critical point is that heritability estimates are informative only about a specific population in a specific environment. Maybe the following example can help.

A heritability estimate (which we will call H) is meant to convey how much of the phenotypic variation (Pv) with respect to some trait is due to genetic variation (Gv)—as in, How much of the variation in milk production from cow to cow is associated with genetic variation? We can express the heritability idea as a simple formula: H = Gv/Pv. If we assume that phenotypic variation is the result of genetic variation (Gv) plus environmental variation (Ev), then we can express that same idea as:

$$H = \frac{Gv}{(Gv + Ev)}$$

Imagine we've got a single bag of corn seed. We sow a handful in a good environment, where the plants will receive lots of food and water and light. We keep the conditions of this environment uniform; that is, we give all of the plants precisely the same good environmental conditions. So what's the heritability estimate for the height of the corn in this population? Since there is no environmental variation in this experiment, Ev = 0. Thus in this case, the formula H = Gv/(Gv + Ev) can be simplified to H = Gv/Gv. Since Gv/Gv is 1, the heritability of height is 1.0, or 100 percent. By stipulating that there is no environmental variation, we know that 100 percent of the phenotypic variation is due to genetic variation. (See figure 3.)

Now imagine that we take another handful of seed from the same bag. But this time we plant the seed in a bad environment, where all the seeds only occasionally receive food and water and light. Although the environmental conditions here are deficient, they are once again uniform. Because the environmental variation in this scenario is once again 0, the heritability of height in the second population is also 100 percent.

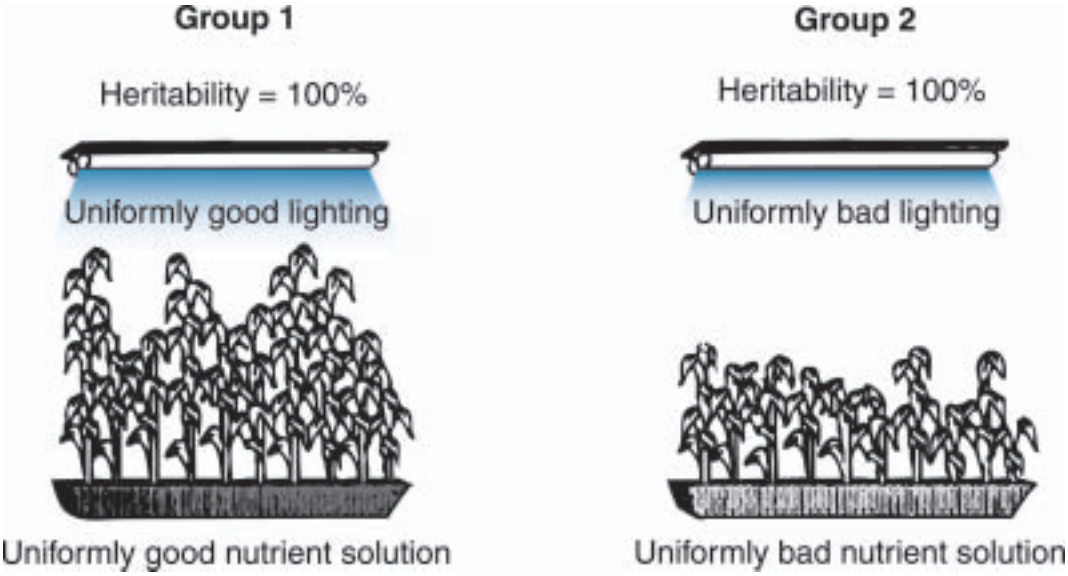

Figure 3. *Heritability estimates and group differences. The heritability of height can be 100 percent in both groups and be of no help in explaining why the groups are phenotypically different.* (Drawing by Nora Porter, based on a figure by Richard Lewontin found in N. Block, "How Heritability Misleads About Race," *Cognition: International Journal of Cognitive Science* 56, no. 2 [1995]: 99-28.)

So we have two populations of the same corn seed in two different environments. The average height in the first population is superior to the average height in the second population, and the heritability of height in both populations is 100 percent. Could we infer from the high heritability of height and the average phenotypic difference that population one is genetically superior to population two with respect to height? Of course not. The heritability estimates were reached with a uniform environment within each study, but across the two studies, the environments were quite different. Thus even though the heritability estimates are 100 percent within each study—limited as they are to a given population in a given environment—there is good reason to suspect that genes do not alone explain the studies' two different outcomes.

Nonetheless, people in our public conversations about the genetic influence on this or that trait succumb to the temptation to leap from observations that a trait is highly heritable and that one population is on average superior to another with respect to that trait—to the inference that the first population is "genetically superior" to the second.

I was tempted to remain silent in this report about whether genetics has taught us anything to explain the widely asserted fact that, on average, African Americans do not score as well as white Americans on IQ tests. (Such a statistic of course says absolutely nothing about any given black or white person; some blacks are at the top of the distribution and some whites at the bottom.) I was tempted to remain silent because, after all, arguments will not persuade racists; racism is not about logic. But I choose to speak to the question because I think many people who fancy themselves enemies of racism, both black and white, ask it. And I think they—you and I—need to understand just how little is understood about genetics, race, and intelligence. Leaving aside for now the important and complicated debates regarding what the terms *race* and *intelligence* mean,[46] I want to emphasize how little is known about how much genetics can help to explain the putative black-white gap. (In the section on molecular methods, we will see that not a single gene that helps to explain phenotypic differences with respect to "intelligence" has yet conclusively been identified—not one.[47])

In fact, no one has conclusively demonstrated that the phenotypic difference on test results is *not* the result of genetic differences. As the social scientists say, proving the null hypothesis is impossible. As the rest of us might say, you can't prove a negative. And it is equally true that no one has conclusively demonstrated that the phenotypic difference *is* the result of genetic differences.[48] But if researchers have not discovered an explanation for the alleged phenotypic difference, then why do so many people seem to arrive at the genetic explanation?

There is only one logical way to get from the known facts of the matter to the genetic explanation. That is to supply an additional premise: that the environments of populations being compared are largely equal. And people who believe or insinuate that whites are genetically superior to blacks with respect to intelligence supply that premise.

Here's a summary of how their line of thought works. First they point to high heritability estimates for intelligence (Hernstein and Murray settle on 0.6). Second, they point to the fact that on average, whites score better than blacks on IQ tests. Steps 1 and 2 are clear-cut; it is the

third step that is never clearly articulated. Although they hardly ever come out and say that the environments of whites and blacks are equal, they insinuate that they are. They say that while racism "is still a factor in American life,"[49] after "more than a generation of preferential social policies,"[50] it is a mistake to think of racism as a relevant variable in trying to explain the putative gap.

If one believes that "more than a generation of preferential social policies" have made the environments of white and black Americans largely equal, then she is compelled by intellectual honesty to consider, as do Jensen, Herrnstein and Murray, and others, that genetics explains the phenotypic difference. Indeed, one of the most surprising things I learned in the course of this project is that there are very highly educated, well-intentioned people who believe that the environments of African Americans and whites are for all intents and purposes equal. They really do *not* seem to want the facts to be that one group is "genetically superior" to the other. But because they believe that the environments are pretty much equal at this point in our country's history, they feel driven by intellectual honesty to adopt the genetic hypothesis.

Some very small (and old) studies have attempted to eliminate "environmental" differences by considering only blacks and whites raised in similar socio-economic circumstances. The results are mixed, some suggesting that the black-white test-score difference persists and some suggesting that it disappears.[51] Those studies (by friends and foes of the genetic explanation for the gap) assume, however, that if we compare blacks and whites from the same "class," the environmental factors that might distort the comparison will be eliminated. That assumption could be true only if racism no longer has effects. And there are strong reasons to reject that assumption.

Claude Steele and Joshua Aronson conducted an experiment which found that when African-American students at Stanford were asked to take a difficult verbal test that they were told was diagnostic of cognitive ability, on average they performed worse than their white counterparts. But when another group of African-American students at Stanford was asked to take the same test, but were told that it was *not* diagnostic of cognitive ability, they did as well as their white counterparts. This experimental result lead Steele and Aronson to hypothesize that when a negative stereotype "demeans something as important as intellectual ability, this threat can be disruptive enough . . . to impair intellectual performance."[52]

New, not-yet replicated evidence suggests that racism does not only take a toll on African-Americans. An experiment by researchers at Dartmouth suggests that racism can create short-term impairments in the intellectual performance of whites. It turns out that well-intentioned white students at Dartmouth are so unaccustomed to black students, or so conflicted about them, that if they take an intelligence test immediately after an encounter with a black student, their performance is worse than if they had no such encounter.[53]

Indeed much empirical evidence suggests that the environments of whites and blacks are not equal.[54] If inspection of your own heart does not reveal residual racism (whether you are white, black, or green), then consider some more facts. Newborn black babies die at more than twice the rate of newborn white babies in the United States. The same is true of blacks in their forties and fifties. The ratio is not quite as bad at other ages, but whites do not start dying at greater rates than blacks until their late 80s.[55] It is not theoretically impossible that genetic differences explain the different mortality rates, but there is good evidence that environmental differences—the many facets of racism[56]—give more explanatory bang for the buck. For one thing, there is empirical evidence to suggest that physicians, who are presumably among the best educated and best intentioned among us, treat white patients more aggressively than they treat black patients.[57] Mountains of evidence suggest that, on average, blacks are subjected to greater environmental risks (lead, carcinogens, and so on) than whites.[58]

Troy Duster suggests another set of facts that argue against the assumption that the environments of blacks and whites are largely equal. These have to do with the different and changing rates at which blacks and whites are incarcerated.[59] In 1933, blacks were incarcerated at twice the rate of whites in the United States. Even though the most enthusiastic proponents of the genetic hypothesis grant that racism was rampant in 1933 in the United States, it is nonetheless theoretically possible that genetics could explain the different rates at which blacks and whites were incarcerated then. It turns out, however, that in 1995, blacks were incarcerated at *eight* times the rate of whites. No matter how committed one is to genetic explanations for such phenomena, there is simply no way to use genetics to explain the *change* in the rate of incarceration. Massive genetic changes do not occur over a few generations. The only way to explain such a rapid change is to consider changes in the environment. Of course, one would be a fool to claim to fully understand the phenomenon. But so too would one be a fool not to recognize that, even after a generation of civil rights legislation, the environments of whites and blacks are not equal enough to permit the leap from a high heritability estimate and a between-group phenotypic difference to a genetic explanation for that difference.

So neither in the case of IQ nor of any other complex trait do heritability estimates help us to better understand why two groups are different. And, it bears repeating, neither do heritability estimates tell us anything about how easy or hard it will be to change a behavior, nor do they tell us anything about *how* genes are involved in the trait of interest, nor even about *which* genes are involved.

To find out *which* genes are involved in a trait of interest, researchers today increasingly turn to what they call

"molecular studies." Molecular studies aim to discover which and how genes influence complex human traits. But before I turn to the molecular studies, I want to briefly describe an arena of research that uses non-human animals to try to understand human behavior. Like the classical studies of humans, which use twins and other family members, some "animal studies" aim at understanding *how much* genes influence a given behavior. And like the molecular studies of humans, some animal studies aim at discovering *which* genes and *how* genes are involved in a given behavior. (The reader in a hurry can proceed to the section on molecular studies in humans.)

Animal Studies: Human Genetics by Analogy

From a behavioral genetics research point of view, one of the big problems with studying human beings is that it is often difficult or impossible to do experiments on them with the kinds of controls behavioral geneticists prefer. One cannot, for example, force human beings to mate over several generations to produce a genetically similar strain of humans and then compare that group to a control group that was not similarly selected. And even if it were ethically possible to undertake such a breeding program, it would not be possible to keep the environments of those humans constant (as would be required by an attempt to get a precise sense of how helpful genes are for explaining phenotypic differences).[60] Nor is it ethically possible to directly investigate the effects of single genes by inserting them into or deleting them from human gametes or embryos. As scientifically informative as results of such experiments would be, they would violate every internationally recognized principle of human subjects research.

Fortunately for all geneticists, however, it is widely viewed as ethically acceptable to breed non-human animals (like flies and worms and mice) and use them in experiments that insert genes into or "knock" them out of gametes or embryos. Studying non-human animals can shed light on the genetics of human behaviors because evolution conserves genes. That is, because all animals evolved from a shared common ancestor, all animals—even fruit flies and worms—share many genes with humans. As Lisa Brooks has put it, "99 percent of our genes are also in mice."[61] Not only are genes widely shared, but there is also considerable overlap in the ordering of those genes in different species. Moreover, the sequences (of base pairs) of those genes are often highly similar. The functions of those genes, too, are thought to be highly similar (see the crucially important caveats below, however).

Learning how much influence genetic differences have on animal behavior

Like twin, adoption, and family studies in humans, non-human animal studies are often used to understand whether and, in most cases, how much genes influence a given trait or behavior. As Robert Plomin and his colleagues state in their basic text on behavioral genetics, "Laboratory experiments that select for behavior provide the clearest evidence for genetic influence on behavior."[62] That it is possible to use breeding techniques to select for mouse traits should come as no surprise to anyone who has observed that different behaviors have been bred into domesticated animals such as dogs. Hunters do not usually take Pekinese out on the trail and parents do not usually let pit bulls romp with their children.

In a classic study, mice were initially selected based upon how active or inactive they were when placed in a brightly lit box. When placed in such a box, some mice actively explored it, while others froze, defecated, or urinated.[63] Researchers selected the most "high-activity" mice and mated them, producing a new generation. Researchers then selected the highest activity mice from that generation and mated them. They did this for thirty generations. They did the same with the "low-activity" mice. Over time, the researchers discovered, the line of active mice became more active and the line of inactive mice became more inactive. As Plomin and colleagues succinctly put it, "Successful selection can occur only if heredity is important."[64] That is, if the genes of the animals selected at each generation are changing, while the environmental conditions remain constant, it stands to reason that genes importantly help to explain the phenotypic changes.

With such a study, researchers can demonstrate that an animal's genotype is significantly correlated with its activity level (or "phenotype"). These animal studies are like twin, adoption, and family studies insofar as they suggest that, and perhaps to what degree, genetic variation influences phenotypic variation with respect to the trait of interest—at least for those populations in that environment at that time. Also like the twin, adoption, and family studies of humans, the animal studies do not teach us anything about which genes are involved in that trait.

Learning which *genetic differences influence animal behavior*

Just as researchers sometimes use "molecular methods" to directly study which genes influence complex human behaviors, researchers sometimes use molecular methods to study which genes influence complex non-human animal behaviors. For example, if a given gene seems to play an important role in a physiological pathway leading to a behavior or trait in a mouse, and if mice and humans share that gene, then there is reason to believe that "the same" gene may play the "same role" in humans. Many of the most widely publicized results on the genetics of behavior have been based on mouse studies, including studies on "intelligence," "novelty seeking," "aggression," "hyperactivity," "addiction," "social interaction," and "depression and neuroticism."[65]

Perhaps the most famous mice produced by researchers investigating the genetics of behavior were those named

"Doogie," after the precocious star of the television show, "Doogie Howser, MD." By adding a single gene to mouse embryos, researchers at Princeton created mice that had enhanced capacities to recognize objects and remember previous experiences. That is, when compared with mice that lacked the genetic addition, the Doogie mice proved to have better memories and to be better learners.

The cover of the September 13, 1999 issue of *Time* magazine invitingly asked, "IQ Gene?" To create further excitement, the authors of the Doogie paper asserted in the final sentence that their experiment provided "a promising strategy for the creation of other genetically modified mammals with enhanced intelligence and memory."[66] Not only is it not clear what effect the same intervention would have in humans, but it appears that this attempt at the enhancement of the mice had the unforeseen and unintended effect of making the Doogie mice more susceptible to pain than unmodified mice.[67]

Though mice and humans may have "the same" gene at a given location, there are reasons to be extremely cautious about making inferences from how the gene appears to affect mouse behavior to how it will affect human behavior. The same gene may well have different functions in different species.[68] Also, the same gene may be expressed at different developmental stages in different species.[69] And since imperceptible environmental differences can affect whether or not a gene is expressed in members of the same species, small environmental differences will likely affect whether a gene is expressed in different species—thus making extrapolation from one species to the next difficult.

It is true that if researchers knock out (or "knock in") a gene in a mouse and then consistently observe how mice without that gene are different from mice with it, then one might reasonably infer that the absence or presence of that gene is in some way related to the behavior. For example, if one thinks that a given gene codes for an enzyme, which is involved in the regulation of a neurotransmitter that is involved in "aggressive behavior," and if one observes that knocking out that gene affects how aggressive the mice are (where "aggression" is measured by how quickly and often mice react to intruders on their territory), then one has support for one's hypothesis. However, such an experiment is a blunt instrument. From it one learns nothing about how the removal of that gene from the complex organism produces the observed change.

Again, discoveries regarding how particular genes operate in animals may provide tantalizing clues about how they operate in humans. But researchers interested in the genetics of human behavior will be satisfied only when they can both identify which genes are implicated in a particular human behavior and say *how* those genes affect the physiological pathways that ultimately influence phenotypes. If researchers are going to answer those questions, they will have to study humans and use "molecular" approaches.

Molecular Studies: Toward Which Genetic Differences Matter

Before introducing two basic strategies of molecular genetics, I want to try to make some distinctions. One of my aims is to show what is needed to develop what I'll call a *strong* understanding of the role of genes in the emergence of complex traits. A second aim is to impress upon you that researchers are very far from having this kind of understanding, and that there are reasons to believe that some enthusiasts may have significantly overstated how large a contribution the science of genetics will be able to make to such understanding. Some critics, of course, may have overstated how small the contribution will be.

Weak versus strong senses of "It's genetic"

It is often asserted that this or that trait "is genetic," as in, "her love of skydiving is genetic" or "his depression is genetic." In the weak sense that genes are a precondition for her skydiving or his being depressed, it must be true that "it's genetic." She could not dive out of airplanes were it not for her genes functioning just as they were, nor could he have experienced his depression without his genes functioning exactly as they did. In that weak sense, all behavior surely "is genetic." But to say that a behavior is genetic in that weak sense is banal.[70]

Presumably, when people say that a trait is genetic, they mean it in the strong sense that genetic differences help to explain why some people like to skydive, or why some people are prone to depression. The twin and adoption studies of the classical behavioral genetic methods aim at determining whether a given trait "is genetic" in that strong sense.

Another common, shorthand way of saying that a trait "is genetic" in the strong sense is to say that the genetic influence on the trait is large. This means that genetic differences are thought to go a long way toward explaining why different people express the same trait to different degrees or why, in some cases, some people exhibit a trait and others seem to lack it altogether. Such was the implication of the Heston study on schizophrenia, which found that if we want to understand why some people exhibit schizophrenia, then we should investigate genetic variation at least as much as we study environmental variation.

Once the classical methods (using twins and adoption studies) suggest that the genetic influence on a trait is large, researchers then have reason to embark upon molecular studies, which aim at understanding which genes and ultimately how genes influence the trait. If researchers could describe how particular gene variants affect pathways that influence phenotypes, then it would be possible to say that they have established a strong *understanding* of the genetic influence on a trait. As we will see, because large genetic influences on complex traits can result from the accumulation of the small effects of many genes interacting with many environmental factors over time, it will

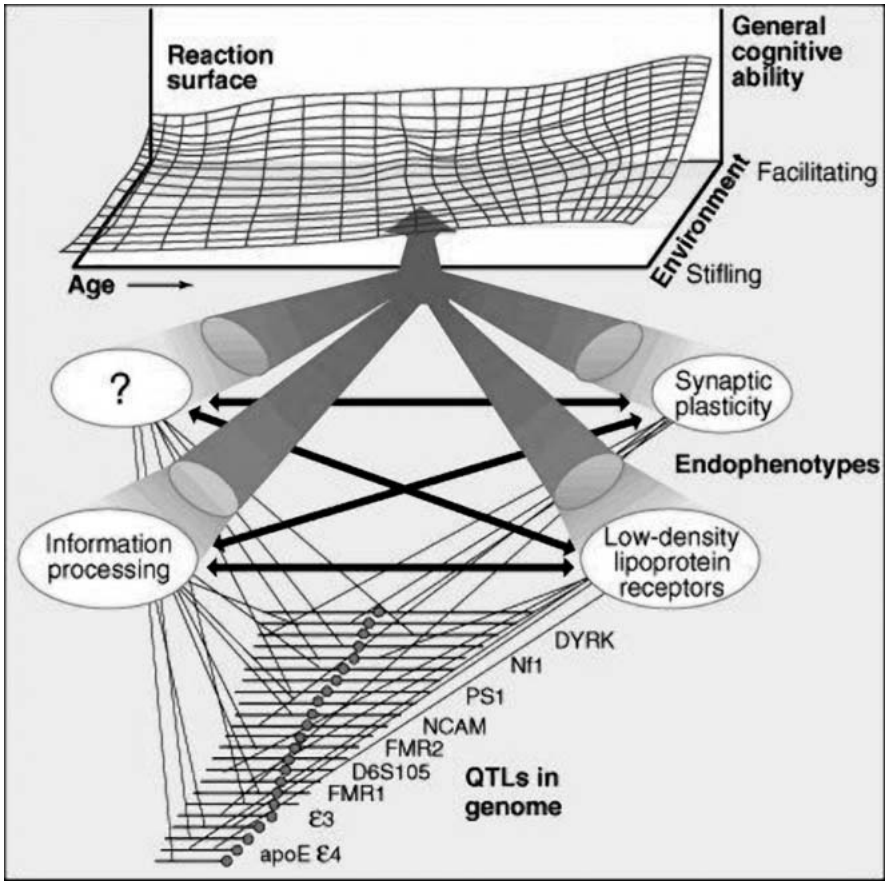

Figure 4. *Some behavioral geneticists think it helps to conceive of continuously distributed (or quantitative) traits like IQ as being constituted by "endophenotypes," each of which is influenced by specific genes (called "quantitative trait loci" or QTLs). Where an individual falls within the range of ability that her genes and experience make possible will also depend on how her genes and experiences interact over time.*
(Reprinted with permission from I.I. Gottesman, "Twins: En Route to QTLs for Cognition," *Science* 276 [1997]: 1522-23. ©1997 AAAS. http://www.sciencemag.org Adapted from C.F. Sing et al, "Traversing the biological complexity in the hierarchy between genome and CAD endpoints in the population at large," *Clinical Genetics*, 46, no. 6 [1994]: 8. http://www.blackwell-synergy.com)

be very difficult to achieve strong understandings of the genetic influence on complex traits. That is, it will be difficult to give full accounts of how individual genes affect specific pathways that have specific and predictable effects on specific traits.

Nonetheless, some researchers believe that we are close to a strong understanding of at least part of the genetic influence on schizophrenia.[71] Please notice: even if researchers achieve a very strong understanding of the role of genes in the emergence of a given trait, genetics alone will never fully explain why a given individual exhibits a complex trait like schizophrenia. *Even so-called "strong" understandings of genetic influences will be only partial*–insofar as they will always only be part of a far more complicated story, which will involve interactions with environmental variables over time.

Consider: for members of the population at large, the risk of developing schizophrenia is about 1 percent. If one identical twin has schizophrenia, however, then the other twin has an approximately 45 percent risk of developing the disease.[72] It certainly seems that genetics plays an important role in the story, and it is hoped that developing a strong understanding of that role will help to develop improved therapeutic responses to schizophrenia. But that 45 percent figure means that the second twin will usually *not* develop schizophrenia.[73] Even though genes seem to play an important role, they do not act alone. Remembering this point is hard. For reasons that may be linguistic and/or psychological, it seems difficult to give genes their due—but not more than their due.

Simple versus complex patterns of inheritance

Huntington's disease is the classic example of a trait that results from a single allele being passed from one generation to the next in the simple manner described by Mendel: the chance of getting one allele associated with the trait is approximately 50 percent, and if a person has the allele, she has a nearly 100 percent chance of developing some form of the disease.

It might not be an exaggeration to say that the isolation of "the gene for" Huntington's was both a triumph for medical genetics and a disaster for public conversation about behavioral genetics. As exciting and important as it was to learn that a single gene was the necessary condition for the presence of Huntington's, the one gene → one trait model is useless for understanding the typical relationship between genes and traits. As we have already seen and will see again, an adequate understanding of complex human traits will entail examining the exceedingly complex relationships among genes and environmental factors, which are mediated in the brain and change during development.

So most complex traits do not in any sense result from single genes being transmitted from one generation to the next in the simple manner described by Mendel. Most traits are not either/or in the way that one either has or does not have Huntington's. Instead, most traits are more like height, which varies continuously within a population. The height one attains is the result of a highly complex process that involves the transmission of many alleles, interacting with each other and the environment in highly complex ways—and in different ways, depending on where the organism is in its development. So even though

most traits "aggregate" in families, as Mendel would have predicted (schizophrenia is much more common in some families than others), they do not result from single alleles segregating in the simple manner he postulated (nothing like 50 percent of the offspring of people with schizophrenia exhibit schizophrenia).[74]

Complex relationships between mutations and phenotypes

Although in a few rare cases a complex trait like Huntington's may be transmitted in the simple pattern predicted by Mendel, the relationship between that mutation and the phenotype rarely is simple. That is, whether one has some complex trait may in a few rare cases depend exclusively on whether one carries a single mutation, but the particular form the trait takes will still usually depend on other, independently inherited genetic variations and environmental influences. So even if the pattern by which these rare traits are inherited is simple, the relationship between the genotype and phenotype is not. Or, as Katrina Kipple and Edward McCabe put it, "For [many] 'simple' Mendelian disorders, the phenotypes are, in fact, complex traits."[75]

Indeed, whether the genetic influence on a trait results from a simple or complex mode of transmission, the relationship between the genotype and phenotype will almost always be complex. If you get the gene associated with Huntington's you'll get the disorder, but what form that disorder takes will depend upon the other genes you inherit and the environment you inhabit.

Large versus small genetic effects

I just mentioned that in some rare cases, single genes can have very large effects; if one carries the mutation associated with Huntington's, then that single mutation will have wide-ranging effects (though as we just noticed, the particular form will depend on other genetic and environmental variables). In most cases, however, differences in single genes will have small effects. For example, recent work suggests that an allele for a receptor for the neurotransmitter dopamine accounts for only 4 percent of the variance between individuals who exhibit different degrees of novelty-seeking behavior.[76] In *Behavioral Genetics in the Postgenomic Era*, Plomin and colleagues suggest that researchers will have to start to identify alleles that account for as little as 1 percent of the phenotypic variance with respect to some trait if they are ultimately to contribute to understanding complex disorders and behaviors.[77]

Because complex phenotypes or behaviors seem to be affected by so many genes of such small effect, behavioral geneticists are turning to what we might call the building blocks of the phenotype, or what some behavioral geneticists call *endophenotypes*.[78] Rather than study the genetics of "intelligence" directly, for example, they study what they believe to be its component parts. So, for example, they might study the building block or endophenotype that is "information processing" or "synaptic plasticity" or even "lipoprotein metabolism."[79] None of those building blocks is itself intelligence, but each is thought to be an essential constituent of the complex chain between the genome and the phenotypes indicating "intelligence." Over time, a large number of endophenotypes will interact with a large number of environmental variables to produce the phenotype of intelligence. Figure 4 illustrates these interactions, although both the number of endophenotypes and number of alleles contributing to those endophenotypes may be much larger than the diagram suggests. Still, behavioral geneticists think that strong understandings of the small effects of many individual genes on these endophenotypes will eventually add up to a stronger understanding of the genetic influence on complex human phenotypes.

What science writer Catherine Baker calls "Gottesman's trampoline,"[80] referred to in his figure as the "reaction surface," is one way to visually represent the simple but fundamental idea that an organism's phenotype will be a function of its genes, its environment, and its age. If the diagram were still more complete (and thus probably too complex to read), the arrows would not just point "up" from genes but also "down" from environmental variables. As Matt Ridley and others have argued, nature always works via nurture: genes are turned on and off in response to environmental triggers.[81]

In almost all cases, the size of the genetic effects will be context-dependent; that is, the same gene (the same sequence of base pairs) will have different effects in different organisms. The same gene also will have different effects in the same organism at different times and in different environments. And in almost all cases, the size of the environmental effect will be "organism dependent," as we might say; that is, the same environmental variable will have different effects on different organisms. The classic diagram in figure 5 shows cuttings from the same yarrow plant (possessing the same genetic stock) growing to different heights at different elevations along a mountain; the cutting that grows tallest at both the highest and lowest altitudes is shortest at the medium elevation. The same variables also will have different effects at different times in the life of the same organism; not drinking milk can have serious consequences for a boy, but probably few for a middle-aged man.

Two basic strategies of the molecular approach

In spite of the extraordinary complexity of the relationships among genes, environments, and time, molecular geneticists have been attempting for the last fifteen years or so to identify which genes are associated with particular traits. To identify which genes are involved, they use a couple of basic strategies (sometimes in the same study). Researchers refer to those different strategies as *linkage* studies and *association* studies.[82]

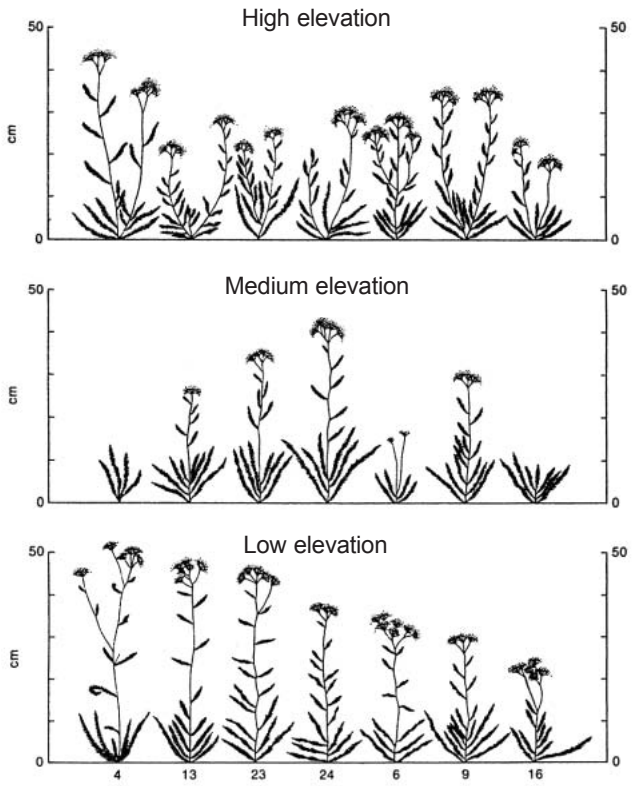

Figure 5. *Plants with the same genotype (from the same parent plant) grow to different heights in different environments (different elevations on the same mountain).* (Reproduced by permission from A. Griffiths et al., *An Introduction to Genetic Analysis* [New York: W.H. Freeman and Company, 1993]. ©1993 by W.H. Freeman and Company/Worth Publishers.)

Insofar as both linkage and association studies aim to establish "links" or "associations" between a gene and a trait, however, the technical labels are not very helpful in keeping straight the difference between the approaches. For my purposes in this report, it will suffice to notice that the older approach ("linkage analysis") begins from the level of behavior and tries to go "down" to the level of specific genes associated with the behavior. The basic idea is that researchers take a family with a history of a particular disease and study DNA samples from successive generations to determine whether particular gene variants show up in most persons with the disease. Results from different families are then pooled and compared. Traditionally, when researchers use such a "top-down" approach, they do not have in mind "candidate genes." That is, these studies have not proceeded from an assumption about the role of a particular gene in a particular behavior.[83]

One simple problem with the "top-down" approach is that finding a sufficiently large number of families with the trait of interest can be costly. Another problem that used to plague these studies (as well as those using the bottom-up approach described below) involves the definition of the trait being studied. If, for example, different researchers use different definitions for what they call the same trait, or if a researcher mistakenly calls different behaviors by the same name, study results will be confused. Researchers have paid considerable attention to these definitional problems, however, and they are increasingly confident that when two different researchers say they are studying "novelty seeking," they are talking about the same thing. I should say, however, that not all commentators are equally confident of this; Nancy Press has pursued a rich exploration of the problems associated with the identification of traits for genetic investigation.[84]

Another problem is that, for technical reasons, the "top-down" approach can identify only those genes that have large effects, while most common diseases and most quantitative traits result from the small contributions of many genes, many environmental factors, and development. In other words, most complex behaviors involve genes of small effects, but most linkage strategies are bad at identifying such genes. Some hope that more powerful analytic techniques will make that problem surmountable.[85] Time will tell.

The newer strategies, called *association studies*, also compare the DNA of individuals to see if genetic differences can be correlated with phenotypic differences. But whereas linkage studies require successive generations of families to study, association studies do not. And whereas linkage studies begin from the behavior of interest and work their way "down" to try to find genes implicated in that behavior, association studies begin with a gene that is suspected to be associated with the behavior of interest and work their way "up" to the behavior. In association studies, researchers start with a gene they have reason to believe may be implicated in the trait of interest and see if they can establish an association between that "candidate" and the behavior of interest. In these bottom-up approaches, the researchers usually identify a group of research subjects who manifest the behavior and a control group in which it is lacking. If a genetic variant is more common in people who have the behavior, the variant might have a causal relationship to the behavior.[86]

One of the strengths of association studies is that, unlike linkage studies, they can identify genes of small effect. Given the fantastic complexity of human physiology, however, the number of possible candidate genes for most traits will be huge—and so will be the task of finding correlations that will turn out to be causally significant and thus ultimately of practical use in understanding complex human behaviors.

One difficulty with association studies is that the correlations they find between a gene variant and a behavior do not always reveal a causal relationship. (Association studies are not alone in this respect; detecting correlations without causation is an ongoing problem in all sciences.) Eric Lan-

der and Nicholas Schork invite us to imagine the following "light hearted" scenario.[87] You are a researcher in San Francisco and you're interested in discovering what gene variants are associated with the trait of "proficient chopsticks use." Imagine that you have reason to believe that the HLA-A1 allele is involved not only in immune response, but also ultimately in manual dexterity. In your population of proficient chopsticks users, you have many "Asians," and in your "control" population of not-so-proficient users, you have many "Caucasians." If you chose as your candidate gene variant one that just happens to be more common among Asians than Caucasians (such as the HLA-A1 allele), you would indeed find a correlation between that gene variant and chopsticks proficiency, but you would make a rather big mistake to infer that the gene variant *caused* the proficient use of chopsticks. Researchers are increasingly clever about guarding against such false positives, however.[88]

Recently, researchers have begun to move from merely making inferences about causal relationships to actually studying when and how much a given gene variant produces messenger RNA.[89] Without going into any of the details, this new technology, called microarray analysis, enables researchers to begin to probe *how* genes affect complex traits. It does that by essentially taking real-time "pictures" of when in development gene variants are turned on and off, which gene products are produced at a given time, and how much product is produced.

If classical behavioral genetics is a first step, in which researchers try to establish *that* genetic variation helps to explain phenotypic variation, and linkage and association studies are a second step, in which researchers begin to identify *which* genes vary with respect to some trait, then microarray analysis seems to be a third step. Here, researchers can begin to understand *how* individual genes help to explain phenotypic variation.

Simple versus complex models of explanation

In light of all this complexity, it might be surprising to learn that, until fairly recently, many behavioral geneticists proceeded from the view that there would be a fairly simple and "direct linear relationship between individual genes and behaviors."[90] Not all, of course: some mainstream behavioral geneticists have long rejected the one gene → one trait model, and Fuller and Thompson's seminal 1960 text never advocated it.[91] Yet some prominent behavioral geneticists have continued to suggest that single genes would often turn out to have large effects on complex behaviors. Dean Hamer, for example, has sometimes written sentences like, "A gene that makes you anxious and sexually active is more likely to survive than a gene that doesn't."[92] To his credit, Hamer published a short but important statement in *Science* in the fall of 2002 that rejects the simple model.[93] In place of the simple model, Hamer describes a complex set of interactions, where "gene networks and multiple environmental factors impact brain development and function, which influence behavior."[94] On this view (illustrated in figure 6), any strong account of the genetic influence on behavior will be highly complex. It will investigate how large numbers of genes and environmental factors interact over time, and how the behaviors that result from such interactions

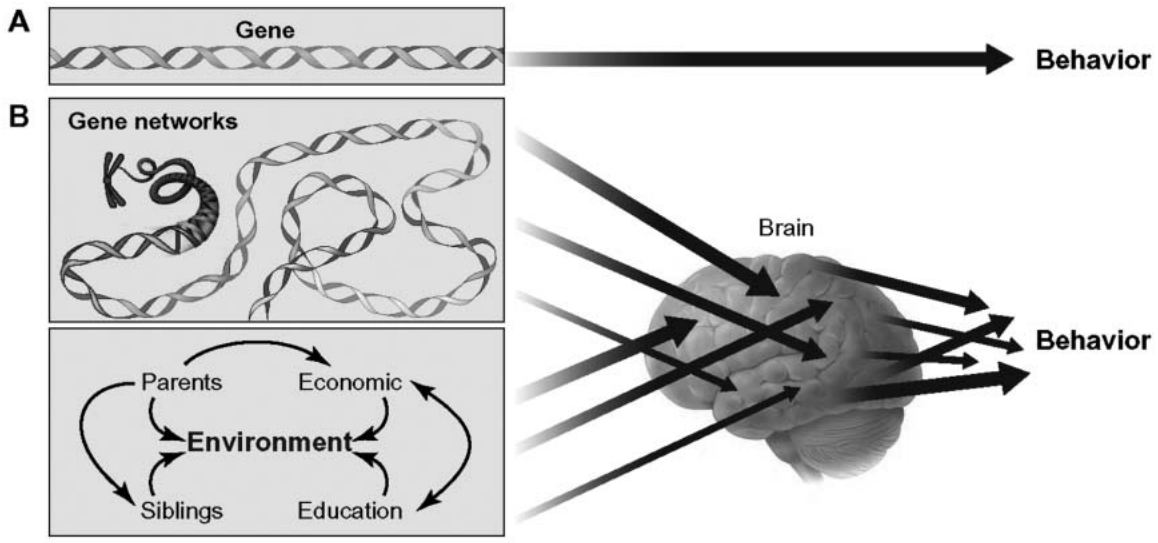

Figure 6. *Two views of behavior genetics: Part A represents the old, wholly inadequate one gene → one behavior model. Part B begins to represent the multiple genetic and environmental variables and their staggeringly complex interactions, which are mediated in the brain and change over time.* (Reprinted with permission from D. Hamer, "Rethinking Behavior Genetics," *Science* 298 [2002]: 71-72. Illustration by Katharine Sutliff. ©2002 AAAS. http://www.sciencemag.org)

themselves influence those genetic and environmental factors.

Hamer's statement, "Rethinking Behavioral Genetics," is unique both in its frankness and in the visibility of the venue where he chose to publish it. He not only acknowledges that the one gene → one behavior model is mistaken, but he acknowledges how exceedingly modest the results of the standard molecular approaches have been. He does not mince words:

> The results [of linkage studies] have been disappointing and inconsistent. Large and well-funded linkage studies of the major psychiatric disorders including schizophrenia, alcoholism, Tourette syndrome, and bipolar disorder have come up empty-handed; not a single new gene has been conclusively identified. Most [association study] findings have failed consistent replication, and even those that have been verified account for only a small fraction of the total variation.[95]

Other researchers have offered similarly frank assessments, including Janine Altmuller and colleagues,[96] Michael J. Owen and colleagues,[97] and Robert Plomin.[98]

Many linkage (or "top-down") studies of disorders like schizophrenia and bipolar disorder initially announced findings that looked significant, and those findings were widely reported in the press as "gene for" discoveries. But when researchers attempted to replicate those findings, they usually could not. One current exception to this rule is the replication of studies finding linkage between an allele on chromosome 6 and reading disability.[99]

Part of the problem is that reports of positive findings often end up on the front page of newspapers, while reports of negative findings get scant attention. The journalists seem, fortunately, to be increasingly skeptical about unreplicated findings of "genes for" complex traits, but it's too bad Hamer's "Rethinking Behavioral Genetics" essay could not become front-page news. (Alas, as this report goes to press, I have learned that in 2004 Hamer will publish a book called *The God Gene: How Faith Is Hardwired into Our Genes*.[100])

As Hamer suggests in his 2002 statement, association (or "bottom-up") studies have also failed consistent replication. Moreover, where results have been replicated, the identified alleles accounted for only a very small part of the variation between the affected and unaffected subjects—a matter of a few percentage points, as with the allele mentioned earlier that may account for 4 percent of the variance between those who engage in more and less novelty seeking behaviors. It is expected that association studies in the future will continue to identify genes of small effect.[101] One of the few exceptions to the rule is the discovery that an allele for an enzyme involved in the metabolism of alcohol may help to explain why some people are less prone to excessive alcohol consumption than others.[102] (Too, if Alzheimer's disease falls within the purview of behavioral genetics, then we should note that association studies have found that individuals with a copy of a particular allele [APOE4] are six times as likely to develop the common form of Alzheimer's disease as those without it.[103])

While Hamer frankly acknowledges the limits of the old model and the extreme modesty of the results it has thus far produced, he remains hopeful that better models and more powerful techniques will give behavioral genetics a boost. One of the studies that gives him hope is the New Zealand study of antisocial behavior in adult males who were maltreated as youths.[104] Many animal and human studies had suggested that levels of the enzyme MAOA can be correlated with antisocial or violent behavior, but no earlier study had shown that MAOA levels (a function of genotype) *interact* with environmental conditions to put some individuals at significantly greater risk of engaging in antisocial behavior (phenotype). That is, these researchers discovered that the likelihood that the youths in the study would engage in antisocial or violent behavior as adults was strongly correlated *both* with how much they were maltreated as boys *and* with whether they produced a little or a lot of the MAOA enzyme. Over 85 percent of the males who had the genotype associated with the *low* production of MAOA *and* who were subjected to abuse as boys developed some form of antisocial behavior by the time they were twenty-six. Those boys who had the low MAOA genotype but were not exposed to abuse were at no greater risk for antisocial behavior than those with the high MAOA genotype. (See figure 7.)

It might not be an exaggeration to say that, if replicated, the Caspi-Moffitt MAOA study will turn out to have been a watershed event in the history of behavioral genetics. Many behavioral geneticists—and medical geneticists[105]—have for some time acknowledged the need to study gene-environment interactions, and even urged the field to do so. But this is the first major study to show so vividly that if we want to understand how genotypes influence phenotypes, then we must understand how genotypes and environmental variables interact. We cannot hope to make a useful guess about the chances that an individual will become antisocial merely by looking at genotypes.

Although the Caspi-Moffitt study is important work, it really offers nothing in the way of a short-term policy payoff. No one is going to say, Well, we know this child has a high-activity MAOA genotype, and we are pretty confident that he'll be resilient in the face of maltreatment, so we need not guard against maltreatment here. Policymakers and parents will and should always do what they can to ensure that no child is subjected to maltreatment, no matter what her genotype.

Another reason the Caspi-Moffitt study is important is that it focuses on differences in the regulatory region of genes rather than in the coding region. In this respect it is importantly different from the famous study of a Dutch family in which members who produced *no* MAOA were

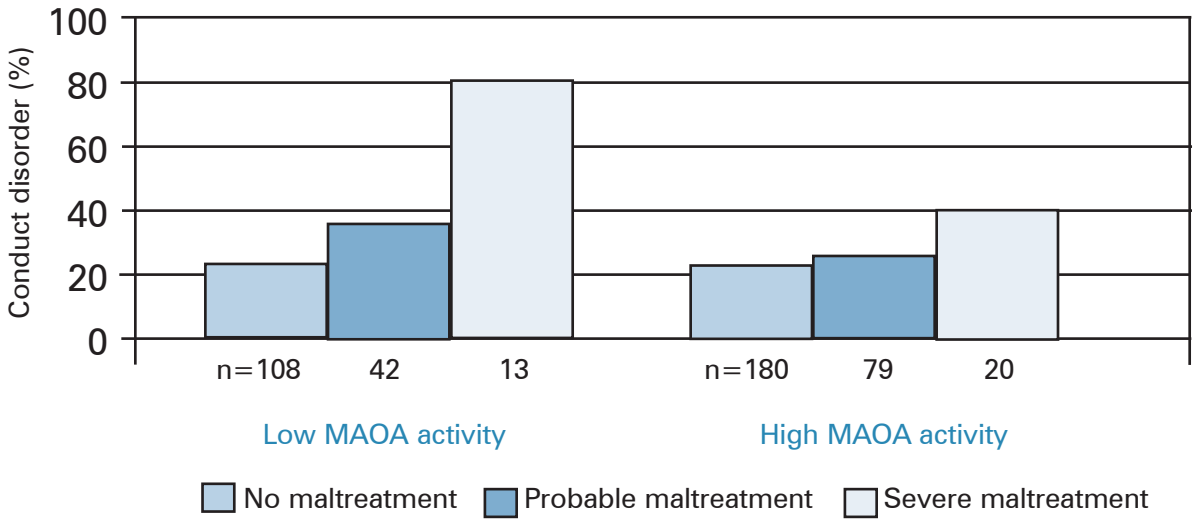

Figure 7. *Individuals who were abused as children and had low levels of an enzyme known as MAOA were twice as likely to engage in antisocial or violent behavior as individuals who had been abused as children but had high levels of the MAOA enzyme.* (Based on A Caspi et al., "Role of Genotype in the Cycle of Violence in Maltreated Children," *Science* 297 (2002): 851-54.)

shown to be prone to impulsive aggressive behavior.[106] In the Caspi-Moffitt study, the two groups of boys produced MAOA, but at different rates. That is, due to differences in the regulatory region of the gene, the same coding sequence was turned on and off at different rates. It seems that producing different amounts of the same gene product (at different times) helps to explain why some boys were more vulnerable to the long-term effects of maltreatment as youths. Time will tell how successful other researchers will be in bringing to bear insights about gene-environment interaction and gene regulation to their studies of the genetic influence on complex traits. As I mentioned above, another more recent and equally striking study by Caspi and others found a similar relationship between the regulatory region of a gene involved in the transport of serotonin (5-HTT) and stress: the chances of stressful life events leading to depression appear to be in part a function of genotype.[107]

Complex interactions among genetic and non-genetic influences in both internal and external environments

Often when we think about "non-genetic" influences, what leaps to mind are familiar environmental influences like child-rearing practices, of the sort relevant in the Caspi-Moffitt MAOA study. In addition to such external environmental influences, *non-genetic* includes, for example, the actions of neurons. Genes provide the code for the building blocks of neurons, but in and of themselves, they do not code for the crucially important patterns in which neurons are laid out in any given brain. Complex models involving genetics are increasingly aware of non-genetic influences in the internal and external environments. To borrow from Ken Schaffner, genes affect behavior largely by collectively building neurons and interacting with the organism's internal and external environments to knit the neurons together into networks, via connections called synapses.[108] But this is a very special and complicated sort of "building," insofar as the causal and informational flow between genes and the other parts of the system in which it "builds" is two-way: genes are turned on and off by proteins and environmental conditions as the network gets built and self-regulates. This idea of the two-way flow between genes and environmental conditions is, again, another way of expressing the insight that nature is always via nurture. Even with an organism as relatively simple as yeast, the flow between "nature" and "nurture" is two-way. About 66 percent of the 6,000 yeast genes are turned on and off *by the environment*—by factors such as temperature, salinity, and food supply.

This system of two-way interactions between genetic and environmental variables is staggeringly complex. As Schaffner suggests, to get a sense of just how complex this system or network is, it helps to remember that the brain has over 100 billion neurons, which are connected to each other by 100 trillion synapses. These circuits work together in largely unknown fashion to produce mental and behavioral processes.

Practical versus theoretical limits to understanding how genes influence complex phenotypes

It is not yet clear whether behavioral genetics research has had modest results mainly because the methodologies and technologies are not yet powerful enough, or mainly because the complex behaviors at issue partly result from

stochastic, unpredictable processes that will always make them recalcitrant to the methodologies of behavioral genetics. Notice, however, that this problem regarding possibly unpredictable effects is not just a problem for geneticists. It also is a problem for those psychologists and sociologists who seek to explain how environmental variables influence behavior in the terms of modern natural science.

To the more optimistic behavioral geneticists, the science is new and the problems are surmountable. They believe that technological and methodological advances will produce more replicable and useful results. As Michael Rutter has put it, "The identification of multiple genes of small effect . . . will be quite difficult. Nevertheless, through the use of multiple research strategies, it is likely that delivery will come even if it takes longer than some expect."[109]

To the more pessimistic behavioral geneticists, the problems are not only practical. The fundamental problem is with the theory, which overestimates how much analysis that begins from genes can explain about the origins of complex human behaviors. As behavioral geneticist Eric Turkheimer has put the point, "No amount of physics would ever lead to an explanation of why some objects are carpets."[110] Understanding gravity, the strong and weak forces within the atoms that make up any given carpet, and so on, may in some sense be relevant to an ultimate understanding of what a carpet is. But to understand how a carpet works, to know what you need to do to repair it if it rips, how some carpets are different from others, and how carpets are different from desks, physics is of little help.

Partial versus total explanations

Even if total or complete natural scientific explanations of complex human behaviors were theoretically possible, no scientist today would expect those explanations to be made by genetics alone. It is impossible to know now how much or little behavioral genetics will contribute to understanding human behavior. But there is little doubt that studying genes will in some cases, to some extent, help to explain the physiological pathways that help to explain why we look and behave differently. Even if one thinks that behavioral geneticists have seriously overestimated how much will be yielded by analysis that begins from genetic variation, it does not follow that such investigations should be abandoned. Partial contributions are usually better than none. Turkheimer, pessimistic though he is about the prospects of success for understanding the genetic (and environmental) causes of human behavior, does not hold that we ought to abandon research in behavioral genetics. He merely suggests that we should be cautious in our hopes about how much genetics will teach us about why some human beings act and appear differently from others.

Part 3. AN INTRODUCTION TO WHAT THE FACTS MIGHT MEAN FOR HOW WE THINK ABOUT OUR IDENTITIES

Behavioral Genetics and the Nature of Choice

Dualist versus naturalist accounts of the self

In the West there is a long tradition of dualist accounts of the self, beginning with Plato and extending to St. Augustine, Descartes, Kant, and even into the present. According to this tradition, body and mind (or "soul") are different substances, one part of this physical or natural world, the other part of a world beyond it, a "*meta*-physical" world. If you think that freedom depends upon the existence of an extra- or meta- or non-natural substance that "rules over" the substance that is your body, then behavioral genetics is just not relevant to your interpretation of freedom. Behavioral genetics does not say anything about extra-natural or metaphysical phenomena. It cannot touch the idea of an extra-natural mind or soul.

I and most if not all of our project members, however, proceeded from the naturalistic premise that when we speak of body and mind (or "soul"), we are in both cases speaking about the natural world. Even though we do not believe that natural science can give a full or adequate account of our experience, we believe that our experience emerges out of natural (as opposed to extra-natural or meta-physical) processes. I hasten to add: the naturalist tent is large, including under it not only natural scientists, but many social scientists, philosophers, theologians, and others. One can be filled with wonder at what emerges out of natural processes without adopting the hypothesis that what emerges out of or precedes those processes is "extra-natural" or "meta-physical." Neither the world in general nor our minds in particular must have been created by a non-natural force in order for them to fill us with awe.

Behavioral geneticists of course do hope to understand how one sort of natural thing, our genes, are related to another sort of natural thing, our behaviors. Insofar as behavioral genetics investigates the relationship between genes and human behaviors, the results of those investigations will be relevant to naturalist understandings of what we call our choices—and what we call our freedom. Those

results will not, however, pose the threat to freedom that they are sometimes said to pose.

Determinism versus genetic determinism

Imagine that someday a scientist demonstrates that, given a set of initial conditions for any system, she can predict all subsequent states of that system. In other words, imagine that someday someone offers an account of how a fantastically complex gene-environment system works over time to produce—or "determine"—the actions of a given individual. If such a theory could be developed, determinism would be true. We would be able to say that and how individual behaviors are determined by a fantastically complex set of gene-environment interactions. But given all that has been said above, such a determinist account could never rely on genetics alone. No geneticist today believes that genes alone determine human behaviors. Genetic determinism is a bugbear, even if determinism of the more general variety is an ancient theoretical option.[111]

Dualist versus naturalist interpretations of freedom

When a dualist like St. Augustine said we are free, he meant that God gave us a non-natural, *meta*physical thing, a soul, which does the choosing; "it" is the real, eternal you, and "it" chooses. "It" rules over your unruly body. Rejecting the idea of a metaphysical soul does not require naturalists to reject the idea of freedom. It requires only that they give some other account of freedom, one that shows how what we call "free choice" is a phenomenon located in the complex web of natural events. Of course, giving a non-dualistic account of an idea that has been articulated in dualistic terms for millennia may not prove easy. I want to sketch two versions of naturalist accounts of freedom.

In one of the papers written for our project, Gregory Kaebnick,[112] following Daniel Dennett and others,[113] suggests that we can continue to talk about freedom of the will as long as we do so in ways that are compatible with the understanding that all behavior is itself causally determined by other natural events. We can do so, according to the "compatibilist," if we adopt down-to-earth definitions of terms like "freedom of the will" so that they make no grand metaphysical claims but are instead merely descriptions of behaviors that have evolved in humans, and that humans exhibit in some circumstances. In this view, to say that a person acts freely is merely to say that the person has the cognitive capacities to set goals, think about how to pursue them, and then act so as to pursue them, and that in deciding to act, the person was not unduly coerced or constrained. If the action was compelled by physical or social circumstances ("I was forced to do it," "I was tricked and did it unknowingly," "A drug administered to me deprived me of muscle control"), then it wasn't "freely taken." So we might even say that, whereas the dualist account of freedom is about what an agent does to the world, the compatibilist account of freedom is about whether the world is doing something to the agent.

My only concern about that sort of account is that it might risk inadvertently reinstating another sort of "dualism" that we naturalists say we want to get over. That is, even though it explicitly rejects the idea of a non-natural soul or mind that rules over the body, it seems in danger of slipping into another version of dualism, in which a stable, internal, core self stands opposite outside forces, and contemplates reasons and acts in accordance with them unless constrained by those external forces.

At the risk of appearing to lapse into a sort of mysticism, I want only to allude to a slightly different version of the naturalist thesis. Like the compatibilist interpretation of freedom, this one proceeds from a rejection of the dualist idea that there is a discrete, non-natural substance that rules or chooses or "does." Indeed, on this alternative version, it is important to emphasize the respect in which the metaphor of one sort of discrete entity (a self) doing something to a different sort of entity (the world) is misleading. According to this sort of naturalist account, freedom isn't so much *done*, as it *happens*. Farther down this path I won't dare try to go. (Were one to try, one might begin with the thinking of Heidegger, who aspires to help us get over the binary modes of thinking that ensnare us when we try to talk about things like what we call "freedom.")

I want to emphasize that these two versions of naturalism represent only a family dispute. What they importantly share is the ambition of figuring out how to stay true (a) to what they believe to be the fact that there are no non-natural substances to help us to understand the idea of choosing, and (b) to *our experience* of choosing. They want to say that there is nothing inconsistent or incompatible about saying both that our choices are the result of natural events and therefore in some ultimate theoretical sense determined, *and* that one of the most significant of human experiences is the experience of making choices. According to these naturalist interpretations of freedom, we are the sorts of organisms for whom, out of an infinitely complex set of natural events, emerges the sometimes exhilarating, sometimes excruciating experience of choosing. The key to such interpretations of freedom is taking our experience seriously, quite apart from how natural science might try to explain that experience.

Understanding that our experience emerges out of a staggeringly complex set of gene-environment interactions that are mediated in the brain and change over time need not in any way diminish our appreciation of the experience that we call "choosing." Maybe it helps to recall what philosophers call "the genetic fallacy"—a concept that was around long before anyone was talking about genes. On one version of the genetic fallacy, the fallacious idea is that to understand a thing's lowly origins (its *genesis*) is to diminish its value or the respect it is due. Yet our awe before

the redwood or the rose is not diminished by our knowledge that they emerge out of dirt! Nor ought our awe before the experience of choosing be diminished by our knowledge that it emerges out of the lowly, if astonishingly complex, workings of nature.

Regardless of which interpretation of freedom we adopt, there will always be hard cases. To what extent, for example, are the choices of someone with bipolar disorder constrained? If she used her rational capacities to contemplate purchasing $50,000 worth of jewelry, purchased it and professed to have done so deliberately, on her own accord, was she free or not? But we have always faced hard cases. Genetics does not make them harder, even if it might make us alter the vocabulary we use to frame them. Way back when we thought bipolar disorder and schizophrenia were the result of bad parenting, we had trouble figuring out the extent to which we should call free the actions of a person with such a condition.

Just as we have to get used to the idea that we have to choose between overall interpretations of freedom, so we have to get used to the idea that we have to choose between interpretations of individual actions. So the answer to the questions regarding the freedom of the person with bipolar disorder or schizophrenia is: Human communities have to acknowledge that we will always face hard cases and that it's our job to distinguish between better and worse interpretations.

We did not in the past—and we need not now—understand such hard cases to be a threat to our interpretation of our choices as free. Regardless of whether our explanation of bipolar disorder relies more heavily on genetics or environment, we recognize that a person with bipolar disorder has an experience of choosing that is different from the experience of someone without bipolar disorder. Granting that exception is not a threat to our humanity, but an example of it.

From choices to temperaments

Even if genetics does not threaten the naturalistic interpretation of our individual actions as "free," nonetheless, as Kaebnick points out, it may someday tell us things about the sorts of choices we want to make or feel predisposed to make—or the sorts of temperaments we are predisposed to have. To the extent that it does, genetic information could, initially, seem to threaten our sense that we are free to choose "the sorts of people we want to be."

Will increased knowledge of the influence of genes on temperaments affect basic ethical and social ideas and practices? It most certainly will, but it is impossible to know in advance what those consequences will be. We can know, however, that those consequences will depend in part on the breadth and quality of our public conversations about these matters. It will depend in part on the extent to which we grasp that *genetic* does not mean unchangeable, and on the extent to which we grasp that determinism could be true without undermining the naturalist's interpretation of her experience as free.

Even if predicting the consequences is impossible, we can identify some obvious alternative responses to the same information. For example:

■ ***Reducing versus increasing sense of personal responsibility.*** On the one hand, an increased understanding of the extent to which genes act to influence our temperaments—and thus the sorts of actions we are predisposed to take—could be used by some to excuse their behaviors. Such understanding could move some individuals to become resigned to their genetically influenced imperfections—and contribute to a reduced sense of personal responsibility. On the other hand, insofar as "genetically influenced" does not mean unchangeable, such self-understanding could give individuals an increased sense that they are responsible to alter those imperfections, to change their temperaments by whatever means possible (exercise, drugs, cognitive therapy, and so on). Peril attends both efforts to relinquish personal responsibility and efforts to use "whatever means possible" to make individuals conform to dominant conceptions of acceptable behavior. Finding a middle way is a task of public deliberation.

■ ***Reducing versus increasing sentences for criminal behavior.*** On the one hand, as Harold Edgar suggests, an increased understanding of how genes act to influence temperaments could move some courts to give lighter sentences, on the grounds that choosing otherwise would be especially difficult for a given individual.[114] David Wasserman invited me to consider the following case: suppose we are sentencing two of Caspi's research subjects, both of whom have committed the same kind of violent crime, and we learn that one has low MAOA activity and was raised by an abusive family, while the other has high MAOA activity and was raised by a loving family. Even if we held both young men responsible for their acts, we might well be inclined to punish the former less harshly, in light of the finding (and its likely explanation in terms of a person's reduced control over impulses) that whereas over 80 percent of young men with low MAOA and childhood abuse engage in disordered conduct, only a little over 20 percent of young men with high MAOA and loving families do the same. On the other hand, however, as Wasserman, Edgar, and others have pointed out, the very same information could move courts in the opposite direction—to give stiffer sentences, on the grounds that this person is "bad to the bone."[115]

■ ***Reducing versus intensifying stigmatization of marginalized behaviors.*** On the one hand, understanding in a deeper way how genes influence temperaments could move us to think that acts or ways of life that once were thought to result from "bad moral choices" (such as homosexuality) were not really chosen, but instead were the

result of an inherited (unchosen) predisposition. The gay researchers who first announced "the gay gene" hoped that their discovery would make homophobes see that being gay is not a chosen orientation any more than is being straight—and thus is no more blame- nor praiseworthy than being straight. The alternative interpretation of the same information, of course, is that homosexual acts or ways of life are all the worse because they are "hard-wired"; on this sort of account, the genetic finding could lend support to the hateful view that people who are gay are "essentially" bad.

■ ***Increasing undesirable versus desirable medicalization.*** Sociologists use the term *medicalization* to identify the process whereby the institution of medicine brings within its jurisdiction human behaviors that once were outside it.[116] *Medicalization* is most often used as a term of blame or opprobrium. It is used to criticize medicine for overstepping its bounds when, for example, it calls the experience of women before their periods Premenstrual Dysmorphic Disorder or when it calls high distractibility and activity in children Attention Deficit Hyperactivity Disorder. The criticism is that medicine is enhancing its institutional power by giving diagnostic labels to behaviors that are perfectly natural variations of human behavior, which ought to be left alone.

But the term "medicalization" can also, at least in principle, be used in an approbatory sense, as when we laud medicine for helping us to treat behaviors that formerly were thought to result from blameworthy choices.[117] Moving from calling alcoholism a moral failure to calling it a disease is an instance of "medicalization" that is often applauded.

Surprisingly, one of the most powerful observations of behavioral genetics could be used either to criticize medicalization or to support it in both its "desirable" and "undesirable" varieties. As I said earlier, different from the species-typicality perspective characteristic of many sciences, which assumes that we can draw fairly clear lines between normal (species-typical) and abnormal (species-atypical) behaviors, the individual-differences perspective of the behavioral geneticists assumes that we cannot. A fundamental premise of the individual-differences perspective is that most traits vary continuously. Just as there is a continuum of heights within a population, there is a continuum of novelty seeking behavior, food intake, memory retention, and the rest. Plomin and fellow researchers have even wondered whether what we call schizophrenia is better understood as a "continuous" disorder rather than as an "on-off" disorder.[118] That is, schizophrenia may be at one end of a continuum on which are ranged other, less severe forms of the same trait. At the very least, it is widely believed, there is a continuum of *liability* to exhibiting schizophrenia.[119]

So, on the one hand, the individual-differences perspective, as an alternative to the species-typicality view, could be used in efforts to undermine the normal/abnormal distinction that is so often used in the medicalization process. The person speaking out of the individual-differences perspective can say, "Look, you speak as if science discovers the border between normal and abnormal. In fact, science doesn't so much discover as invent those borders. It has always been up to us to decide what counts as disease and what doesn't. Now, with the science of genetic differences, we can back up that long-standing social-constructionist insight. Bottom line: stop labeling an ever-increasing range of behaviors as in need of medical intervention."

Of course, the very same insight, which suggests that what counts as disease and as needing medical treatment is largely up to us, can be used to facilitate the medicalization process. After all, why shouldn't we broaden the range of behaviors that we decide we will treat, if people request them and say that those "treatments" make them more satisfied? If we think that what counts as disease is not written in nature, if we think that disease is what we say it is, then who's to say we shouldn't call behavior X a disease, if doing so serves our purposes?

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Even if it is impossible to know in advance what the consequences will be of new genetic information about our temperaments, again, it is imperative to recognize that such information may have different consequences for different groups. While it might be a good thing if behavioral genetics brings to light interventions that can help people change themselves in accordance with their own sense of responsibility, it is important to remember that not everyone will be able to afford such interventions. Knowing how best to deal with the differential access issue is notoriously difficult, but it would be a profound mistake to ignore it. Indeed it is possible that the same or similar information will be interpreted and applied differently by and to different groups. It is all-too-easy to imagine that white-collar criminals will be likelier to receive lighter sentences if juries are persuaded that their choices were constrained by, say, bipolar disorder, while street criminals will be likelier to receiver harsh sentences because juries will see them as bad to the bone.

Again, the consequences of new information about genetic influences on the sorts of people we are will to a large extent be determined by the interpretations we arrive at in the course of our public conversation.

Behavioral Genetics and Equality: From Equal Moral Worth to Equal Opportunity

The idea of moral equality has deep roots in Western religious traditions, which teach that human beings

are equal before God. If you think that the moral equality of humans derives from the existence of a God-given immortal soul, then what behavioral genetics has to say is utterly irrelevant to moral equality. Again, behavioral genetics is about this world of nature, not about any extra-natural or meta-physical realm.

It certainly is true that many secular conceptions of moral equality have intellectual roots in those religious traditions. But one need not endorse a religious or metaphysical account to be committed to the idea of moral equality. Even in the absence of any particular religious or metaphysical system, one can make the imaginative leap: perhaps others value their lives as much as I value my own. If I think others ought to respect the value of my existence, then perhaps I ought to respect theirs equally. Naturalists as different as Hobbes and Mill have offered variations on such an account for a very long time.

If one's commitment to the idea of moral equality depends on such an imaginative leap, then, again, the findings of behavioral genetics are in an important respect irrelevant. Indeed, we have always known that people are not created equal in the sense of being physically the same. Long before behavioral genetics came along people knew that some are taller than others, some quicker than others, some more prone to dark moods than others. Put positively, in theory at least, the findings of behavioral genetics pose no threat to the idea of moral equality. Moral equality never was about the equality of traits.

In practice, however, the situation is more complicated. Behavioral genetics is a science of human differences. Insofar as it investigates how genetic differences affect phenotypic differences among individuals, and to the extent that people believe that inherited differences are "essential" differences, the findings of behavioral genetics could be used to shore up hateful ideas about the unequal moral worth of some individuals.

Imagine, for instance, that geneticists have conclusively demonstrated that a particular allele predisposes individuals to be unable to metabolize alcohol and thus more likely to be teetotalers. In a society that valued abstinence from alcohol, it is conceivable that individuals with that allele would be esteemed more than those who, say, had an allele that increased their chances of engaging in alcoholic behavior. That is, it is not difficult to imagine that even in a society that professed a fundamental commitment to the moral equality of all persons, findings in genetics might help shore up the view that some people are better or more deserving than others. And if it were found that some groups are more prone than others to alcoholic behavior and other groups more prone to teetotaling than others, then some people will almost inevitably use those facts to shore up invidious comparisons.

If people want to use the findings of behavioral genetics to shore up their views, then it will not matter that differences that are strongly genetically influenced are no more "essential" than are differences that are strongly socially influenced, nor that genetically influenced differences are not necessarily more fixed than other differences. We do not yet have a cure for the desire to use whatever is at hand to shore up one's sense of self. Vigilance regarding that desire in ourselves and others is probably all anyone can prescribe.

So it is true that findings from behavioral genetics regarding differences among individuals could be used by those who want to reinforce hierarchies, arguing that they are rooted in "nature." But those same findings could be used to try to challenge hierarchical institutions. To see why, we need to move from talking about the equal moral worth of individuals to talking about equal opportunity and ultimately the problem of distributive justice.

As Dan Brock points out in his contribution to our book, if we understood the genetics of complex traits well enough to manipulate them, we would be forced to reexamine what we think about distributive justice. Until now, many political philosophers have argued that, while there is an obligation for societies to compensate for socially created disadvantages, there is no such obligation to compensate for disadvantages that are rooted in nature. If you believe that individuals are essentially stuck with their draw in the natural lottery, then you concentrate on responding to disadvantages that do not depend on biology; you spend your time concentrating on disadvantages that in general seem more amenable to social intervention.

Leaving aside for the moment the mistake in thinking that natural differences are fixed, we can see how, at least in principle, new genetic knowledge explodes that old assumption. Now it seems possible at least to ask, If—and this is a gigantic *if*—it were feasible to genetically "enhance" human traits and capacities, would there be an obligation to use that power to equalize the opportunities of those whose opportunities are limited by a bad draw in the genetic lottery? Even more bluntly: if it is sometimes appropriate to respond to social inequalities with social forms of affirmative action, then would it be appropriate to respond to those same inequalities with genetic or "natural" or "medical" means?

If we ought to use social means to equalize opportunities, and if there were no moral difference between using social and medical means, then one might well think that, if it were feasible, we ought to use medical means to equalize opportunities. Indeed, one might conclude that it is senseless to treat social disadvantages without treating natural ones, if both are unchosen and both have the same undesirable effects.

It is not at all obvious, however, that there is no moral difference between using social and medical means. Different means can make a moral difference.[120] Put most simply, different means express different values. A classic example to make this point involves the different means that can be used to treat mild forms of depression or "dysthmia." The psychodynamic approach, which uses words to explore the conflicts that give rise to the symptoms, ex-

presses the value of *engagement* with another and depends upon the understanding of persons as wholes or reason givers.[121] The pharmaceutical approach expresses the value of *efficiency* and depends upon the understanding of persons as complex machines. In principle, the two means and understandings are complementary, but in practice the one means and understanding increasingly crowds out the other.[122]

Not only can social and medical (or genetic) means express different values, but using genetic means to compensate for natural advantages could seem to put us on the path of reducing the diversity of human forms. If we began using both social and genetic means to compensate people for inequalities in access to resources, then we would move increasingly away from equalizing opportunities toward equalizing outcomes. If you really could use genetic means to compensate for natural differences, then as Dan Brock (following Bernard Williams) puts it, "equality becomes identity."[123]

This is indeed a surprising juncture, where some liberals could find themselves arguing *against* genetic affirmative action on the grounds that promoting it would put us on the path to eliminating diversity—on the grounds that making our personalities more alike is too high a price to pay for equalizing opportunities. (Some optimists argue, however, that an "irreducible plurality of reasonable conceptions of the good" may offer a powerful protection against any such feared loss of diversity.[124])

Fortunately, we will have a long time to contemplate whether or how much we want to use genetics to compensate for natural disadvantages. Genetic enhancement of complex human traits and capacities will not be possible for a long time, if ever. Indeed, it is difficult for those of us who think and speak about bioethical matters to strike a balance between responsibly contemplating theoretical possibilities like "genetic enhancement" and responsibly conveying how little is currently understood about how such enhancements might be achieved.

If we cannot create enhancements by adding or altering genes, some may at least hope to develop tests to try to identify and select "naturally" enhanced children. Surely entrepreneurs will step forward with preimplantation genetic diagnostic (PGD) tests and prenatal diagnostic (PND) tests that purport to pick up alleles associated with valued or disvalued traits. Indeed PGD and PND are already commonly used to test against some diseases, although the practice is controversial.[125] Sorting out the ethics of testing against disvalued traits versus testing for valued traits using PGD and PND is far beyond the scope of this project. Given the modesty of the results of behavioral genetics research so far, it is ethically imperative that we scrutinize any entrepreneur's claim to sell a genetic test that can determine the chances of a fetus or embryo expressing some complex trait.

Part 4 . THE ASPIRATION TO "PUBLIC CONVERSATION"

Public conversation

In this country, there is a sophisticated and highly developed theory devoted to understanding what it takes to engage in a special form of "public conversation" often called "rational democratic deliberation."[126] According to this body of theory, which builds on the work of John Rawls, it is possible to bring together people who are internally conflicted about some emerging question of public policy, and who are willing to give reasons for their views, to deliberate together and reach a consensus about some contested public policy matter. Indeed, one member of our working group, Len Fleck, has led two ELSI-funded projects aimed at creating "public conversations" of exactly this sort.

In public conversations aimed at reaching a consensus about a contentious policy question growing out of some new scientific discovery, the first order of business is to get clear about the scientific facts relevant to that policy question. In such conversations and in most bioethical debates, scientists can agree about the relevant scientific facts of the matter. Though it usually takes a little time, we non-scientists can usually understand enough of the science so that everybody can agree that the real debate is about the ethical and social implications of the science. For example, when scientists and non-scientists meet to discuss genetic testing, embryonic stem cell research, or the prospect of making inheritable genetic modifications, the biggest difficulty is not in agreeing about what the science says or promises. The problem is rather in agreeing about the moral implications of the science. Should insurers have access to genetic test results? Is it ethical to do research on embryos? Would it be detrimental to children to make genetics changes in the human germ line? And so on.

Our working group's situation was entirely different. Our major activity was to try to reach a common understanding of what the science has shown and promises to show in the future. Among our major difficulties were getting agreement among the scientists about what the sci-

ence has accomplished and getting the non-scientists to understand those debates.

Some of the disagreements among the scientists may at least in part have been due to the fact that they came from different fields. Jon Beckwith, who is a cancer geneticist, and Marcus Feldman, who is a population geneticist, had very critical things to say about, for example, the equal environments assumption that is so crucial in behavioral geneticists' twin studies (and ultimately in the calculation of heritability estimates). Other disagreements, however, were among behavioral geneticists themselves. Some, like Irving Gottesman, are optimistic about the prospects for clinically useful results in their field, in spite of the dearth of results so far.[127] Others, like Eric Turkheimer, are more pessimistic about the prospects for identifying the systematic effects of individual gene variants.[128] So within the scientific community there is debate about both the meaning of the findings of classical behavioral genetics and about the prospects of the molecular approaches.

It is sometimes difficult for those of us who are not scientists to know what to make of such disagreements. That difficulty is compounded by the fact that, even to begin to understand them we have to learn more about the science than has been necessary in most other bioethical debates. If one wants to follow the debate about group differences and "the genetics of intelligence," for example, she needs to know what a heritability estimate is, how it is reached, and what conclusions may be drawn from it. This is all to say that our project was about trying to understand what the science has found and what it might mean, as opposed to trying to respond to a particular policy issue through the procedures associated with rational democratic deliberation.

Tools

When we say that our project aimed to produce *tools* for public conversation, we meant that term in two senses. First, we meant conceptual tools—basic concepts and distinctions. We sought to identify the basic concepts and distinctions that one must have in hand to talk productively about what findings by behavioral geneticists mean for important ethical and social ideas. Our public conversation about behavioral genetics can be no better than our grasp of the basic scientific concepts. Likewise, we require a basic grasp of some of the different things philosophers, lawyers, and others mean when they talk about freedom and equality.

Second, we meant products, which we would create and disseminate. The report you are reading is a "tool" aimed at promoting understanding of the scientific, ethical, and social basics. To disseminate our findings to a broader audience, we also commissioned Catherine Baker to write a primer of behavioral genetics. We also created a web site (http://www.aaas.org/spp/bgenes) where people can download Baker's primer, this report, information about our project, and other information about behavioral genetics. Our working group has also created a volume of essays that explores in great detail the issues raised in this report and many more.[129]

So our project created tools that we hope will be useful for those who engage in the kind of public conversation that Len Fleck and others call rational democratic deliberation. We also hope that our tools will be of use to those who engage in the far looser but also important public conversation that consists simply in discussing and writing about what the facts of behavioral genetics are and what they mean.

There is of course no single public that engages in conversation. "Public" comprises many categories: people who watch TV or read newspapers and are moved to think about what the latest behavioral genetics findings mean for how they conceive of themselves and others; lawyers and judges who need to understand whether the latest findings should affect how they think about criminal culpability; people in health care who want to know whether the latest findings are of any clinical relevance; people who write to their congressional representatives to urge them to vote to fund or not fund research; and so forth. What all those overlapping segments of society share is the daunting task of understanding what the facts are and arriving at interpretations of what they mean for their views on fundamental issues like freedom and equality. The "conversations" aimed at such understanding happen in many places at once and are ongoing, with wildly differing degrees of clarity.

Obstacles

Surely one of the greatest obstacles to conversation about behavioral genetics is what sometimes seems to be a rather unexciting preference for simple answers. As I was trying to finish this report, I learned of an Associated Press story which asserts that "humans, like worms, may . . . possess a single gene for drunkenness."[130] In some ways, it would be nice if there were a single "gene for drunkenness" or "intelligence" or "schizophrenia" in the sense that there's a "gene for" Huntington's. If there were a gene for bipolar disorder, then presumably a cure would be more likely (although even curing so-called single-gene disorders like Huntington's has proven to be far more difficult than anyone anticipated ten years ago). If there were a gene for drunkenness, then presumably we would not have to strain to understand the complex psychological and social influences that help to explain why humans drink to excess. And if there were a gene for intelligence, then perhaps we could give everybody more of it and we could stop asking annoyingly difficult questions like, What *is* intelligence?

Unfortunately, some people are certain to have incentives to indulge the preference for simple answers. Sometimes it will be in the interest of researchers, who are in constant need of funds, to exaggerate the significance of their findings. Journalists, too, benefit from exaggeration.

The more exciting the findings sound, the easier it is for journalists to sell a story idea to their editors. "Researchers Find Gene for Bipolar Disorder" is much more arresting than "Researchers Identify Gene that May Have Small Role in Bipolar Disorder; Results Await Replication." The first formulation can work as a *New York Times* headline, the second can't. (To be fair to the reporters, they don't write the headlines. As *Washington Post* reporter Rick Weiss says in his essay for our book, reporters usually see the headline for their story when they wake up, just like the rest of us.)

Nor do bioethicists escape the temptation to exaggeration. When journalists write their stories about gene discoveries, it spices up the story to have a bioethicist pronounce that the finding will transform our understanding of what it means to be human, will lead to an overhaul the health care system, and so on. And bioethicists like to see their names in print.

Far less attractive still than the preference for simple answers and the desire to further one's own professional interests is the seemingly widely felt desire to believe that the differences we observe among individuals and groups are "hardwired," unchangeable. In U.S. culture, many people seem to believe that whites and blacks are "hardwired" to be different from each other. The sort of information produced by behavioral genetics, information about how inherited differences help to explain why people appear and act differently, can be used all too easily to reinforce the view that the differences in power that attend those phenotypic differences are fixed by biology and unchangeable.

And none of us seems to be free of hopes about how the science will turn out. Some would be pleased if behavioral genetics would overcome its problems of the last fifty years and uncover enough about the correlation between genotype and phenotype to let us screen embryos to determine the chances that an embryo will exhibit one or another complex trait. Others would be pleased if behavioral genetics never achieves that sort of understanding. Both camps need to sort out the difference between what they would like the facts to be and what the facts are. And both need to better remember that no social or moral conclusions follow either from findings that genetics is of great predictive value, or from findings that it is of little predictive value.

What we do with the findings that continue to come out of behavioral genetics is up us. Nowhere is it clearer than in the science of behavioral genetics that the facts do not speak for themselves. It is our task to interpret them and put them to salutary purposes.

Coda

The best behavioral geneticists have two admirable motivations: They hope that their research will ultimately help to reduce the suffering that attends complex disorders like schizophrenia, depression, and autism, and they hope to contribute to answering the fundamental, endlessly interesting question, Why do we behave, why do we act and appear, the way we do?

In thinking about what worries people about behavioral genetics research, it is helpful to see that there are two general sorts of concerns. One sort arises from the fact that the research purports to teach us something about human *behavior*. It might teach us, for example, that freedom is an illusion. I have tried to say why that concern is not warranted, even if we were to accept that all behavior is ultimately determined. Too, if ultimately our behaviors are determined, then among the vast array of causal forces at work are the pressures that human beings exert on each other with words and actions and social institutions. All behavior results from the interaction of genetic and environmental variables. It's always nature *and* nurture, and nature *via* nurture.

The second kind of concern is harder to allay. These concerns center on what behavioral genetics will tell us about human *differences*. In particular, by investigating the causes of human differences, people worry, behavioral genetics will undermine our concept of moral equality. The classical methods of behavioral geneticists try to suggest the extent to which genetic differences among people help to explain why people are different with respect to some trait. The molecular approaches aim to discover the specific genetic differences that help to explain those phenotypic differences. We might say that in this important respect, behavioral genetics is always about how we are *vis-à-vis* each other.

Unfortunately, there is an old and perhaps permanent danger that inquiries into the genetic differences among us will be appropriated to justify inequalities in the distribution of social power. As long as creating an identity for ourselves entails specifying how we are different from others, a science of human differences will risk being appropriated to justify claims about why some enjoy more power than others.

If it would be a serious mistake for behavioral geneticists to forget that their findings can be used to justify inequalities, it would be an equally serious mistake for commentators to ignore how the individual-differences perspective of behavioral genetics research could be put to salutary purposes. The individual-differences perspective is importantly different from the species-typicality perspective of many other scientific disciplines because it focuses on variation, not "normality." Behavioral geneticists do not deny the usefulness of the normal-abnormal distinction altogether, but they deny the brightness of the line between what we call normal and abnormal. From the individual-differences perspective, the normal-abnormal dichotomy, which is native to the species-typicality perspective, does not teach us what to expect when we observe real human beings. Because complex traits emerge out of a process in which many genes and many environmental

factors interact, we should almost always expect a continuum of phenotypes.[131]

When we observe real human beings, we should expect most of their traits to be distributed in the continuous manner depicted by the bell-shaped curve. The question is, when we look at that curve, what do we see? The species-typicality perspective trains us to focus on the mean, but the individual-differences perspective directs our eye to the continuous variation. Because of this feature of behavioral genetics, its insights should lend themselves well to the effort to affirm human variation—yet without justifying unequal access to privilege, status, wealth, and power.

So while the individual-differences perspective harbors an old danger, it also harbors a new opportunity. The science of behavioral genetics cannot teach us what we ought to do with its findings. We must teach ourselves to affirm the variation that behavioral geneticists describe and aspire to illuminate.

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