MISINFORMATION, MISREPRESENTATION, AND MISUSE OF HUMAN BEHAVIORAL GENETICS RESEARCH

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I

INTRODUCTION

"Behavioral genetics" does not describe a single field with a single set of methodological tools, nor does it describe a single explanatory project. Rather, different researchers are interested in answering different questions about the relationship(s) between genes, behaviors, and development, and they use different methodologies to answer their questions. The same diversity holds for human behavioral genetics: different researchers are interested in different questions, and in attempting to answer those questions they use different approaches.

At the broadest level, one can distinguish between (1) research into the *differences* in behaviors between different individuals and (2) research into behaviors *shared* by (most) individuals. It is obvious that some traits vary between people. Different people tend to act differently—when, for example, someone is said to be shy, it follows that, in general, they act differently at parties than people who are said to be gregarious. It is equally obvious that some traits do not vary much between people—although different people may speak different languages, all normal human adults (unlike other animals) use some complex language and learn that language while growing up.

Researchers interested in the differences within a population will focus on the variation within that population. For example, within normal human populations, some people are taller than others, some people score higher on standardized intelligence tests than others, and some are more prone to violent behavior than others. Researchers interested in such differences attempt to discover how these differences are associated with the presence or absence of particular genes or environments. In other words, are particular genes associated with being more (rather than less) prone to violence? Do particular environments in which children grow up result in their being more (rather than less) likely to score highly on intelligence quotient (IQ) tests?

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More generally, such research focuses on particular differences in the resources used in organismal development. Humans, for example, develop over time from a single fertilized egg to an adult capable of a variety of complex behaviors, behaviors that require a body consisting of an astonishingly complex organization of many different types of cells. The development of any complex organism requires a variety of resources. Some of these resources are genetic (the genetic material inherited from the parents), some are environmental (from the prenatal environment of the mother, to the provision of food, and so forth), and some are hard to classify (the complex subcellular systems that, in conjunction with genes, make proteins, etc.). The outcome of this development is a complex organism that differs from (and, of course, resembles) other organisms in the population in a variety of ways. The goal of research focused on differences is to find ways to associate different phenotypes with differences in how the organisms developed—whether different phenotypes had, for example, different genes or experienced different environments.[1] In these projects, the hope is that researchers will be able to explain how differences in available resources produce different outcomes.

More specifically, human behavioral genetics research that is focused on variation in human behavioral tendencies tries to associate different behavioral tendencies with genetic differences. It asks, for instance, if people who are more prone to violent behavior are also more likely to have certain genes, or if people who tend to score highly on standardized intelligence tests also share particular genetic traits.

On the other hand, researchers interested in behaviors that do not vary significantly within a population have other goals. In the study of behaviors shared by (most) humans, the purpose is to figure out how particular traits are produced in normal development. For instance, all normally developing humans acquire the ability to use language, despite growing up in different

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1. It is traditional in genetics research to distinguish between an organism's *genotype* and its *phenotype*. The genotype of an organism is the complete complement of genetic material—all of its DNA. DNA consists of a deoxyribose sugar and phosphate "backbone" linked to nitrogen-based bases. These bases, adenine (A), guanine (G), cytosine (C), and thymine (T) are the *nucleotides*, and each location on a DNA molecule where one of these bases can occur is a nucleotide site. Particular stretches of DNA are called nucleotide *regions*, which are simply a "mapping" convenience and which can be entirely arbitrary. *Genes*, on the other hand, are generally thought to be *functional* nucleotide regions (*but see* Box 1, *infra*, at 51). The most obvious functional regions are those that code for proteins; nucleotide *triplets* (three base-pairs) or *codons* can specify which of twenty amino acids gets used in forming a protein. It is these proteins, consisting of many amino acids, that are used in the cellular processes resulting in growth, reproduction, development, and the like. Famously, DNA forms a *double-helix*; these helixes themselves are wrapped tightly, and form *chromosomes*, each of which is a linear arrangement of the DNA.

An organism's *phenotype* consists of all the measurable traits of the organism *except* its DNA sequence. So while, for example, the height of a plant is an aspect of its phenotype, so would be the concentration of a particular protein in a particular leaf of that plant. As with most distinctions in biology, there are fuzzy areas—for example, the way a particular chromosome is folded can influence which genes are expressed, what proteins get made, and so forth; is this folding pattern an aspect of the organism's genotype or phenotype? Most researchers would consider them phenotypic, though they are not obviously so.

environments and having a different complement of genes. What makes this possible? How is our ability to use language produced in the process of normal human development? The goal of these studies is to discover the particular developmental pathways—the biological systems that direct development—that transform developmental resources (genes, environments, etc.) into (nearly) universal outcomes, even though only some resources are shared universally.

Researchers interested in understanding either the causes of variation in human behaviors or how human behaviors develop are at a disadvantage compared to researchers interested in answering similar questions associated with nonhuman organisms. First, ethical restrictions on human experimentation make many kinds of experiments that are standard in other model organisms impossible to perform on humans. Second, compared to traditional model organisms used in the study of behavior (nematode worms, fruit-flies, mice, etc.), human development is a very slow process. The average human lifespan is very much longer, the behavioral repertoire of humans is larger, and the individual behaviors of interest are often more complex than those of other model organisms. Despite these disadvantages, there have been active research programs in human behavioral genetics for the past century, and although progress in human behavioral genetics has been uneven, the field has advanced remarkably, given the difficulties inherent in such research. Indeed, human behavioral genetics research programs have been quick to take advantage of the advances in molecular biology and human genetics more generally.

This piece will explore some of the limits of human behavioral genetics research, focusing especially on how these limits affect the reasonableness of the interpretations and uses of the research results. Despite enormous improvements in the techniques used by human behavioral genetics research, especially over the past decade, it is still too easy to mis- or overinterpret the results of particular research projects. As the power and reliability of the tools used by researchers increases, it is especially important to keep in mind the conceptual limitations of the methodologies employed in human behavioral genetics research. Even when the technical results themselves are impeccable (itself a rarely achieved feat, given the methodological difficulties with carrying out human genetics research of any sort), one must be very careful when interpreting—and especially when using—those results. This is particularly true in areas where the results might be (mis)interpreted as having public policy or other social implications. Studies of the relationship between human behavioral genetics and the criminal law provide ample room for such dangerous mis- and overinterpretations. Given the focus of this current volume, then, there are good reasons to be particularly alert to the possibility of such misleading (and mistaken) interpretations.

II

DEVELOPMENTAL BIOLOGY AND THE INTERACTIONIST CONSENSUS

To fully understand the conceptual strengths and weaknesses of the various research techniques and the particular difficulties with adapting these techniques to studies of human behavioral genetics, one must first be familiar with what has become known as the interactionist consensus.[2] According to the interactionist consensus, organisms and all their traits are the products of development processes that involve the interaction of genetic and environmental resources at every stage. Hence, every trait of an organism is the result of the interaction of various genes and environments during the developmental process. In order to be successful, organismal development always requires the presence and coordinated actions of various kinds of resources (genetic, epigenetic, and environmental, to name a few), so it makes no sense to ask if a particular trait is genetic or environmental in origin. Understanding how a trait develops is not a matter of finding out whether a particular gene or a particular environment causes the trait; rather, it is a matter of understanding how the various resources available in the production of the trait interact over time.

However, too many references to the interactionist consensus fail to address, or even to suggest, the complexity of those interactions between genes and environments. The very possibility of the development of any given trait requires the coordinated actions of both many genes and many aspects of the developmental environment. For example, the production of a working human hand is contingent on the development of a more or less normal human body, which itself requires a vast array of genes and many environmental resources (proper food, shelter, and such).

Indeed, when it comes to the interaction of genes and environments, it is often not even particularly clear what a gene is. The same stretch of DNA (the same nucleotide region) can be involved in the production of many different proteins (through various forms of alternative splicing), and this can occur at different times and in different amounts (via regulatory genes).[3] Furthermore, proteins coded by different genes can interact to form different proteins (physical epistasis) or can merely complement or impede the action of one another (statistical epistasis).[4]

Similarly, references to the developmental environment tend to downplay the complexities and ambiguities inherent in this concept. Developmental environments include not only external environmental factors (such as food, shelter, and parental care), but also the cellular structures and organization that

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2. *See generally* Susan Oyama, Paul E. Griffiths & Russell D. Gray, *Introduction: What Is Developmental Systems Theory?*, *in* CYCLES OF CONTINGENCY 1 (Susan Oyama, Paul E. Griffiths, & Russell D. Gray eds., 2001) (providing a brief history and discussion of the concept).

3. For a very brief review of DNA and "genes," see *supra* note 1.

4. *See* Box 1.

make coordinated protein synthesis possible. For example, the formation of new membranes (a necessary step for cellular reproduction, and hence for life as we know it) is impossible without pre-existing template membranes; changes in the membranes used as templates have been implicated in important speciation events.[5] Hence, the entire environment makes possible the development of all the traits that make up the organism's phenotype, and variations in either external environmental factors or cellular structures can influence development in any number of complex ways.

Essentially, many factors contribute to developmental environments; they can be inherited from the organisms' ancestors, found in the world, or constructed by the organisms themselves.[6] Development is emphatically not merely a matter of genes providing the heritable instructions and the environment providing the raw materials. Although such an image remains popular, it is deeply misleading and empirically inadequate. Even though, in a sense, the interactionist consensus itself perpetuates this image by artificially dichotomizing the resources involved in development into genes and everything else, it is better to keep clearly in mind that genes (or, more precisely, nucleotide regions) are simply one developmental resource among many.

Box 1. Genes and Gene Expression

In the classic gene concept, a gene codes for one protein. Structurally, each gene is a string of nucleotides identifiable by a triplet of bases that form a start codon and another that forms a stop codon.[7] However, contemporary genomic research has painted a very different picture of the relationship between the physical triplets of base-pairs and the proteins that are produced; the one gene–one protein picture is no longer even remotely viable. Some ways in which the current picture is more complex are the following roles for various genes:[8]

(1) Regulatory Genes. Regulatory genes include sequences of nucleotides that, by binding particular proteins, result in other genes being made more or less active. So-called promoter regions enhance the protein production associated with other genes, whereas so-called silencer regions act to suppress the production of proteins associated with other genes.

5. EVA JABLONKA & MARION J. LAMB, EVOLUTION IN FOUR DIMENSIONS: GENETIC, EPIGENETIC, BEHAVIORAL, AND SYMBOLIC VARIATION IN THE HISTORY OF LIFE 121 (2005).

6. *See generally* LENNY MOSS, WHAT GENES CAN'T DO 75–117 (2003); F. JOHN ODLING-SMEE, KEVIN N. LALAND & MARCUS W. FELDMAN, NICHE CONSTRUCTION: THE NEGLECTED PROCESS IN EVOLUTION (2003); SUSAN OYAMA, THE ONTOGENY OF INFORMATION: DEVELOPMENTAL SYSTEMS AND EVOLUTION (2d ed. 2000).

7. *See* Karola C. Stotz, Adam Bostanci & Paul Griffiths, *Tracking the Shift to Post-Genomics*, 9 COMMUNITY GENETICS (forthcoming Spring 2006), *available at* http://www.pitt.edu/~kstotz/genes/Stotz_Bostanci.pdf.

8. *See id.* at 6–9; Karola Stotz, Paul E. Griffiths & Rob D. Knight, *How Biologists Conceptualize Genes: An Empirical Study*, 35 STUD. HIST. PHIL. BIOL. & BIOMED. SCI. 647, 649–54 (2004).

(2) Frame Shifting. In frame shifting, one continuous stretch of DNA is involved in the production of two (or more) different proteins as the two different messenger-RNA (mRNA) strands are produced from overlapping parts of that stretch of DNA.
(3) Overlapping Genes. In the case of so-called overlapping genes, one continuous stretch of DNA is involved in the production of two (or more[9]) different proteins as the different mRNA strands are produced, each from parts of the DNA stretch.
(4) Trans-splicing. In trans-splicing, two (or more) discontinuous stretches of DNA are involved in the production of two (or more) separate pre-mRNA strands that then combine to form a single mature RNA strand. The stretches involved in trans-splicing may also be involved in the production of other mRNA strands, and hence other proteins.
(5) Physical Epistasis. In the case of physical epistasis, two (or more) discontinuous stretches of DNA are involved in the production of two (or more) separate mRNA strands and two (or more) different proteins that then interact to form a third protein, which has a developmental function different from that of either of the two proteins that interact to form it.

Gene action can also be influenced by such heritable epigenetic mechanisms as the following:

(6) DNA-methylization. Methyl groups are attached to the DNA strand, influencing the activation of gene transcription. These attachments can be reliably inherited through non-genetic pathways, primarily via physical imprinting.
(7) Chromatin condensation. The shapes into which chromosomes fold influence which genes will be most easily accessed and transcribed. Variation in chromatin condensation patterns is heritable by non-genetic pathways—again, primarily by physical imprinting.

A sense of the complexity of the interactionist element of the interactionist consensus is apparent in a brief summary of behavioral genetics research on the nematode worm *C. elegans* by Kenneth F. Schaffner, listing eight rules governing the relationship between the worm's genes and behaviors.[10] These rules include the expectation that

(1) any given gene will affect many different behaviors, in part by affecting many different neurons (pleiotropy—one gene affects many traits);
(2) any given neuron will be affected by many different genes (statistical epistatis—each trait is affected by many genes);

9. Documented cases of nineteen—and more—exist!

10. Kenneth F. Schaffner, *Genetic Explanations of Behavior: Of Worms, Flies, and Men, in* GENETICS AND CRIMINAL BEHAVIOR: METHODS, MEANINGS, AND MORALS 88–90 (David Wasserman & Robert Wachbroit eds., 2001). Shaffner is a researcher involved in the conceptual bases of behavioral genetics.

(3) different genes will interact in complex ways to affect the development of particular neuron(s) (physical epistatsis—gene products interact to form new proteins);
(4) any given behavior will involve many different neurons;
(5) any particular neuron will be involved in multiple behaviors;
(6) different developmental environments will result in different behaviors in genetically identical organisms (phenotypic plasticity);
(7) development is stochastic—genetically identical organisms raised in seemingly identical environments will express different behaviors via different neuron formation (caused by developmental noise and unique environmental influences); and, finally,
(8) gene expression depends on (often heritable) epigenetic factors, such that the local developmental environment of the gene(s) in question can be expected to influence behavior.

Given the relative simplicity of the *C. elegans* nervous system and of the behaviors studied, Schaffner argues that these rules should be regarded as the default assumptions for the study of the behavioral genetics of any multicellular organism.[11] There might be cases in which the particular organism is simpler than these assumptions imply, but these will likely be very rare. Usually, the developmental pathways between genes, developmental environments, and behaviors will demonstrate at least the level of complexity these rules suggest.

Box 2. *C. Elegans*—the Reductionist's Delight

The nematode *C. elegans* has been a staple of developmental biology research since the 1960s, in large part because of its relative simplicity and its straightforward developmental progress (in addition to the advantage of its being mostly transparent, a boon for researchers wishing to keep track of which cells end up where).[12] Indeed, studying its development in a stepwise fashion seems so straightforward that Robert Cook-Deegan has referred to it as "the reductionist's delight."[13]

C. elegans has two forms—a hermaphrodite and a male form. The adult hermaphrodite has 959 somatic cells; of these, 302 are neurons, making its nervous system by far its largest organ.[14] The male is far less common in the wild, and it has slightly more somatic cells (1031); of these, 381 are neurons.[15]

In many ways, the development of *C. elegans* is very well understood. For example, it is known how each cell in *C. elegans* arrives at its final location in the organism, including which cells suffer programmed cell death as *C. elegans*

11. *See* Box 2. Shaffner, *supra* note 10, at 89–91.
12. Shaffner, *supra* note 10, at 85–86.
13. ROBERT COOK-DEEGAN, GENE WARS 53 (1994).
14. *C. elegans* has ninety-five muscle cells, the second largest system.
15. *See, e.g.*, THE NEMATODE CAENORHABDITIS ELEGANS (William B. Wood ed., 1988).

grows. It is even possible to produce a wiring diagram that shows how the synapses connect the neurons to each other and to the somatic cells.

C. elegans has a relatively small genome (about 97 million base-pairs), and a number of *C. elegans* genomes have been sequenced.[16] Although researchers have begun to understand how different genes and different environments influence the behavioral repertoire of *C. elegans*, they have not yet, despite extensive effort, determined all the genes and developmental pathways involved in, for example, the mating behavior of *C. elegans*.[17] Even though that mating behavior (involving four separate steps) is considered quite complex, it is of course vastly simpler than most human behaviors of interest to researchers involved in human behavioral genetics.

Such complexities highlight the difference between research that attempts to understand the development of traits that are widely shared within a population of organisms and research that attempts to find associations among differences in such traits. *C. elegans* researchers focus on how worms develop shared traits. For instance, in studying the worms' ability to exhibit mating behavior, they seek to identify the genes used in producing certain physical traits and to investigate to role of different aspects of the environment. This kind of work permits them to explain, in some detail, how a particular behavior is produced—what physically causes that behavior and how the structures necessary for that kind of behavior come to exist in a particular worm. This kind of research requires techniques different from those used in research focused on differences between individuals. Research focused on differences can, for the most part, ignore any environmental or genetic resources that are *shared* by all members of a population. If there is no variation in a resource, then there can be no variation in traits associated with differences in that resource.[18]

For behavioral genetics research that attempts to understand the causes of individual variation in particular *human* behaviors, it is appropriate to focus on differences in particular developmental resources (genes and environments, for example) that are causally associated with the behavioral variation. For instance, when studying why some people are more prone to violence than others, it may be appropriate to study variation in their home lives—for example, whether they were abused as children. However, this is not the same task as determining which resources are involved in the development of the trait more generally. Again, if resources do not vary within the population, they will not be identified by research programs attempting to explain individual variation in a trait, even if those resources are of fundamental importance to the

16. A variety of *C. elegans* genomes are available for download from http://www.wormbase.org.

17. *See* Caenorhabditis Elegans WWW Server, http://elegans.swmed.edu/ (last visited Sept. 25, 2005) (providing recent *C. elegans* papers, research, and the like).

18. *See generally* Robert Plomin, John C. DeFries, Ian W. Craig & Peter McGuffin, *Behavioral Genetics, in* BEHAVIORAL GENETICS IN THE POSTGENOMIC ERA 531–40 (Robert Plomin et al., eds., 2003) (outlining how this distinction is used in behavioral genetics more generally).

proper development of the trait. On the other hand, research that attempts to understand the development of behaviors more generally, including behaviors that are (essentially) universal in the populations in question, will tend to focus on all the different kinds of resources used in producing traits, including all the developmental pathways that produce organisms capable of those kinds of behaviors and that result in those sorts of behaviors being expressed.

III

TECHNOLOGIES AND TECHNIQUES: RESEARCH METHODOLOGIES AND (SOME OF) THE LIMITS OF CONTEMPORARY HUMAN BEHAVIORAL GENETICS

Since its inception, human behavioral genetics has pursued research into the possible biological bases of violence and criminality.[19] Although in recent years this research has often been seen as politically and socially controversial,[20] various research programs have continued to generate results receiving extensive attention in both scientific journals and the popular press. The following discussion introduces some of these contemporary research programs into the possible biological bases of violence and criminality[21] as examples of research techniques pursued by human behavioral genetics research programs.

This discussion will highlight two fundamentally different problems: first, the empirical difficulties facing these research programs, and, second, the conceptual limitations of the techniques these programs use. The first problem is fundamentally practical in nature. Some of the research techniques currently used in behavioral genetics research are generally very difficult to adapt to human behavioral genetics. Studies that attempt to do so produce results that are often less reliable than one might wish. Though these are empirical problems, some of them are likely unsolvable, for example because ethical restrictions on human experimentation make certain kinds of information regarding human development very likely unobtainable. On the other hand, the second problem is conceptual in nature. For some techniques, critiquing the reliability of the data obtained is less important than understanding the limits of the data's legitimate uses and interpretations. In these cases, the techniques in question, even if applied perfectly, answer very specific questions in very specific domains. As such, the results of these studies cannot simply be extended to other domains, nor can they be used to answer other kinds of questions.

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19. *See, e.g.*, DANIEL J. KEVLES, IN THE NAME OF EUGENICS: GENETICS AND THE USES OF HUMAN HEREDITY (1985).

20. *See, e.g.,* Natalie Angier, *Disputed Meeting to Ask if Crime Has Genetic Roots*, N.Y. TIMES, Sept. 19, 1995, at C1; Natalie Angier, *At Conference on Links of Violence to Heredity: A Calm After the Storm*, N.Y. TIMES, Sept. 24, 1995, at C8.

21. *See also* Laura A. Baker, Serena Bezdjian & Adrian Raine, *Behavioral Genetics: The Science of Antisocial Behavior*, *in* 69 LAW & CONTEMP. PROBS. 7 (Winter/Spring 2006).

A. Statistical Analysis of Variance: Heritability, Plasticity, and All That. . .

1. What is Heritability and How is it Measured?

Heritability is perhaps the most controversial concept in human genetics research, especially in human behavioral genetics. Over at least the past three decades, various authors have criticized both the techniques used to generate estimates of heritability in human populations and the interpretations and uses of these estimates. This is especially true for behavioral traits.[22] However, since researchers in human behavioral genetics, including those working on biological associations with variation in violence and criminality, continue to cite heritability estimates relatively often,[23] it is worth briefly covering some of the traditional difficulties inherent in the concept.

Heritability is usually interpreted as a measure of the proportion of the variance in a particular trait in a particular population that is associated with genetic variation in that population.[24] Put more simply, heritability is a measure of the extent to which related individuals in a population resemble each other more than they resemble unrelated individuals. It can be thought of roughly as a measure of how much children can be expected to resemble their parents more then they resemble the average member of the population. Heritability is, then, appropriate only for research programs interested in understanding the causes of differences between individuals within a population. Heritability will be undefined for any trait that is shared by all organisms within a population,

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22. The classic critical article is Richard Lewontin, *The Analysis of Variance and the Analysis of Causes*, 26 AM. J. HUMAN GENETICS 400 (1974). *See also* Elliot Sober, *Separating Nature from Nurture, in* GENETICS AND CRIMINAL BEHAVIOR: METHODS, MEANINGS, AND MORALS, *supra* note 10, at 47.

23. *See, e.g.*, Baker et al., *supra* note 21, at 25 (citing a broad-sense heritability estimate of 0.41 for antisocial behavior); *see also* S. H. Rhee & I. D. Waldman, *Genetic and Environmental Influences on Antisocial Behavior: a Meta-analysis of Twin and Adoption Studies*, 128 PSYCHOL. BULL. 490, 490–529 (2002) (providing a meta-analysis of over 50 studies on the heritability of criminality).

24. *See* Lewontin, *supra* note 22, at 402–09; Baker et al., *supra* note 21. This refers more particularly to broad-sense heritability. Briefly, if the total amount of phenotypic variation in a particular trait in a particular population is given by the total variance in that trait, VP (roughly, the average deviation in that population from the mean value of that trait within the population), then that variation can be partitioned out as follows:

$$V_P = V_G + V_E + V_{GxE} + e \quad \text{(Equation 1)}$$

where VG is that portion of the variation from the mean phenotypic value in the population associated with genetic variation in that population, VE is the portion of the variation from the mean phenotypic value associated with environmental variation, VGxE is the portion of the variation from the mean phenotypic value associated with gene-by-environment interactions (associated with genetic and environmental variations other than the additive effects of VG and VE), and e is everything else (in practice this includes unique environmental effects, developmental noise, and measurement errors). Broad-sense heritability, the portion of phenotypic variation association associated with genetic variation, is therefore expressed as

$$H^2 = V_G / V_P \quad \text{(Equation 2)}$$

Broad-sense heritability includes both additive and nonadditive genetic effects, whereas narrow-sense heritability includes only additive effects. Narrow-sense heritability is important in plant and animal breeding, as it provides a measure of likely response to short-term selection; it is not, however, of any particular use in human behavioral genetics.

because all the organisms in the population resemble each other equally with respect to that trait. If there is no variation in the trait, then neither genes nor environments can be associated with that variation.

It follows that heritability cannot be properly thought of as a measure of the extent to which genes are involved in the development of a particular trait. Rather, the development of a trait can involve the actions of many genes that are all critical for that trait's formation. But if those genes are shared by all organisms in the population, the trait, if it varies at all in the population, may still have a heritability of zero, since, because those genes do not vary, none of the variation in the trait is associated with genetic variation.

Accurately determining the heritability of a trait generally requires being able to sort organisms with known genotypes from a given population into known environments and to follow them throughout their development. In nonhuman animals, this is usually done through controlled breeding experiments in which organisms with known particular genotypes are physically sorted into the particular environments in which they are raised. But this only gives an estimate of heritability in the environments actually tested and for the population actually used. Accurately finding the broad-sense heritability of a trait in natural populations, where the organisms in question are not deliberately sorted into particular environments, is quite difficult; in the case of humans, it is all but impossible. That is, generating reliable estimates of heritability in humans through the sort of controlled breeding experiments done to generate estimates of heritability in nonhuman animals is not possible; it is possible, however, to generate rather inaccurate and less reliable estimates through other methods. These methods aim to separate out shared genetic variation from shared environmental variation. However, since children growing up in families usually share with each other and their parents aspects of both their environment and of their genes, teasing apart any associations these different aspects might have to variation in phenotypes is tricky.

Estimates of the heritability of traits in humans are generated from studying situations in which it is thought possible to separate the influences of shared environments from the influences of shared genes. These situations include adoption studies, monozygotic (MZ) and dizygotic (DZ) twin studies, and studies about monozygotic twins reared apart.[25]

In adoption studies, the shared variation in the phenotype of interest in siblings adopted into separate families is compared to the shared variation in siblings raised together, as well as between those groups and unrelated individuals adopted into the same and different families. The assumption is that those siblings adopted into separate families will resemble each other more than they resemble the population at large only insofar as they share similar genes; on the other hand, those siblings raised together in the same family will share

25. *See, e.g.*, ROBERT PLOMIN, JOHN C. DEFRIED, GERALD E. MCCLERN & PETER MCGUFFIN, BEHAVIORAL GENETICS (4th ed. 2001) (discussing adoption studies and providing numerous examples).

both genetic and the environmental components, and unrelated individuals adopted into the same families will share only the environmental components.

The assumption in monozygotic and dizygotic twin studies is that both mono- and dizygotic twins share (roughly) the same environmental influences (since they are raised in the same home), and hence any difference in the degree to which MZ and DZ twins resemble each other more than they do the population at large can be attributed to different amounts of shared genetic resemblance.[26]

If monozygotic twins are separated at birth (or, better yet, at conception, to avoid the shared gestational environment) and reared in uncorrelated environments, the heritability of the trait in question can be determined simply by the degree to which the twins resemble each other more than they resemble the population at large. Insofar as studies of monozygotic twins reared apart thus resemble each other more than others to a greater or lesser degree, the heritability of the trait of interest can be estimated.

None of these methods of study is ideal or, in practice, even very good. There are too many confounding factors, and it is too difficult to separate shared environmental influences from shared genetic similarities in humans.[27] But the accuracy of the heritability estimates of human behaviors emerging from these studies is really not the issue.

Heritability estimates, no matter how accurate, are of very limited use. Heritability estimates have been published for such human psychological traits as antisocial behavior and such particular behaviors as criminality and violence. Estimates of the heritability of antisocial behaviors (construed broadly) are usually said to cluster around 0.5, with only a relatively small number of studies reporting much lower or higher estimates.[28] This means that roughly half the observed variation in antisocial behavior is associated with the genetic variation present in the tested societies, rather than, say, environmental variation or other effects. Studies of violent behaviors (construed somewhat narrowly) have been less consistent, with reported estimates ranging from no discernable heritability up to around 0.5.[29] It is not surprising that different studies generate very different heritability estimates, even when they are supposed to be measuring the same behaviors or traits. This is because estimates of heritability can easily vary with the particular methods used (for instance, MZ/DZ versus adoption studies), the particular way that the trait in question is operationalized, and the particular population tested.

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26. Monozygotic twins share all of their DNA, whereas dizygotic twins share only half their DNA (the same amount as "ordinary" siblings).

27. *See* Sober, *supra* note 22, at 55–62.

28. *See, e.g.*, Nuffield Council on Bioethics, *Antisocial Behavior*, *in* GENETICS AND HUMAN BEHAVIOR: THE ETHICAL CONTEXT 72–96 (2002), *available at* http://www.nuffieldbioethics.org/fileLibrary/pdf/nuffieldgeneticsrep.pdf; Baker et al., *supra* note 21.

29. *See, e.g.*, Nuffield Council on Bioethics, *supra* note 28, at 87–96; Baker et al., *supra* note 21.

However, as has often been stressed in the literature, the problem is not the difficulty of generating accurate estimates of heritability in human populations; rather, the problem is due to the locality of the measure itself and the extent to which estimates of heritability are uninformative with respect to the causal pathways involved.[30] These two problems are briefly addressed below.

2. The Locality of Heritability

A trait that is highly heritable in one environment may have a very low heritability in another environment. For example, the heritability of adult human hair color is rather high in cultures with no tradition of dyeing hair, but it is likely much lower in cultures with such a tradition. Less trivially, in populations in which individuals have radically different access to adequate food, such environmental differences may be strongly associated with adult height, so the heritability of height may be reduced; in cultures in which adequate access to food is more common, the role of that environmental difference will be reduced, and genetic differences will be more strongly associated with height variation. Heritability estimates, then, are local—the heritability of a trait can vary with variations in the environment or with the makeup of the population. One classic approach to making the locality of heritability perspicuous is to consider the genotype's norm of reaction for a particular trait, given the possible developmental environments of interest—that is, to consider what the resulting phenotype will be, given a particular genotype and a particular developmental environment.[31] Dobzhansky, one of the founders of modern genetics, claimed that although it was incorrect to think of an organism's genotype as determining its phenotype, it was correct to think of the genotype as determining the "reaction norm" of the phenotype.[32]

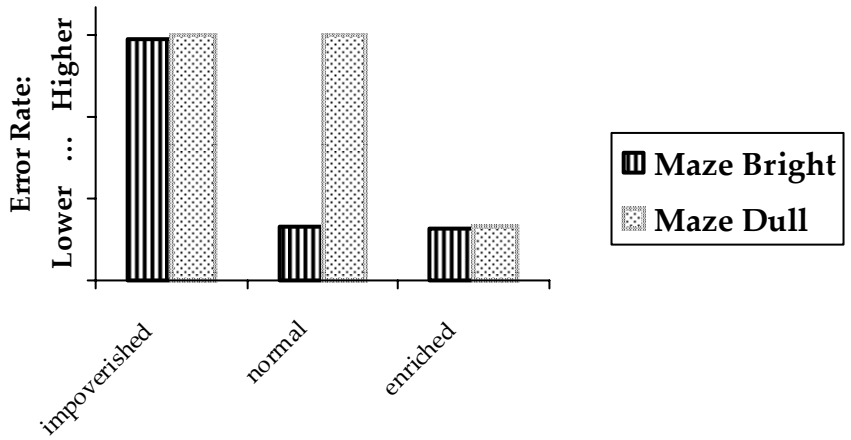
Of course, Dobzhansky was quick to note that the complete reaction norm of a genotype could never be completely known, since that would require knowing how the particular organism would develop in every possible combination of environments.[33] Partial norms of reaction represent how one trait varies when some aspect of the environment is varied. They are incomplete in that they fail to account for how variation in other aspects of the environment might affect the trait. Even so, partial norms of reaction remain a good way to understand phenotypic plasticity—that is, the differences in phenotype that emerge in different environments, even in organisms with the same genotypes. A phenotypic trait is plastic insofar as it varies with variation in the developmental environment of the organism. For example, in humans, the number of limbs is generally nonplastic (most developmental environments result in people having the same number of limbs), whereas the specific

30. Lewontin, *supra* note 22. *See also* Sober, *supra* note 22, at 47.
31. Lewontin, *supra* note 22. *See also* Sober, *supra* note 22, at 47.
32. THEODOSIUS DOBZHANSKY, EVOLUTION, GENETICS, AND MAN 74–75 (1955).
33. *Id.*

language spoken is highly plastic (whether one speaks French or English, for example, depends almost entirely on the language heard during development).

Technically, generating even partial norms of reaction requires raising genetically identical organisms in a particular set of environments. In practice, when a group of organisms is thought to possess a particular, similar genotype (by virtue, say, of their having adapted to a particular local condition), the norm of reaction is often associated with organisms with that sort of genotype, rather than with being the genotype's norm of reaction per se.[34] This is exemplified in Cooper and Zubek's work on rats bred to be either particularly good at running mazes (maze-bright), or particularly bad at running mazes (maze-dull).[35] Cooper and Zubek started with rats that were either maze-bright or maze-dull when raised under normal laboratory conditions. However, when reared in enriched environments, such as laboratory cages with lots of toys, the maze-dull rats improved immensely, while the maze-bright rats did not get much better: in the enriched environment, the two lines of rats performed similarly well. On the other hand, when raised in impoverished environments (in gray cages with no mobile objects), the maze-dull rats did not get much worse, but the performance of the maze-bright rats suffered enormously; under these conditions, the two lines of rats performed similarly poorly. Graphed, these three performances under each of the three environments can be thought of as a partial, or generalized, norm of reaction.[36]

Figure 1. Heritability, Plasticity, and Cooper and Zubek's Rats



34. These have been called "generalized" norms of reaction. *See, e.g.*, Sahotra Sarkar & Trevon Fuller, *Generalized Norms of Reaction for Ecological Developmental Biology*, 5 EVOLUTION & DEV. 106 (2003).

35. R.M. Cooper & John P. Zubek, *Effects of Enriched and Restricted Early Environments on the Learning Ability of Rats*, 12 CANADIAN J. PSYCHOL. 159 (1958).

36. *See* Figure 1.

Caption, Figure 1: This partial norm of reaction shows the different ways in which two different kinds of genotypes respond to three different developmental environments. Rats bred under normal laboratory conditions to be either very good or very bad at running mazes show a significant difference in maze-running ability when raised in those normal conditions. However, when raised in an impoverished environment, the difference in the ability of the maze-bright rats compared to that of the maze-dull rats is not statistically significant. When raised in an enriched environment, again, there is no statistically significant difference in the maze-running abilities of the two strains. Although the maze-dull rats show marked improvement between normal and enriched environments, the maze-bright rats show no such improvement. Similarly, while maze-bright rats do show a marked improvement between the impoverished and the normal environments, the performance of maze-dull rats is unchanged.

The maze-running ability of these rats show significant *plasticity* with respect to the environment in which they are raised; neither kind of rat performed equally well in all the environments. Further, the two populations display plasticity under different environmental conditions. The maze-dull rats show little plasticity in performance between impoverished and normal environments, whereas the maze-bright rats show significant plasticity in that range. The maze-dull rats, however, show significant plasticity in performance between the normal and the enriched environments, whereas the performance of the maze-bright rats is unaffected by that variation. In all three environments the rats' behavior displays a strong gene-by-environment interaction effect. That is, variation in the performance of the rats cannot be accounted for by simply adding the overall effects of the environmental variation and the overall effects of the genetic variation. Rather, different genotypes interact differently with the various available environments.

So what is the heritability of maze-running ability in these rats? The question simply cannot be answered without more information—in fact, the question does not make sense unless one knows the environments and structure of the two rat populations. If one looked only at the normal laboratory environment, the heritability of maze-running ability would be quite high; most of the variation in maze-running ability would be associated with the genetic differences between the two kinds of rats in the population. However, if one looked only at the impoverished and enriched environments, the heritability of maze-running ability would be quite low (essentially zero), since most of the variation in maze-running performance would be associated with the different environments in which the rats were reared; there would be no statistically significant difference in ability associated with the genetic differences in the two strains of rats. Given a particular population of rats, with particular numbers of maze-bright and maze-dull rats distributed in a particular way in the developmental environments, heritability could of course be calculated, but that number would hold only for that particular population. If one changed the distribution of rats in the environments, one would likely change the heritability of maze-running ability as well. It may be difficult or impossible to perform similar studies on humans, but this generalization still holds true: just because a trait (say, anti-social behavior) has a particular heritability in a particular

population at a particular time, this does not necessarily mean it will have a similar heritability in other environments or in other populations.

Heritability, then, is emphatically not a measure of the degree to which a particular trait is genetically determined. A trait can have a heritability of one hundred percent in one developmental environment, but a heritability of zero percent in another. The heritability of a trait can be one hundred percent in each of two populations, but its average difference in each population can be due entirely to environmental factors.[37] And the development of a trait can critically involve any number of genes, yet the trait itself can have a heritability of zero.[38]

Box 3: Heritability, Locality, and Genetic Determinism

I. A trait can have a heritability of one hundred percent in one environment, yet a heritability of zero in another environment. In the case of Cooper and Zubek's rats, the heritability of maze-running ability in the normal environment would be quite high; however, in the impoverished and enriched environments, it would be zero.

II. The heritability of a trait can be one hundred percent in each of two populations, but the difference in the mean value of the trait between the populations can be entirely environmental in origin. Example: Two (genetically distinct) varieties of corn, type 1 and type 2, are planted in two different fields, A and B. Each field is uniform with respect to water, nutrients, and so forth, but field A is fertilized better than B. In each field, the differences in performance between type 1 and type 2 corn will be due to the genetic differences, but the difference in performance between the fields may be due entirely to the different environmental treatments the two fields received.

III. The development of a trait can critically involve any number of genes, yet the trait itself can have a heritability of zero. Example: Normal limb development in humans involves the activity of a large number of different genes, yet the heritability of the number of legs in humans is essentially zero—almost all the variation in leg numbers in humans is associated with environmental causes (usually trauma), and not with genetic variation.

3. Heritability and Alternative Causal Pathways

Given the difficulty in sorting humans with particular genotypes into particular developmental environments, estimating the heritability of traits in humans is likewise hard. Estimates of the heritability of particular human behavioral traits tend to vary widely between studies, but this is hardly

37. *See, e.g.*, Lewontin, *supra* note 22.
38. *See* Box 3.

surprising, given the difficulties with estimating heritability in humans and the locality of heritability as a measure. But the difficulties involved in human experimentation add an additional level of uncertainty to heritability estimates. In humans, even if the heritability of a trait is known, the causal pathways through which the trait's heritability is expressed are very difficult to disentangle.

For example, in the American population as a whole, African Americans score, on average, significantly lower on standardized IQ tests than do white Americans.[39] Combined with the heritability of performance on IQ tests,[40] these statistics have been interpreted by some to imply that genetic differences in the two populations are responsible for the difference in scores.[41] Leaving aside for the moment the different environments experienced by each population and the impossibility of using heritability estimates to support a trait's heritability when the two populations experience different environments,[42] good reasons support assuming that the causal pathway is unclear. Steele and Aronson, for example, demonstrate that African American students perform significantly worse when told they are taking an IQ test then when told they are taking a test unrelated to IQ; white American students do not perform significantly differently under these circumstances.[43] Steele and Aronson attribute the underperformance of African students on IQ tests to "stereotype threat"—the threat that performing badly on an IQ test will reinforce a particular harmful stereotype—a worry that white students simply do not share. Given that skin color is heritable and that these stereotypes do exist in our society, the performance differences in IQ tests will be heritable, but not because of any genetic difference causally related in any ordinary way to IQ test-taking skills. Rather, these differences will be associated with a particular social environment—associating one's race with particular stereotypes. If that environment were changed, then the heritability of test performance would also change. The lesson here is that even an accurate estimate of heritability can say little about the causal pathways involved in generating the variation in any one trait.

Some researchers interpret these limitations to imply that finding the heritability of a trait is only a first step, which, ideally, should be followed with

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39. *See generally* Ned Block, *How Heritability Misleads About Race*, 56 COGNITION 99 (1995) (arguing that authors frequently misinterpret the concept of heritability, leading to fallacious conclusions about race).

40. Nuffield Council on Bioethics, *supra* note 28, at 72 (reporting estimates ranging between 0.35 and 0.75).

41. *See, e.g.*, RICHARD J. HERRNSTEIN & CHARLES MURRAY, THE BELL CURVE: INTELLIGENCE AND CLASS STRUCTURE IN AMERICAN LIFE (1994); Arthur R. Jensen, *How Much Can We Boost IQ and Scholastic Achievement?*, 39 HARVARD EDUC. REV. 1 (1969); J. PHILIPPE RUSHTON, RACE, EVOLUTION AND BEHAVIOR (1999).

42. *See* Box 3, II, *supra* p. 62.

43. Claude M. Steele & Joshua Aronson, *Stereotype Threat and the Intellectual Performance of African Americans*, 69 J. PERSONALITY & SOC. PSYCHOL. 797 (1995). Indeed, merely being asked to indicate one's race on the test form (by checking a box) lowered the average scores of black students, but had no effect on the average scores of white students.

studies into the mechanisms responsible for that trait's development, focusing especially on those differences in the available developmental resources that make a difference in the development of the phenotype involved. Thirty years ago, when the discovery that a particular trait in model organisms was heritable was sometimes the first step into studying the complexities of the developmental process, this line of argument was more plausible. Now, however, researchers in model organisms tend to skip estimating heritability and move directly to approaches that attempt to identify nucleotide regions associated with the observed differences. If researchers attempting to estimate the heritability of various behavioral traits in humans were more cautious and circumspect about the claims they made respecting their research results, this first-step characterization of the research would seem, if not convincing, at least harmless. However, estimates of heritability get reported in ways that make interpreting them as full-blown causal accounts all too easy. These estimates are then used in legal cases and in framing public policy issues without the appropriate cautions.[44]

4. Heritability, Causation, and Changes

Heritability, then, is a local measure—it can, and often does, change with changes in the environment or in the population more generally. It must not be interpreted as a measure of the extent to which genes are involved in the development of a trait, nor should it be thought of as revealing the causal processes by which a trait is produced. However, despite all that is known theoretically and empirically about the locality of heritability and about heritability's inability to provide causal information, strong claims continue to be made regarding what knowing the heritability of a trait entitles one to say about, for example, the possibility of changing that trait, the causal genesis of that trait, and the social policies relevant to that trait that ought to be pursued. For example, Hamer and Copeland take the high heritability of performance on standard IQ tests to mean that "no other single factor is more important than genes in determining cognitive ability."[45] They claim that the very high heritability ("70 to 90 percent") of very shy or inhibited personality types is "probably the reason such personalities do not change much during a lifetime."[46] The same kind of causal language appears when Kendler argues that the frequency of "stressful life events" encountered is "genetically influenced" through the high heritability of temperament, and that it is "because of differences in genetic constitution" that people "select themselves into high versus low risk environments."[47]

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44. *See, e.g.*, JONATHAN M. KAPLAN, THE LIMITS AND LIES OF HUMAN GENETIC RESEARCH: DANGERS FOR SOCIAL POLICY (2000).

45. DEAN HAMER & PETER COPELAND, LIVING WITH OUR GENES—WHY THEY MATTER MORE THAN YOU THINK 219 (1998).

46. *Id.* at 66–67.

47. Kenneth S. Kendler, *Major Depression and the Environment*, 31 PHARMOCOPSYCHIATRY 5, 7–8 (1998).

Once such causal language is accepted, then applied to explain behavior, its use in support of social policy recommendations is rarely far behind. Infamously, Murray and Hernstein argue from the high heritability of IQ to conclusions regarding appropriate social policies. Starting from the kinds of claims made by human behavior genetics researchers, they argue that in a society that sorts itself according to ability, some people are going to be stuck at the bottom because of genetically mediated, inherited differences in ability. Therefore, they conclude nothing much could, or should, be done about this; social programs aimed at helping the children of poor parents to achieve academic success are, in this view, a waste of money.[48] In a slightly more cautious vein, DiLalla and Gottesman argue from the high heritability of "anti-social behavior" (citing estimates of around 0.5) to the conclusion that understanding "intergenerational transmission" of violence and abusive behavior will require understanding the "genetic and biological factors" which "influence violent crime" and that "social policy decisions" formed without such an understanding will likely be "faulty."[49] Their conclusion implies that knowing the heritability of a trait can, and should, influence social policy.

The same kind of reasoning has also been used in legal cases. For example, Judge Parslow, deciding the famous custody battle of *Johnson v. Calvert* in California, cited the high heritability of IQ and other behavioral traits as a reason why genetic parenthood should determine custody.[50] Because of the high heritability of these traits, the genetic parents of a child will resemble that child more than other individuals and will thus be in a better position to understand the child. Interestingly, some authors have argued from the high heritability of IQ to the conclusion that a child's best interests might not lie with giving custody to his or her genetic parents, since that child would probably fare about the same in life, whatever the environment.[51] When the developmental environment is not strongly associated with variation in those traits, the parent's identity just does not matter that much.

In cases involving liability for lead poisoning, the high heritability of IQ has been used to justify testing the intelligence of parents and other relatives.[52] The theory was, apparently, that if the parents are none too bright and if IQ is

48. *See* HERRNSTEIN & MURRAY, *supra* note 41, at 10 ("[B]ecause IQ is substantially heritable, because economic success in life depends in part on the talents measured by IQ tests, and because social standing depends in part on economic success, it follows that social standing is bound to be based to some extend on inherited differences."); Jensen, *supra* note 41.

49. Lisabeth F. DiLalla & Irving I. Gottesman, *Biological and Genetic Contributors to Violence—Wisdom's Untold Tale*, 109 PSYCHOL. BULL. 125, 128 (1991).

50. Johnson v. Calvert, 286 Cal. Rptr. 369, 380–81 (Cal. 1993) (upholding the trial court's decision to give custody to a child's "natural" genetic mother rather than the child's surrogate birth mother).

51. *See, e.g.*, George J. Annas, *Crazy Marking: Embryos and Gestational Mothers*, HASTINGS CENTER REP. Jan.–Feb. 1991, at 35, 37 (1991); Todd M. Krim, *Beyond Baby M*, 5 ANNALS HEALTH L. 193 (1996); STEVEN PINKER, THE BLANK SLATE (2002) (offering a more contemporary spin on this idea).

52. *See* Jennifer Wriggins, *Genetics, IQ Determination, and Torts: The Example of Discovery in Lead Exposure Litigation*, 77 B.U. L. REV. 1025, 1059–65 (1997).

heritable, then the lead probably was not at fault for the child's cognitive problems, after all.

But if one takes what is known about the locality of heritability estimates seriously, it is immediately obvious that none of these claims is supportable by heritability estimates, no matter how high or how accurate that estimate may be. Hamer just gets it wrong when he writes that the high heritability of IQ implies that "no other single factor is more important than genes in *determining* cognitive ability."[53] The only supportable claim in this regard is far more cautious—namely, that within the developmental environments experienced with reasonably high frequency by the populations tested, the high heritability of IQ implies that genetic differences are more strongly associated with differences in the scores achieved on IQ tests than are other factors. But this is not Hamer's claim. Differences in performance on IQ tests might be strongly associated with any number of environmental factors, but if these factors did not happen to vary in the populations tested, their influence would be missed by analyses of variance and hence would not appear in heritability estimates.

Replacing language of association with more causal language would be misleading and indeed might mislead in socially dangerous ways. Development of complex phenotypes (including the ability to engage in complex behaviors) is marked by systems of complex feedback between the different resources available to human development. As such, a particular gene does not do the same thing throughout development, and the environment it encounters changes as the organism develops. So, for example, the development of a complex behavior could easily be influenced by environmental differences that themselves emerged from the development of an entirely different and otherwise independent phenotype. Thus the trait would show high heritability if the independent phenotype was heritable, but the heritability of the trait would be the result of the different environments encountered. Under such circumstances, to say genes associated with the differences in the independent phenotype *caused* its differences would stretch the ordinary meaning of cause almost beyond recognition.

In controlled breeding studies, these kinds of effects can usually be disentangled, but not through estimates of heritability. Rather, what environmental factors might co-vary (that is, be systematically related to each other) and how particular environmental variations might co-vary (perhaps in complex ways) with genetic differences must be considered. The experiments can then be repeated, eliminating the kinds of co-variation concerned. In the case of Cooper and Zubek's rats, for example, some researchers claimed that maze-bright and maze-dull rats did not differ in learning ability per se, but rather in curiosity. Under normal conditions, the more curious rats performed better on maze-running tasks than the less curious rats, and hence they

53. HAMER & COPELAND, *supra* note 45, at 219 (emphasis added).

appeared to be better learners; but on other tests of learning ability, in which curiosity was not a factor, this effect could be eliminated.[54]

The problem is that such studies are impossible in human populations; one cannot simply breed a new population of people and systematically test the effect of changing the environment they grow up in. So one is left with the results of research done in a particular environment, and those results are of very limited generality. A high heritability for behavioral tendencies such as antisocial behaviors or violence does not reveal the developmental causes of such behaviors or personalities. Nor does it necessarily offer a window into how such behaviors might, or might not, be modified. Again, variation in a particular trait can have a heritability of one hundred percent, yet a change in the developmental environment can result in the radical modification of that trait in part or all of the particular population. As Figure 1 shows, any argument that the high heritability of maze-running ability in Cooper and Zubek's rats in a normal laboratory environment signified the irrelevance of environmental interventions would be *false*.

B. Differences and QTLs: Statistical Correlations Made Physical

Improvements in gene mapping and sequencing over the last few decades have made finding genetic markers associated with phenotypic differences much easier. Though the power of such techniques is still somewhat limited, further improvements can be expected to make finding the particular genes (or at least small chromosomal regions) associated with phenotypic differences possible, even when the associations are weak. One technique is quantitative trait loci (QTL) analysis.

QTL analyses seek chromosomal regions that are statistically associated with differences in the particular phenotype of interest. In medical genetics, QTL studies have revealed that variations in certain regions of particular chromosomes are associated with different likelihoods of disease; so, for example, women with mutations in the BRCA1 and/or BRCA2 genes are more likely to develop breast cancer than are women without those mutations, all else being equal.[55] Importantly, these techniques are essentially statistical in nature. A successful QTL analysis reveals only that differences in a particular chromosomal region are associated with differences in the phenotype of interest; it does not provide information about the developmental pathways (if any) with which the genes in that region are involved. Indeed, at least currently, QTL analyses do not find the gene or genes associated with any particular phenotypic variation.[56] Instead, they simply identify the chromosomal region in which the putative gene can be supposed to lie. And, of course, QTL

54. *See* Norman D. Henderson, *Relative Effects of Early Rearing Environment and Genotype on Discrimination Learning in House Mice*, 75 J. COMP. & PHYSIOLOGICAL PSYCHOL. 243, 247–48 (1972).

55. Andrea Veronsi et al., *Familial Breast Cancer: Characteristics and Outcomes of BRCA 1-2 Positive and Negative Cases*, 5 BMC CANCER 70 (2005).

56. On the difficulties inherent in defining and identifying genes, see Box 1.

analyses are essentially a tool to be used for exploring differences in traits, such as how likely a person is to develop a disease or whether a person is more or less prone to violent behavior. The analysis cannot discover the genes involved in the development of any traits for which there is no variation in the particular population involved.[57]

It follows that a QTL analysis done in one environment might reveal an association between a particular chromosomal region and differences in a particular trait, but the same analysis done in another environment might reveal nothing. Imagine if the genotypic difference between the maze-bright and the maze-dull rats was at a single locus. In the normal environment, a QTL analysis would show an association of this locus with the difference in maze-running performance. However, QTL analyses done in either the enriched or impoverished environments would reveal no associations between chromosomal regions and differences in maze-running ability.

In fact, exactly this kind of plasticity has been reported in human behavioral genetics research focused on violence and antisocial behavior. In the early 1990s, researchers studied a family in the Netherlands in which many of the men (but none of the women) had a record of abnormal behavior (including violent and antisocial behaviors). Biochemical testing revealed these men to be severely deficient in Monoamine Oxidase A (MAOA), and genetic testing revealed a "nonsense point" mutation in the MAOA gene explaining the absence of MAOA.[58] But it rapidly became clear that complete MAOA deficiency was extremely rare, and studies attempting to link partial MAOA deficiency to aggressive antisocial behavior tended to be inconclusive.[59] A study subsequently performed on a population in Dunedin, New Zealand, considered the effects of the early developmental environment (mainly, the extent to which the developing child was exposed to physical abuse in the home) and found a strong relationship between growing up in an abusive household and the

57. *See, e.g.*, Sober, *supra* note 22, at 48–55. To explore how genes for which there is no natural variation in the pertinent population play roles in development, one can, in model organisms, design "knock out" experiments. In such studies, the target gene is rendered nonfunctional at some point in development. Alternatively, one can trace the activity of genes with RNA transcription in particular tissues. But ethical restrictions prevent the use of knock-out studies in humans (it is also too hard to control for confounding factors in natural experiments), and they likewise prevent the results of micro-array activation studies from being aggressively pursued. For these reasons, naturally occurring genetic variation and appeals to the results of studies in model organisms remain the primary source for the generation and testing of hypotheses in the human case.

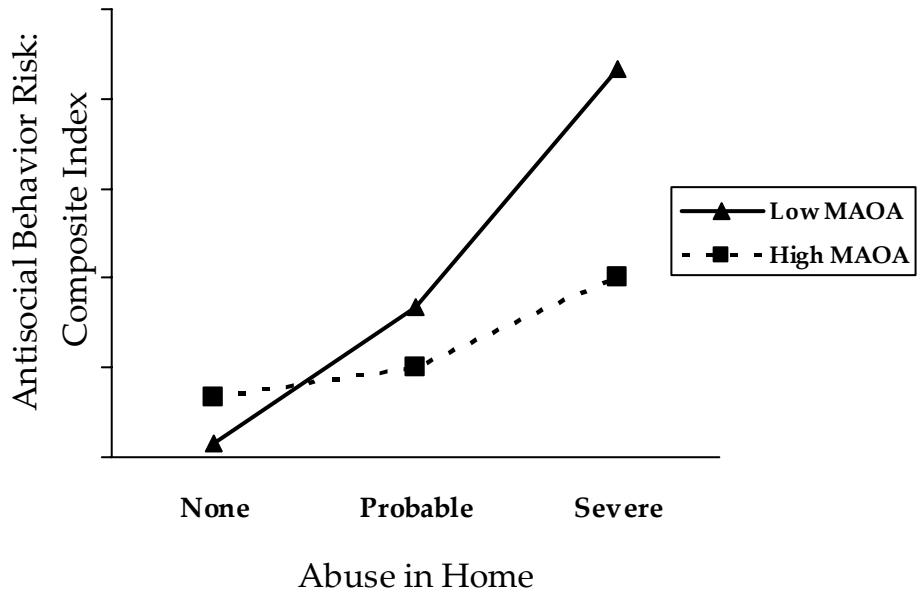
58. Han G. Brunner et al., *Abnormal Behavior Associated with a Point Mutation in the Structural Gene for Monoamine Oxidase A*., 262 SCIENCE 578, 579 (1993). A nonsense point mutation is one that prevents the formation of a protein that is usually produced; because of the redundancy of the genetic code, some mutations will have little or no effect on the protein produced, and others will result in a different protein being produced. Nonsense mutations stop transcription and prevent the protein from being produced.

59. *See*, *e.g.*, Avshalom Caspi, Joseph McClay, Terrie E. Moffitt, Jonathan Mill, Judy Martin, Ian W. Craig, Alan Taylor & Richie Poulton, *Role of Genotype in the Cycle of Violence in Maltreated Children*, 297 SCIENCE 851 (2002). This study has not been replicated, and while it appears to have been very well performed, the results may not be general in the way suggested here. But these doubts are secondary to the more significant conceptual issues discussed below.

likelihood of aggressive violent behavior later in life. This association was much stronger in people with low levels of MAOA activity.

In the Dunedin study, children were categorized according to the likely level of physical abuse in their homes, abuse generally directed at the children and their mothers. Three categories of abuse were identified: "likely none," "probable/moderate abuse," and "likely severe abuse."[60] The likely MAOA level of the children was determined by the genotype of their MAOA promoter region. Children who grew up in households rated as likely nonabusive had the same low risk of becoming violent adults, regardless of their level of MAOA. However, children who grew up in abusive households had a much greater risk of becoming violent adults if they had promoter regions associated with low-MAOA activity than if they had promoter regions associated with high-MAOA activity.[61]

Figure 2. The Dunedin Study: A Generalized Norm of Reaction—The Relationship Between Childhood Abuse, Likely MAOA Level, and the Risk for Antisocial Behaviors



This graph demonstrates the relative risk of antisocial behavior for adults who grew up in households with no abuse, probable abuse, or severe abuse, given likely low levels of MAOA or likely high levels of MAOA. The difference between the risk of becoming a violent adult is not significantly different for children with low and high MAOA levels in houses without abuse or in houses with probable (and likely mild) abuse. Children who grow up in households with severe physical abuse have a significantly higher risk of becoming violent adults than those who grew up in houses

60. *Id.*, at 297. Based on previous studies, longer promoter regions were associated with greater MAOA activity.

61. *See infra* Figure 2.

> without abuse, *whatever* their MAOA levels. But in those households, those with low MAOA levels have a significantly higher risk of this than do those with higher MAOA levels. This graph does not highlight the extensive variation in antisocial behaviors within each category.[62]

A QTL analysis would find an association between the MAOA promoter regions and antisocial behaviors only if the children were exposed to fairly serious abuse in their homes. In other environments, the different promoter regions were not associated with statistically significant differences in the likelihood that violent or antisocial behaviors would develop. This kind of plasticity may partly explain why it has been so difficult to replicate many of the studies that found loci associated with particular behavioral traits.[63] Similarly, since in this case the heritability of antisocial behaviors will vary based on the prevalence of abuse in the society in question, studies of the heritability of antisocial behaviors will yield very different numbers in different social environments.

The same problem—that genes associated with a difference in one environment may not be so associated in another environment—holds for differences in the genetic environment, as well. Epistasis, the phenomenon in which genes affect the expression of other genes, is a central fact of development. A gene associated with a particular form of a trait in one genetic context may not be associated with that form of the trait in another genetic context.[64] Indeed, with several diseases associated with particular genes in humans, the different expression of the genes in individuals is likely to be associated with differences in their genetic background. For example, how much the presence of genes associated with breast cancer raise one's lifetime risk of breast cancer is currently an area of active research. It seems likely that the answer depends on the presence of other genes. For instance, in some genetic contexts the BRCA1/2 mutations may raise the lifetime risk of breast cancer to over eighty percent, whereas in the presence of other genes, the same mutations are associated with only a twenty percent lifetime risk.[65]

Like studies of heritability, QTL analyses are a good first step in research aimed to untangle the complex developmental pathways involved. Finding chromosomal regions associated with particular differences can provide an entry into research focused on understanding those developmental processes more generally. For example, further work can be done narrowing the

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62. Redrawn from Caspi et al., *supra* note 59. On the concept of a generalized norm of reaction see Sarkar & Fuller *supra* note 34, at 71.

63. *See, e.g.*, D.C. Rowe, *Assessing Genotype-Environment Interactions and Correlations in the Postgenomic Era*, *in* BEHAVIORAL GENETICS IN THE POSTGENOMIC ERA, *supra* note 18, at 71.

64. See the developmental rules above, especially rules 2 and 3.

65. *See, e.g.*, Colin B. Begg, *On the Use of Familial Aggregation in Population-Based Case Probands for Calculating Penetrance*, 94 J. NAT'L CANCER INST. 1221, 1221 (2002) (reporting estimates of BRCA1/2 risks); *accord* Ulrich Wolf, *Identical Mutations and Phenotypic Variation*, 100 HUM. GENETICS 305 (1997) (examining similar data for cystic fibrosis, tumors, and a number of other human disorders); Barbara R. Grubb & Richard C. Boucher, *Pathophysiology of Gene-Targeted Mouse Models for Cystic Fibrosis*, 79 PHYSIOLOGICAL REV. 193 (1999) (examining the effect of the genetic background in mouse models).

chromosomal region and looking for particular candidate genes. Differences found in these genes can be analyzed with an eye towards understanding what functional differences they make to development. And that, of course, leads to the differences in development that matter to the production of different versions of the trait in question. But, again, in the case of associations between genetic markers and human behavioral traits, such research is just the first step; all too often it suggests a promise for the future, not a conclusion for the present. What gets reported and unwisely used in the framing of social and political debates, as well as in legal decisionmaking, is not the work that remains to be done (and the complex context that work involves), but that a "gene for" some behavioral trait or other has been discovered.[66]

Research into the relationship between variation in the MAOA promoter regions and antisocial behaviors can reasonably claim to have gone beyond generating mere statistical associations. For example, the MAOA system is known, at least in animal models, to be involved with metabolizing various neurotransmitters, and differences in neurotransmitters are implicated in behavioral differences (including aggression) in those model organisms.[67] It is therefore hardly a stretch to suspect that the associations found involve causally salient differences at the level of the developmental pathways.

Even so, it would be unwise to assume that the results of the Dunedin study will be easily replicated in other populations since development can be sensitive to many different environmental factors; so the correlations found in Dunedin may not be found elsewhere. But even if Dunedin results do turn out to be typical, it is unclear what, if any, policy implications they might have. After all, significant variation in antisocial behavior cannot be accounted for by associations between environments or by the different promoter regions. That is, low-MAOA activity, even when coupled with growing up in a violent household, does not guarantee that a violent or antisocial adult will result; conversely, high-MAOA activity, even when coupled with growing up in a nonviolent household, does not guarantee that the result will be a nonviolent adult. And surely the most important lesson of the Dunedin study is that growing up in a violent household is associated with an increased risk of antisocial behavior, whatever version of the promoter region one has.

This suggests in part that—despite the claim of the Dunedin researchers that the link between low MAOA, childhood exposure to violence, and antisocial behavior implies that "these findings could inform the development of future pharmacological treatments"[68]—any reasonable public health approach to reducing the prevalence and impact of violent behaviors in society ought to

66. Jonathan M. Kaplan & Massimo Pigliucci, *Genes 'For' Phenotypes: A Modern History View*, 16 BIOLOGY & PHIL. 189 (2001) (providing examples and a general critique of speaking about a gene being "for" a phenotype).

67. *See, e.g.*, Brunner et al., *supra* note 58, (correlating a mutation in part of "the MAOA structural gene" with an increase in impulsive aggression).

68. *See* Caspi, *supra* note 59, at 853.

focus on reducing violent abuse in the home rather than on MAOA levels. No pharmacological interventions are currently available; but even if they were, they would still very likely be less effective than programs aimed at reducing the prevalence of domestic violence. And given the other effects of domestic violence (including not only the direct physical and psychological harms, but also such subtler harms as the reduction of IQ scores in children exposed to domestic violence),[69] even if such programs were not wholly successful, they would still seem to be more worthwhile than pharmacological interventions.

From the standpoint of human behavioral genetics research more generally, limits in the current understanding of the developmental pathways that produce particular personality types or behavioral tendencies are to be expected; such limits do not count as a criticism of the field. Indeed, that substantial variation in the particular behaviors cannot be associated with variation in either genetic or environmental developmental resources should be expected in the study of any complex behavior, or, for that matter, that of any complex trait more generally.[70] But these limits mean that current research does not point towards any substantial predictive abilities respecting the behavior of individuals. Any public policy implications drawn from this research are very limited, indeed, and will likely remain so for the foreseeable future.

C. Evolutionary Accounts

Some researchers interested in human behavioral genetics have appealed to evolutionary accounts of particular behavioral traits. These appeals have taken several distinct forms. For instance, evolutionary psychology aims to uncover universal developmental systems that could be adaptive responses that evolved to solve particular kinds of problems in ancestral humans.[71] So some hypotheses suggest that the development of linguistic ability in humans was a solution to the problem of social coordination, and that the preference for sweet, high-fat foods as a solution to the problem of food choice for hunter-gatherers in broadly unstable environments. Evolutionary psychiatry is a related project that attempts to account for particular kinds of psycho-pathologies by reference to the mismatch between an adaptive behavioral response in the ancestral environment and current environmental conditions.[72] Insofar as a taste for high-fat foods is no longer adaptive (because it is associated with obesity), an evolved preference for such foods would represent a mismatch of this sort. Researchers engaged in more traditional behavioral genetics research will

69. *See, e.g.*, Karestan C. Koenen, Terrie E. Moffitt, Avshalom Caspi, Alan Taylor & Shaun Purcell, *Domestic Violence is Associated with Environmental Suppression of IQ in Young Children*, 15 DEV. & PSYCHOPATHOLOGY 297 (2003).

70. See the developmental rules above, especially rule 7.

71. *See, e.g.*, JEROME H. BARKOW, LEDA COSMIDES & JOHN TOOBY, THE ADAPTED MIND: EVOLUTIONARY PSYCHOLOGY AND THE GENERATION OF CULTURE 3 (1992).

72. *See* MICHAEL MCGUIRE & ALFONSO TROISI, DARWINIAN PSYCHIATRY vii–x (1998).

sometimes appeal to evolutionary accounts in order to explain the continued presence of particular kinds of heritable variation in traits.[73]

However, evolutionary psychology has been widely criticized, and these criticisms apply in large part to evolutionary psychiatry as well.[74] The primary difficulty with accounts of the adaptive evolution of human behaviors is that studying evolutionary adaptations in humans is notoriously difficult. All the standard techniques used in evolutionary biology to gather evidence that phenotypic traits (including behaviors) are adaptations are either impossible to perform or of somewhat limited use in humans.[75] Moving from a plausible account of how a particular behavioral tendency might have been adaptive in some ancestral environment to being able to provide compelling evidence that the tendency is itself an adaptation is, at best, very difficult.

Box 4: Evolutionary accounts and human adaptations

The following table lists some standard techniques used to find evidence in support of adaptive hypotheses in evolutionary biology, the kind of evidence each technique is supposed to gather, and the difficulties with using that technique in the human case.

73. *See, e.g.*, Henry C. Harpending & Patricia Draper, *Antisocial Behavior and the Other Side of Cultural Evolution*, *in* BIOLOGICAL CONTRIBUTIONS TO CRIME CAUSATION 293, 293 (Terrie E. Moffitt & Sarnoff A. Mednick eds., 1988) (developing a theoretical framework "in which antisocial behavior makes evolutionary sense").

74. *See, e.g.*, DAVID J. BULLER, ADAPTING MINDS: EVOLUTIONARY PSYCHOLOGY AND THE PERSISTENT QUEST FOR HUMAN NATURE 93-106 (2005) (arguing that there are "intractable obstacles to discovering our psychological adaptations via evolutionary" analysis because (1) it is impossible to specify what adaptive problems our ancestors faced or what would solve them, and (2) even if we could determine how our ancestors adapted, "no reliable chain of inference" connects our ancestor's adaptive problems to our current problems); Jonathan M. Kaplan, *Historical Evidence and Human Adaptations*, 69 PHIL. SCIENCE 294 (2002) (arguing that it is difficult or impossible to apply information from even the closest extant relatives to humans to evolutionary psychology because (1) either the traits studied are not widely shared in the species, or (2) the difference between humans and their relatives is big enough that any information about the relatives is likely too simplistic).

75. *See* Box 4.

Technique:	Evidence Gathered	The Trouble with Humans
Phenotypic manipulation (laboratory or field). In these studies, the trait in question is modified by the researcher.	Fitness consequences of the traits in question, causal mechanisms associated with traits and fitness consequences.	Ethical constraints; no controls in natural cases (trauma, genetic diseases, etc.).
Transplant studies. In these studies, organisms are physically moved from one location to another location with a different environment.	Fitness consequences, hypotheses about local selective pressures, hypotheses about local adaptations to local environmental factors.	Ethical constraints; few ways to control for confounding variables in natural cases; little known systematic (behavioral) variation between populations.
Laboratory evolution. Here, populations of organisms are kept and bred under controlled conditions.	Robustness of pathways (given the same environment, does the same trait develop?), strength of constraints.	Ethical constraints; also, humans are very poor model organisms due in part to their long lifespans.
Optimism analyses. These investigate what the best trait would be, given some ancestral version of the trait, problems posed by the environment, and variation in developmental resources.	Qualitative assessments (for quantitative plausibility), sensitivity, path-dependence.	Little extant knowledge of relevant selective history.
Phylogenetic analyses. These studies focus on the history of the lineage of the organisms in question—what traits do they share or not share with other species that share a recent common ancestor?	History of trait; homology (shared trait derived from common ancestor) versus homoplasy (similar traits with independent evolutionary origins).	Very sparsely populated clade (few extant relations, none of them very close); little known about environment of speciation events.

Regression analyses and comparative analyses. These compare particular versions of various traits to each other and to environmental changes over time.	Relationship between trait and environmental variables, strength of relationship, relationship between trait and fitness, relationship between trait and other traits.	Very little known about environment-trait relationships; little known about current or historical trait-fitness relationship; little known systematic variation within populations.

Nevertheless, evolutionary accounts that attempt to explain the prevalence of violent behaviors in societies have been presented in the literature. These include models that link the development of particular kinds of violent tendencies to particular environments, as well as models of frequency-dependent selection that maintains genetic variation for a propensity to violence. An example of the first is an influential article in which Margo Wilson and Martin Daly argue that many behavioral features of "violent criminals" who grew up in "high-crime" neighborhoods could be explained as rational, adaptive responses to the particular developmental environments in which they found themselves.[76] Working within the framework of evolutionary psychology, Wilson and Daly hypothesize that human psychological development reveals adaptive plasticity with respect to particular kinds of developmental environments. They argue that the development of human behavioral tendencies is determined by a number of universal developmental programs that respond to local environmental variation in ways that would have been adaptive in the environments in which their ancestors evolved. All people share the same potential to develop various psychological traits; whether they actually develop any particular trait will depend on the developmental environment they experience.[77]

Since our brains evolved to solve "important" problems regarding maximizing reproductive success within complex social systems, Wilson and Daly suggest that we should expect to find that behavioral tendencies will vary based on the developmental environment encountered and that we should thus be especially sensitive to the social environment. One upshot of this is that everyone will share the same potential to become a violent adult; whether someone actually becomes a violent adult will depend on whether he or she experiences environments that, in the past, made being violent a good strategy to adopt.[78] In environments in which the average lifespan is relatively long, rates of violent death relatively low, opportunities for low-risk social

76. Margo Wilson & Martin Daly, *Life Expectancy, Economic Inequality, Homicide, and Reproductive Timing in Chicago Neighbourhoods*, 314 BRIT. MED. J. 1271 (1997).

77. *See, e.g.*, *id.* at 1271–72.

78. *See, e.g.*, *id.* at 1273–74; *see generally* MARTIN DALY & MARGO WILSON, HOMICIDE (1988).

advancement widely available, and reasonable levels of social success widely distributed (that is, social environments with relatively low levels of social inequality), Wilson and Daly suggest that adopting a long time-horizon would be adaptive (which is to say, such a strategy will tend to increase reproductive fitness). This would include delaying reproduction, pursuing relatively low-risk, long-term strategies, and avoiding risk-taking—behaviors that all lead to broadly nonviolent psychological features. On the other hand, in environments in which the average lifespan is relatively short and the chance of dying early through violence is relatively high, in which there are large social inequalities and low-risk strategies are unlikely to accrue reasonable amounts of social success (so-called "winner take all" societies), and in which risk-taking may have large social rewards, Wilson and Daly argue it is in fact adaptive to adopt a short time-horizon. This would include early reproduction, high-risk pursuits, and high-reward strategies, including violent ones.

Wilson and Daly claim the neighborhood patterns of life expectancy, homicide rates, and reproductive timing in the United States conform to this hypothesis.[79] Neighborhoods with relatively little violence are those with relatively long life expectancies (and better overall health), relatively little economic inequality (and overall reasonably high incomes), and relatively numerous educational and economic opportunities. The most violent neighborhoods are those with relatively short life expectancies (and poor overall health), great economic inequality (and overall very low incomes), and relatively few educational and economic opportunities.[80]

Based on such data, some authors suggest searching for biological differences between more and less violent people may, in general, be pointless.[81] Instead, the differences between people may not lie in different biologies per se but rather in the different developmental environments to which the people were exposed. Although different developmental environments may produce different behavioral phenotypes that are related to different brain chemistries, for example, these latter features are best thought of as caused by environmental differences.[82]

Evolutionary psychology as a field has come under serious attack,[83] but its methodological weaknesses and poorly supported assumptions are not, fundamentally, the most serious problems facing its published results. Indeed, although Wilson and Daly acknowledge the "daunting" number of complex "feedback loops" in the pathways between the developmental environment, reasonable expectations regarding life expectancies and life strategies involving

79. Wilson & Daly, *supra* note 76, at 1273–74.

80. *Id.*

81. Alan Gibbard, *Genetic Plans, Genetic Differences, and Violence: Some Chief Possibilities*, *in* GENETICS AND CRIMINAL BEHAVIOR 169, 192–94 (David Wasserman & Robert Wachbroit eds., 2001).

82. *See, e.g.*, *id.;* Vernon L. Quinsey, *Evolutionary Theory and Criminal Behaviour*, 7 LEGAL & CRIMINOLOGICAL PSYCHOL. 1 (2002).

83. *See* Kaplan, *supra* note 74.

violence do not, and cannot, address the developmental biology of the behaviors in question. From the standpoint of how, developmentally, particular behaviors come to be expressed in particular individuals, these kinds of evolutionary accounts are silent.

Rather, the kind of evolutionary approach favored by Wilson and Daly is supposed to reveal the ultimate causes of violent behaviors[84]—to explain why such behaviors exist, not how those behaviors develop. For this reason, such approaches count as behavioral genetics only insofar as the evolution of stable developmental pathways involves the selection and maintenance of particular genes. However, it has become increasingly clear that adaptations may not involve the selection of particular genes, but rather the selection of developmental mechanisms that result in the resources available (including genetic resources) being used in different ways.[85] So, even if a particular set of evolutionary accounts is correct, it may not point towards particular genes that are uniquely involved in the production of the behaviors in question.

It may be that projects like these that attempt to uncover ultimate, evolutionary accounts would be intellectually interesting if they were better supported,[86] but because they do not address the causal pathways involved, they cannot support arguments surrounding individual differences in behavior except through the statistical associations between those behaviors and particular environments. As it is already well established that growing up in a violent society with high levels of economic and social inequality and so on is statistically associated with becoming a violent adult, the additional evolutionary account does not seem to add anything to the explanation of differences within particular populations. It certainly adds nothing that would be of value to social policies directed at individual variations in behaviors.

A second approach to using evolutionary accounts to explain violent human behaviors links genetic variation associated with behavioral differences to the likelihood of antisocial or violent behavior.[87] It does so by appealing to evolutionary accounts that could generate or maintain such genetic variations, either between or within populations. In the case of between-population differences, people in different populations might face different environments due either to the structure of the societies or to other local environmental features. These differences might, in turn, lead to local populations adapting to

84. *See, e.g.*, Quinsey, *supra* note 82, at 1–2 (explaining the concept of "ultimate" causes compared to "proximal" causes in this context).

85. *See, e.g.*, MOSS, *supra* note 6, at 75–116.

86. *But see* Richard Lewontin, *The Evolution of Cognition: Questions We Will Never Answer*, *in* 4 METHODS, MODELS, AND CONCEPTUAL ISSUES, AN INVITATION TO COGNITIVE SCIENCE 107, 118–130 (Don Scarborough & Saul Sternberg eds., 1998) (presenting a compelling argument that, in this case, the required evidence is not, and will never be, available).

87. *See* Ian Pitchford, *The Origins of Violence: Is Psychopathy an Adaptation?* 1 HUM. NATURE REV. 28 (2001); Harpending & Draper, *supra* note 73, at 293–307.

these local features and hence having different distributions of genes.[88] In the case of within-population differences, an evolutionary equilibrium might occur in which different strategies, associated with different genetic features, might exist at stable levels. However, neither the account in terms of equilibrium strategies nor the account in terms of local adaptations does much to explain the differences in rates of violence between or within populations, and neither has been seriously investigated in recent years, so neither is particularly helpful in explaining associations between genes, behavior, and environment.

IV

CONCLUSION: EVIDENCE AND THE USES OF EVIDENCE

Despite well over a quarter-century of criticism of the methodological limitations of human behavioral genetics research and the weak evidence offered for linking particular variations in human behaviors to particular biological features,[89] the standards required to publish and publicize particular claims in human behavioral genetics still remain disappointingly low. This should not come as a surprise: in nonhuman behavioral genetics and in evolutionary biology more generally, the evidence generally required to link particular behaviors to particular biological differences or to particular accounts of adaptation is also relatively weak. But there is an important difference. In the case of accounts involving, say, locally adapted populations of plants or fruit flies, the willingness to formulate and publish hypotheses on the basis of weak evidence is not deeply problematic. Nor is the acceptance or rejection of any particular adaptive account based on nonhuman studies; even if criticism of poor evidence and new hypotheses does not occur for some time, it is unlikely that any real or lasting harm will be done by the (temporary) acceptance of the original, poorly supported hypothesis about plants or fruit flies.

The situation in commercial plant and animal breeding might be considered rather different. Here, mistaken hypotheses, if not discovered quickly, could be costly. But commercial plant and animal breeders have important advantages over researchers hoping to understand the development of traits in natural populations. Studies relevant to commercial plant and animal breeding can, and do, control the environments in which the experiments are performed. This is not problematic in the least, as the goal is not to mimic some range of natural environments, but rather to recreate the artificial environments in which those

88. *See* Massimo Pigliucci & Jonathan Kaplan, *On the Concept of Biological Race and its Applicability to Humans*, *in* 70 PHIL. OF SCI. 1161 (Supp. 2003) (discussing the possibility of human ecotypes (locally adapted populations)).

89. *See generally* PHILIP KITCHER, VAULTING AMBITION: SOCIOBIOLOGY AND THE QUEST FOR HUMAN NATURE (1985); RICHARD C. LEWONTIN, BIOLOGY AS IDEOLOGY: THE DOCTRINE OF DNA (1991); Richard C. Lewontin, *The Evolution of Cognition: Questions We Will Never Answer, in* METHODS, MODELS, AND CONCEPTUAL ISSUES, *supra* note 75, at 107. RICHARD C. LEWONTIN, STEVEN ROSE & LEON J. KAMIN, NOT IN OUR GENES: BIOLOGY, IDEOLOGY, AND HUMAN NATURE (1984); HILARY ROSE & SEVEN ROSE, ALAS POOR DARWIN (2000); MICHAEL RUSE, SOCIOBIOLOGY: SENSE OR NONSENSE? (2d ed. 1979).

plants or animals are usually raised. For this reason, studies involving commercial plant and animal breeding do not need to control for the manipulation of their subjects; the subjects of such studies are always manipulated.

Similarly, in principle it would be possible to pursue human behavioral genetics research that would be accurate, as long as one could selectively breed humans in controlled environments, and as long as all one wanted to know was the relationship between particular genes and particular behaviors in those populations and in those controlled environments. However, even if such studies were ethically feasible (not to mention practically feasible), they would still be inadequate for understanding the relationship between variation in human genetics and variation in human behavior. Human behavioral genetics is not—and ought not be—interested in the relationship between particular genes and particular behaviors in some particular, artificially structured population and in some artificially controlled environment. The goal of behavioral genetics is not to find associations that hold true within artificial environments or artificially created populations. Rather, the environments of interest to human behavioral genetics are those natural environments in which the populations of interest develop.

Further, insofar as human behavioral genetics is expected to generate research results with public policy implications, there are good reasons to reject any research model that fails to focus on the actual environments encountered during development and on the actual populations experiencing those environments. Public policy decisions cannot be sensibly made on the basis of what might happen to a particular population in a particular controlled environment. Rather, such decisions should take into account the ways in which the developmental environments encountered vary, within populations, between populations, and especially over time.

Even if contemporary approaches to finding biological correlates to violent, antisocial, or criminal behavior are successful, the research results are unlikely to contribute meaningfully to shaping public policy, to making better legal decisions, or to improving our understanding of the causes of violence within societies. Because contemporary techniques cannot reveal the causal pathways of development in the human case, they cannot be used to predict how particular developmental resources will be used in different developmental environments. Hence, they cannot predict the results of any particular social policies. Changes in social policy are, after all, environmental changes, and changes in the developmental environment may well change the associations between particular genes and particular behaviors. As a result, any social changes made in response to research might change the associations uncovered by the research itself. The conceptual limitations of the techniques employed by these research programs simply do not permit the results of such research to be used in the kinds of explanations or predictions that could meaningful influence social policy.

A substantial literature is emerging from moral philosophy, legal studies, and the social sciences on how to deal with possible discoveries of links between biology and violent crime. Various authors have argued that some possible discoveries would force a major rethinking of basic moral intuitions or major revisions in the public policies surrounding violent crime.[90] In these hypothetical cases, one is often asked to imagine discovering biological correlates to criminal violence that predispose an individual to commit violent acts in every possible developmental environment or biological correlates that make an individual much more impulsive and thus more unable to control his or her temptations than the norm.[91] More dramatically, one could imagine discovering biological pathways that make it certain an individual will commit violent acts—pathways that would determine him or her to be a violent or antisocial adult.[92]

Interesting as they are, perhaps, as an intellectual exercise, these hypothetical cases should not be permitted to distract from what is already known about how to reduce the prevalence and ameliorate the impact of violent, antisocial, and criminal behaviors. And despite the over-bold claims of some human behavioral genetics researchers, it is not at all likely that more biological data would even help in reducing such behaviors. Rather, what seems to be missing from attempts to deal seriously with these problems is a willingness to act on what is already known about techniques for reducing them. It seems reasonable to suggest, therefore, that people concerned with actually reducing the prevalence and impact of violent behavior in societies or interested in finding ways to reform the legal and penal systems should focus on these broader political questions. To reduce the prevalence and ameliorate the impact of violent, antisocial, and criminal behavior within societies, such people should treat biological research as, at best, intriguing distractions from the hard work ahead.

90. See especially Part II of GENETICS AND CRIMINAL BEHAVIOR, *supra* note 10.

91. *See* Marcia Baron, *Crime, Genes, and Responsibility*, *in* GENETICS AND CRIMINAL BEHAVIOR, *supra* note 10, at 204–05.

92. *See* Peter Van Inwagen, *Genes, Statistics, and Desert*, *in* GENETICS AND CRIMINAL BEHAVIOR, *supra* note 10, at 225 (discussing the moral culpability of individuals genetically predisposed to commit violent acts).