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Genetic Fatalism and Social Policy: The Implications of Behavior Genetics Research

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Recent advances in molecular genetics methods have provided new means of determining the genetic bases of human behavioral traits. The impetus for the use of these approaches for specific behaviors depends, in large part, on previous familial studies on inheritance of such traits. In the past, a finding of a genetic basis for a trait was often accompanied with the idea that that trait is unchangeable. We discuss the definition of "genetic trait" and heritability and examine the relationship between these concepts and the malleability of traits for both molecular and nonmolecular approaches to behavioral genetics. We argue that the malleability of traits is as much a social and political question as it is a biological one and that whether or not a trait is genetic has little relevance to questions concerning determinism, free will, and individual responsibility for actions. We conclude by noting that "scientific objectivity" should not be used to conceal the social perspectives that underlie proposals regarding social change.

> "We used to think our fate was in our stars. Now we know, in large measure, our fate is in our genes" (J. Watson, quoted in *Time Magazine*, March 20, 1989).

The power of new genetic technologies has revolutionized the study of the biological underpinnings of many human traits. In the last several years, these new approaches have been used in attempts to identify genes associated with certain human behaviors. Geneticists employing linkage studies have reported the location of genes correlated with alcoholism [1], manic depressive illness [2, 3], schizophrenia [4], Alzheimer's Disease [5–7], sexual orientation [8] and aggression [9]. In several cases, the initial reports of positive results have been subsequently withdrawn or severely criticized [10–14]. Nevertheless, these reports, and the publicity attending them, have given renewed public attention to the thorny subject of genetics and human behavior.

Biological explanations of human behavior have a long history. One important belief often associated with these explanations is that, if a behavior can be said to be "genetic," then that behavior is fixed and unchangeable. We will refer to such a belief as "genetic fatalism." During the first quarter of this century, researchers in the United States concluded that the newly developed I.Q. tests measured inborn and fixed intelligence [15, 16]. They argued that the low I.Q. scores of immigrants from Southern and Eastern Europe meant that the influx of these "biologically inferior" people into the United States would have deleterious effects on the quality of the gene pool in the United States. In

[a]*To whom all correspondence should be addressed.* Department of Chemistry, University of Massachusetts-Boston, 214 Huntington Avenue, Boston, MA 02125. Tel. (617) 287-6134.
[c]*Abbreviations used:* RFLP, restriction fragment length polymorphisms; MDI, manic depressive illness; PKU, phenylketonuria.

1975, E. O. Wilson stated that the science of sociobiology, which applies Darwinian evolutionary theory to the study of animal and human societies, showed that differences between men and women in societal achievements were genetically based. These differences cannot be easily eliminated, according to Wilson, since "the genetic bias is intense enough to cause a substantial [sexual] division of labor even in the most free and egalitarian future societies" [17]. Subsequently, in a modified version of this genetic fatalism, Wilson, while ceding that genetic does not necessarily mean unchangeable, suggested that efforts to interfere with our evolutionary heritage through social engineering would generate new social problems [18]. In 1991, researchers Michael Bailey and Richard Pillard argued that there is a substantial genetic contribution to homosexuality [19]. Such a conclusion, they contend, undermines a fundamental argument for discrimination, that homosexuality is a "social corruption" [20]. Implicit in the researchers' arguments for the social acceptance of homosexuality is the assumption that, if the behavior is genetically based, then gay men have little choice over their sexual orientation.

Historically, beliefs in genetic fatalism, particularly as they relate to human behavior, have been used to inform and influence social policy. Hereditarian explanations of human behavior have served to rationalize and justify existing social arrangements, such as class structure, male dominance, and xenophobia. It has been argued that these phenomena are universal (present in all societies), genetic, and resistant to change. Further, it is sometimes claimed that, even if these traits could be changed, they are "optimal" in the sense that they represent the best possible result given an imperfect human nature. Thus, most often, genetic fatalism has been used in a conservative fashion, to support the naturalness of the *status quo*.

However, conservative social policy is not a necessary corollary of arguments for a fixed biological basis for human behavior. In the early part of this century, eugenics policies, based on beliefs in genes for intelligence and other social traits, were advocated by socialists as well as conservatives [16, 21]. While the right supported eugenics to prevent "degradation of the gene pool," the left offered alternative rationales for eugenics, including the goal of improving the lot of the working class. Bailey and Pillard, who base their conclusions on twin studies [19], and Simon LeVay, who studied the relationship between brain structure and homosexuality [22], hope that their results will lead to greater social acceptance of homosexuals [20, 23].

Further, not all those who pursue studies on the biological components of behavior subscribe to the concept of genetic fatalism. Melvin Konner, a sociobiologist [24], Sandra Scarr, who studies the biological basis of intelligence [25], and Patricia Davidson, a mathematics curriculum developer who cites studies on the effect of brain differences between boys and girls on math performance in her work [26], all believe that traits with a biological base can be changed by changing the social and educational environment.

In this paper, we examine these different perspectives on the social implications of studies on the biological basis of human behavior. We begin by reviewing the definitions of some of the concepts of human behavioral genetics and comparing the various approaches used to study the genetic basis of traits. We then discuss the malleability of traits with either a strong environmental or a strong genetic component. Finally, this background is used to explore the social policy implications of behavior genetic studies

and to examine the relationship between free will and genetic determinism.

In discussing genetic theories of human behavior, we find it useful to divide the approaches into two categories: nonmolecular and molecular. Nonmolecular studies involve such disciplines as sociobiology, psychology (e.g., identical twin and adoption studies), anatomy (e.g., the study of anatomical brain difference between gay and straight men or between men and women), and physiology (e.g., the study of particular receptors in the brains of people with and without alcoholism). Molecular studies are designed to obtain information about the actual genes or gene products responsible for the behavior.

The distinction between molecular and nonmolecular studies is important because, for the most part, the methodologies used are quite different. Since the molecular studies involve a search for the genes themselves, certain aspects of the technical analysis and interpretation are relatively straightforward. In contrast, nonmolecular studies rely on less direct evidence for the existence of genes correlated with a behavior. For instance, the use of the abstract concept of heritability developed by quantitative geneticists is essential to this approach. Furthermore, there is an overall discernable difference in the attitudes of molecular and non-molecular researchers with regard to the malleability of biological traits. This difference can lead to contrasting views on the political and social implications of finding that a trait is "biological."

GENETIC FATALISM, HERITABILITY AND NONMOLECULAR STUDIES

Current research endeavors to find specific genes associated with human behavioral traits derive directly from decades of behavior genetics research. Without the suggestions from nonmolecular studies of a genetic basis for schizophrenia [27], alcoholism [28, 29] and, most recently, homosexuality [19, 22], there would be considerably less incentive for the molecular biologists to devote their time to gene-hunting projects. Consequently, it is important to evaluate the methods used in nonmolecular studies of behavior in order to determine how solid a basis there is for the molecular studies. Elsewhere, we have discussed some of the pitfalls in approaches, such as studies of identical twins [30]. Here, we consider the language used in assessing genetic contributions to human behavior and the assumptions that underlie this terminology.

The modifier "genetically determined" is used to describe a trait that exhibits the same or a similar phenotype in all usual environments. Number of ears and hair color are two examples of genetic traits. Unlike "genetic fatalism" which assumes that a genetic trait cannot be changed, the word determined does not necessarily indicate how malleable that trait might be. Hair color, for example, is readily changed by dyes. And, in a society that punished criminals by cutting off one or both ears, the number of ears would be a malleable trait. Moreover, since our knowledge of the interactions between genes and environment is quite limited, it is often not possible to ascertain how the genotype will be expressed in a new untested environment.

Instead of calling a trait genetically determined, it is often stated that a trait has a large genetic component. If "large genetic component" is taken simply as a synonym for genetically determined as defined above, then this usage is unproblematic. However, it is meaningless to attribute a portion of a trait to a genetic component and the remainder to the environmental component. For example, one cannot say that 60 inches of a six-foot

man's height are due to his genes and 12 inches are due to the environment.

Quantitative geneticists introduced the concept of heritability in order to allow quantitative estimation of the importance of genes in contributing to a trait [31]. Heritability does not provide information on the genetic component of a trait in any one individual. Rather, heritability is a measure of the fraction of the variability of the trait in a population of individuals living in a particular range of environments that is due to genetic differences within the population. More precisely, heritability is defined as the variance of the trait seen in a population due to genetic differences divided by the total variance in the trait. The variation in the trait that can be attributed to genes is called the genetic variance and the total variation is the phenotypic variance. The phenotypic variance is the sum of genetic and environmental variances plus the variance due to the complex interrelationships between genes and the environment. While "heritable trait" is synonymous with "genetic trait," a genetic trait, as we show below, need not have a high heritability.

An accurate estimate of a heritability coefficient depends, in part, on being able to separate the contributions to the phenotypic variance arising from genetic and from environmental causes. However, there are two contributions to the phenotypic variance that make this separation difficult: genetic-environmental covariance and genetic-environment interaction [31].

Genetic-environmental covariance occurs when certain genotypes and certain environments are associated. For example, let us assume that I.Q. is influenced both by heredity and by the degree of intellectual stimulation in the environment. If that is the case, then parents with high I.Q.'s, due in part to genes, will tend to provide a milieu for their children that enhances their performance on the tests beyond that expected from their "intelligence genes." It is a matter of debate whether the enhanced performance due to the covariance should be included in the genetic or environmental variance in the calculation of a heritability coefficient.

Genetic-environment interaction occurs when one genotype may lead to a phenotype that is considered better in environment 1 than in environment 2, while another genotype might be better in environment 2 than 1. For example, students with different genotypes for math performance (assuming that such "math genes" exist) might require different pedagogical approaches in order to reach their full potential. Patricia Davidson argues that girls learn certain fundamental principles in mathematics differently from boys [26]. If this were true, it could well be that providing the best teaching approach for girls as the standard would lead to girls outperforming boys on SAT tests. In this example, such changes in the environment will result in large changes in the heritability estimates for the trait.

Dissecting out the importance of these different contributions to phenotypic variance is a very difficult task. In studies of the heritability of a trait in people, researchers are obviously unable to perform the type of controlled experiments that can be used in heritability studies of nonhuman animals and plants. For example, in order to measure gene-environment interactions, one would need many groups of people with individuals within each group having the same genotype. Then, for every group, each individual would be placed in a different environment. Since such experiments cannot be conducted with people, researchers studying human traits must either ignore the covariance and interaction,

assume that they have little effect on heritability coefficients, or attempt to apportion these factors between purely environmental and genetic variances. These various strategies for dealing with the inherent difficulties of determining heritability have elicited strong criticisms [32].

It is a paradoxical consequence of the definition of heritability that traits normally regarded as being genetic do not necessarily have a high heritability. As indicated above, the trait of having two ears is a genetic one; in all usual environments practically every individual will have two ears. Since variation in ear number is rarely due to a genetic difference, but rather more commonly is the result of an accident or other environmental event, the genotypic variance is much less than the environmental variance. Thus, the heritability, the genotypic variance divided by the total variance, is close to zero. Other examples of this property of heritabilities are traits that are closely connected with reproductive fitness. These traits, though clearly genetic, tend to have low heritabilities [31].

We emphasize that the heritability of a trait is defined for a particular population and for a particular range of environments. This point was raised most prominently during the debate about I.Q differences between the races [33]. Opponents of the view that the difference between the average I.Q score of whites and blacks is genetically based emphasized that a finding of high heritability of I.Q. among the white population in the United States provides no information about its heritability among African-Americans, since the two groups experience very different environments. Moreover, there is no theoretical reason why performance on I.Q. tests and the heritability of I.Q. could not change if the environment (social, psychological, nutritional, etc.) of every child were improved.

The heritability of intelligence, as measured by I.Q. tests, has been estimated to be as high as 80% [34, 35]. This high figure, if correct, means that the differences currently observed in I.Q. among people are largely due to genetic differences. Based on this estimate, it has, at times, been concluded that these differences are irremediable and cannot be significantly reduced by compensatory education [34]. But, an estimate of heritability provides no information on what the impact of novel educational approaches might be. As a result of interactive effects, a change in environment could have a greater effect on the I.Q. scores of some children than on others. In other studies of identical twins, researchers have claimed that such traits as personality, job satisfaction, and religious interests are approximately 50% heritable [36]. These findings have led some commentators to argue that the home environment and other life experiences have little effect on these behavioral characteristics [37, 38].

Despite this fundamental feature of population genetics, some scientists and more especially the popular media have tended to equate high heritability with "large genetic component" and have assumed that both concepts imply genetically fated.

The heritability concept was invented for analyzing the inheritance of traits that are believed to arise from the combination of the effects of many genes [39]. For such highly polygenic traits it is questionable whether molecular methods, which study the operation of a single gene or of a few genes at most, will be useful for the foreseeable future in determining a genetic causal mechanism. Consequently, most researchers who study the genetics of complex human behavioral traits such as intelligence and aggression continue to use nonmolecular approaches and focus on obtaining heritability estimates.

MOLECULAR APPROACHES TO HUMAN BEHAVIOR GENETICS

If a trait is influenced by a single gene or, at most, a few genes, then it is often possible to obtain detailed information about the causal mechanism of that trait by using molecular approaches. For certain non-behavioral conditions, such as cystic fibrosis, many mutations in a single gene have been discovered in the last few years and a great deal has been learned about the gene product and its function [40]. Cystic fibrosis studies have already dramatically increased our understanding of the biochemical and physiological processes responsible for the disease. In a more complex condition with behavioral aspects, Huntington's disease, RFLP[c] analysis showed linkage to markers on chromosome 4 [41]. This knowledge led to the development of diagnostic tests that, together with a family history, can determine the probability that a person at risk for Huntington's disease will develop the disease. Most recently, a candidate for the Huntington's disease gene has been identified [42].

The RFLP technique has also been applied, although with less success, to psychiatric diseases such as MDI [2, 3] and schizophrenia [4]. A wave of recent articles describe the difficulties and pitfalls involved in linkage studies, especially of complex psychiatric diseases [11, 13, 30, 43–47]. These include problems in establishing definitive diagnostic criteria for complex behavioral traits, errors in experimental design, and questions concerning the appropriate statistical measures for deciding that a linkage is significant. Thus, despite the more precise nature of the linkage approach to studying human behavior, even molecular studies are prone to serious error. However, molecular studies can be readily repeated by other researchers and, as a result, any flaws in the original studies are likely to be revealed.

Unlike the molecular studies, it is questionable whether twin and adoption studies are truly replicable. The variation in the method of ascertainment of subjects, the array of social settings in which such studies are done, and the use of different criteria and measurements to define a behavior make such heritability studies incommensurable. In fact, different familial studies of the inheritance of such traits as intelligence have produced dramatically different heritability estimates. A widely cited article reviewing these studies simply combined the various heritability estimates rather than providing critical evaluations of each of the studies [35].

THE MALLEABILITY OF GENETIC TRAITS

There is little debate about the proposition that, in principle, even highly heritable traits can be changed. Debate arises concerning the questions of whether such changes are possible in practice and, if possible, whether such changes would be accompanied by unacceptably high costs.

Researchers engaged in molecular studies of human behavior and human diseases tend to be optimistic about the possibility of changing traits [48, 49]. Once the molecular or biochemical cause of a disease is determined, it may be possible to develop a treatment involving either gene therapy (delivering the non-mutant form of the gene to the patient) or environmental therapy (delivering or countering the effect of the altered gene product) in order to correct the biochemical problem.

An analysis of the variability of genetic conditions suggests that this optimism may

underestimate the complex etiology of genetic diseases. Consider MDI. Suppose that MDI is discovered to be due to a mutation in a gene involved in a biochemical pathway that results in the decreased concentration of a chemical in the brain required for normal behavior. It is possible that, despite the fact that a chemical is involved, MDI is triggered by environmental factors like stress that lower the concentration of the chemical from a low but tolerable value to an intolerably low one. In this case, counseling involving, for instance, stress management techniques could possibly be a more appropriate therapy than one involving gene therapy or medication, which might be accompanied by undesirable side effects.

Now suppose that the etiology of MDI is even more complex; the condition diagnosed as MDI might arise from several distinct causes. Some individuals diagnosed with MDI might carry the mutation making them susceptible to particular environmental influences. Others might be affected by a different mutation, one which will manifest itself in essentially all environments. Still others might have a condition that is entirely environmentally induced. It may well be the case that as genetic mapping techniques improve, the dissection of complex behavioral conditions like MDI, schizophrenia, and alcoholism into separate diseases, each with its own etiology, can be accomplished. (We are assuming for the sake of argument here that all cases of these diseases are genetic. We doubt that this is so.) In this eventuality, specific treatments might be needed for individuals with specific forms of the disease.

But even in the case of a disease caused by a single genetic mutation that is expressed in all environments, it may not be a simple matter to find a treatment or cure. Although the exact amino acid change in hemoglobin that causes sickle-cell disease has been known for over three decades [50], there is still no cure and none of the treatments for the disease are based on the knowledge of the molecular basis of the disease [51]. Given the rapid progress in molecular biology, there is more optimism now that characterization of disease genes can lead to treatments. In some cases, such as the recently discovered gene for amyotrophic lateral sclerosis, the identification of the function of the gene product immediately suggests possible means of intervention [52]. However, this is a relatively unusual example, and the speed with which knowledge of the genetic basis of a disease can be translated generally into treatments or cures is highly uncertain.

Researchers involved in non-molecular studies of the biological basis of behavior are usually less sanguine about the possibility of change. Some have expressed the opinion that evidence for a genetic basis for a behavior indicates that it will be very difficult to change that behavior [53, 54]. Since, according to researchers, complex behavioral traits result from the effects of many genes [39], it is difficult to see how knowledge of the gene products will be of value in developing biological or chemical interventions. Another argument that is used by some who work in this area is that behavioral traits are the product of optimal Darwinian natural selection, and, thus, could be changed only by environmental changes that might have severe negative consequences for society [18].

Even if one accepts (as we do not) the high estimates of heritability reported for such traits as intelligence (≈ 0.8), it does not follow that the trait is difficult to change. A high heritability simply means that very little of the variation in the trait from one individual to another is the result of differences in the environments experienced by that population of

individuals. No conclusion can be drawn about how the genes would respond to a different environment. In fact, studies by Scarr and Weinberg [55] and by Schiff et al. [56] have shown that changing family environment (race or social class) can have a substantial effect on I.Q. scores.

It has been argued that, although changing the environment may affect performance on intelligence tests, such environmental changes can do little to eliminate inequality because they will simply raise the performance levels of all children to the same degree, leaving differences intact [34]. This argument ignores the possibility of a genetic-environmental interaction. Even if the lower performance by girls on the SAT mathematics tests is due to sex differences in hormones or in brain structure, this does not mean that in every educational environment girls will achieve less than boys. In recent years there has been a great deal of progress made in determining the types of environments in which the mathematical potential of girls can be realized [57, 58].

Nor does the fact that a trait has been subjected to evolutionary pressures mean that it is unchangeable or that it cannot be changed without causing harm to either the individual or society. A species is exposed to only a limited variety of environments throughout its history, and the process of natural selection can operate only within this range of environments. Although one could make the argument that the genes responsible for a trait that has been subject to evolutionary pressures are in some sense optimal for the environments encountered at some point in the history of the species, we have no idea how these genes would interact with the current environments.

Perhaps the basic fallacy underlying the nature-nurture debate is the assumption that if a human behavioral trait is "genetic," it will, of necessity, be much less malleable than an "environmental" one. It is our contention that the potential malleability of a trait depends on the details of the development of that trait and on our state of knowledge of it. Knowing only that the trait is genetic, or even that it is the result of a single gene mutation, does not provide enough information to assess its malleability. PKU [59] and Tay-Sachs Disease [60] are both caused by single gene mutations. PKU, however, is readily treated by a modification of the diet, while Tay-Sachs Disease remains refractory to treatment.

Traits that are "environmentally determined", i.e., show the same expression irrespective of the genotype, also vary in the degree to which they can be modified. Many vitamin deficiency diseases are easily treated by supplying the missing vitamin. However, children raised in poverty who were subject to deprivation and abuse may develop psychiatric problems as adults that are very difficult to alleviate. And children who were exposed to environmental toxins such as lead may suffer from biochemical or structural changes that are essentially irreversible.

SOCIAL POLICY IMPLICATIONS OF GENETIC BEHAVIORAL STUDIES

We have presented arguments critical of the views that (1) genetically controlled or highly heritable traits are unchangeable, (2) even if these traits can be changed, they should not be changed because the existing forms are the result of Darwinian selective pressures, and (3) genetically-based traits are necessarily more difficult to change than environmentally-based ones. However, these arguments do not address the philosophical

and ethical question of whether society should attempt to intervene in the development of human behavioral traits and abilities and the intertwined economic and political question of whether society should devote its resources to effecting the change. Many of the traits that are the focus of discussion involve inequalities. African-Americans score lower on I.Q. tests; women score lower on certain advanced mathematical tests at the upper range of test scores. Should our society allocate funds to foster equality in mathematics between the sexes by learning how to improve the math performance of women and to change social conditions so that girls receive as much encouragement at home and in school as boys in mathematics? Or would those funds be better spent on exploiting the talents of the boys who already score highest on the aptitude tests (assuming there was not enough money for both purposes)?

Consider an analogy with genetic diseases. Many of these diseases affect only a tiny proportion of the population, and some are not fatal or even severely disabling, yet great efforts are made to understand their etiology and to find cures and treatments. Shouldn't comparable efforts be made to investigate and possibly quantify the effects of disadvantageous environments on I.Q. and mathematics test performance? In our highly technological society, which is becoming increasingly dependent on an educated work-force, the substandard performance of a large proportion of people of a particular gender or ethnic group means that the potential contribution to society of millions of people is being lost. We would argue that systematic studies of environmental factors such as inadequate diet, unsuitable pedagogical techniques, and prejudicial attitudes about the intellectual ability of women and minorities are at least as essential as the search for genetic factors that might result in poorer performance on the tests.

The question of whether society should intervene is a philosophical, economic, and political one. It cannot be decided by resort to scientific arguments. We have argued here that there is no foundation for basing that decision on the knowledge that the trait has a strong genetic base or has environmental origins since genetic traits can be as malleable or non-malleable as environmentally affected traits.

Consider two children who do poorly in school. In one case, the school failures may be due to a deprived childhood, poor early nutrition, or other aspects of the child's background. In the other, it may be due to the specific genetic make-up of the child (given the educational environment). In neither case, can we predict how much the intellectual performance of the child can be changed by intervention. Nor is there any basis for deciding that one type of educational program would work for child 1 and not for child 2. If we wished to improve educational performance, a reasonable strategy might involve trying alternative educational techniques. Of course, it is possible that a gene might be found that is responsible for an intellectual deficit and that studies on the gene and its product might give clues to possible biochemical intervention. But, even if such a gene did exist, the strategy based on educational and other environmental interventions would remain appropriate. Moreover, given the present level of biological knowledge, even taking into account the extraordinary rate of progress in the last few years, we believe that an understanding of genetic components of educational performance is very far off indeed.

Even if the optimism of the molecular researchers is justified and the program of identifying genes that cause disease and social pathology based on this knowledge of

genetics is successful, we believe the program itself is problematic. Suppose, as is likely, this program is successful in finding treatments for certain well-characterized diseases. Then there will be strong pressures to regard this program based on a "technological fix" as the solution to the problem of all conditions which are thought to arise from the actions of a single gene or a few genes [61]. These pressures will come from the researchers themselves who will seek expanded funding for a research field that has shown considerable success. It may also come from conservatives who do not believe social problems can or should be ameliorated by governmental social interventions.

There is little doubt that the biotechnological program will ultimately result in treatments and cures for many genetic diseases. However, this program, if carried out to the exclusion of other means of ameliorating social problems, will be of limited use. If, as we have suggested, MDI is actually a label given to a constellation of traits with different etiologies, the search for a biotechnological treatment or cure may be of benefit to only a fraction of those people with MDI. The search may be more useful, in fact, in identifying the class of people for whom biological or chemical interventions would not be useful.

Alcoholism may be an even more instructive example of the possible negative consequences of the biotechnological approach. On the basis of the present state of knowledge, we believe that it is not unlikely that some forms of alcoholism may be correlated with a genetic predisposition, while other forms are purely environmental. If research on alcoholism is focused exclusively on the genetic forms, people with the environmentally induced forms of the disease may not be helped. Equally troubling, however, is the possibility that if it is recognized that some forms of alcoholism are genetic and others are environmental, there may be a tendency on the part of society and of people with alcoholism themselves to blame those who have the environmentally induced form of alcoholism. According to this view, people with the environmentally induced form, unlike those with the genetic form, should have much more control over their behavior.

FREE WILL AND GENETICS

As the alcoholism issue illustrates, the belief that people bear no responsibility for aspects of their behavior that are genetically influenced can be correlated with a genetic fatalist perspective. These aspects are regarded as being beyond their control. People are thought to be more responsible for non-genetically influenced behavior because it is believed that such behavior can be changed. In this section, we discuss the relationship between free will, genetic traits, and societal attitudes towards people whose behavior is considered to be "deviant" by the majority of the population. It is our contention that knowledge that a behavior is genetically rather than environmentally determined (or *vice versa*) is essentially irrelevant to deciding whether a person bears responsibility for that behavior.

In discussing their study on the genetic basis of male homosexuality, Bailey and Pillard [20] argue that, if it can be shown that homosexuality has a genetic basis, then people would be more prepared to accept homosexuals and not discriminate against them. This argument appears to be based on the following premises: (1) if homosexuality is not genetic, then either (a) people have chosen of their own free will to be homosexuals, or (b) homosexuality is an illness brought on by such environmental factors as abnormal

family interrelationships; and (2) if homosexuality is genetic, then it is fated; potential homosexuals have no choice over their sexual orientation. (This latter argument would also apply to homosexuality if individuals are born gay because of intrauterine environmental effects.) If (1a) is true, then people who believe that homosexuality is unnatural could argue that homosexuals have chosen a deviant path. If (1b) is true, then homosexuality might be treated in the same manner as other nongenetic psychiatric illnesses. If (2) is true, then homosexuality might be considered to be "natural" and, therefore, should be accepted as part of the normal range of human behavior.

There are several problems with this argument. First, consider the analogous argument for alcoholism. The implications of premises (1a) and (1b) are completely parallel, but those of (2) must be modified. In this society, even people who believe that alcoholism has a genetic basis do not regard it as a "natural" or "normal" condition; alcoholism is considered by many to be a disease. Thus, showing that homosexuality has a genetic origin would not in itself be sufficient to convince many people that homosexuality is "natural" and that homosexuals should be treated as the equals of heterosexuals. In fact, during the Nazi era, part of the justification for the extermination of homosexuals as well as Jews was the claim that their "traits" were genetic and, thus, a threat to the "racial hygiene" of the German nation.

Second, as we have argued in this paper, there is no reason to believe that environmentally controlled traits are any more malleable than genetically controlled ones. We are not taking a position on the philosophical doctrine of determinism; a philosophical determinist would say that all of our actions are determined and that free will does not exist. It is our position that if one is not a philosophical determinist, then it is contradictory to believe that we possess free will regarding (and are thus responsible for) behavior that is environmentally controlled, but that we lack free will regarding biologically controlled behavior. For example, one person might be able to control angry rages brought on by a hormonal imbalance due to an altered genotype more easily than another might be able to control the same type of rages brought on by years of suffering as an abused child.

Third, suppose that subsequent research shows that, although homosexuality is genetic, it is incompletely penetrant (i.e., some individuals with the genotype are not homosexuals). In fact, the Bailey-Pillard study itself shows that individuals with identical genetic makeup (identical twins) are only 52% concordant for homosexuality. What is the explanation for the other 48% who are not concordant? Assuming that the "homosexual genotype" exists and is present if a man is gay, then one possible explanation is that the straight cotwin chose to be straight of his own free will. Another is that environmental forces (e.g., societal pressure) were strong enough to overcome the natural homosexual tendencies in the cotwin who became straight. Thus, Bailey and Pillard's own data can be used to argue that, if there is a genetic component to homosexual behavior, there is still substantial play in the development of that behavior.

If it is proved that either of these explanations is correct or that male homosexuality is not genetic at all, would Bailey and Pillard then argue that it is acceptable for society to regard these men as deviant because their behavior is not determined by their genes? Clearly they would not. We agree with Bailey and Pillard that discrimination against homosexuals is wrong. But it is wrong for reasons that involve ethics and morality. The

"naturalness" of behavior, the flexibility in the development of a behavior, the desirability of changing the behavior, and the responsibility of an individual for his or her behavior rarely have anything to do with whether the behavior is environmental or genetic in origin.

CONCLUSION

In this paper, we have discussed the various positions on the implications of behavior genetics research for social policy. We have stressed the crucial importance for these discussions of a correct understanding of the concept of a genetically determined trait, the definition and meaning of heritability, and of the relationship between these characterizations of traits and the malleability of those traits. While we have seen remarkable advances in molecular genetics in recent years, we are still far from being able to use the results of research in this field to effect changes in the manifestations of behavioral traits or even to determine the malleability of those traits. Further, the complex interaction of genetics and environment, which has been emphasized by the most thoughtful geneticists, will always make the application of scientific results to such issues as education and so-called deviant behavior problematic.

Policy decisions on these issues depend on more than the feasibility of genetic intervention. Of necessity, they will reflect the philosophical, social, political, and economic views of both decision makers and the general public. We believe that too often these decisions are presented as questions that can be resolved in an objective fashion by relying on scientific results. Such a presentation can conceal the interests of the parties making the decision to the detriment of those whom the decisions affect. Despite these reservations, a deepened understanding of issues of genetics and behavior should contribute to an appreciation of the wealth of human diversity and of the untapped potentiality inherent in each of us.

In our view, an understanding of the relationship between genetics and the environment shows that an individual is no less responsible for her biologically based behavior than for her environmentally based behavior. Even those studies that appear to argue most strongly for a genetic basis for one or another behavioral trait can be interpreted to provide evidence of the importance of personal choice and the environment in the assumption of personality characteristics or the development of specific abilities.

As we enter an era when more and more genes associated with behavioral traits will be discovered, it is crucial that scientists learn how to present the results and discuss their implications (or lack of implications) in a responsible way to the public. If not, we fear, these studies may continue the history of the use of biological theories of human behavior for the purpose of justifying discrimination and inequality.

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