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THEORETICAL NOTES

Heritability and Biological Explanation

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Modern neuroscientific and genetic technologies have provoked intense disagreement between scientists who envision a future in which biogenetic theories will enrich or even replace psychological theories, and others who consider biogenetic theories exaggerated, dehumanizing, and dangerous. Both sides of the debate about the role of genes and brains in the genesis of human behavior have missed an important point: All human behavior that varies among individuals is partially heritable and correlated with measurable aspects of brains, but the very ubiquity of these findings makes them a poor basis for reformulating scientists' conceptions of human behavior. Materialism requires psychological processes to be physically instantiated, but more crucial for psychology is the occasional empirical discovery of behavioral phenomena that are specific manifestations of low-level biological variables. Heritability and psychobiological association cannot be the basis for establishing whether behavior is genetic or biological, because to do so leads only to the banal tautology that all behavior is ultimately based in the genotype and brain.

Biological Explanation

A "News and Comment" headline in *Science* (Barinaga, 1991) asked the question, "Is Homosexuality Biological?" *Nature* (Maddox, 1991), in a "News and Views" headline, put it this way: "Is Homosexuality Hard-Wired?" Recent advances in the technology of neuroscience and genetics have produced an exponential increase in the ability to detect neurological and genetic correlates of complex behavior. The debate concerning homosexuality is but one example of a contemporary fascination with whether a wide variety of behaviors and psychological syndromes might be characterized as biological. Other topics of interest have ranged from schizophrenia, which is by now widely considered to be a biological disease (Lieberman & Corrigan, 1992), to criminality (Mann, 1994), to intelligence (Davis, 1993), and to practically every form of psychopathology (Guze, 1989).

Concern with the biological basis of behavior is a modern manifestation of a more familiar problem with which it has a great deal in common: the so-called nature-nurture question, which has occupied behavioral scientists for more than a century and has shown no recent signs of abating (e.g., Herrnstein & Murray, 1994). Evidence for genetic components of human variation in intelligence, personality, and psychopathology (Plomin, 1991) continues to accumulate, and behavior genetic findings are now being extended to realms previously thought to be more exclusively in the domain of family and culture, including social attitudes (Martin et al., 1986), religiosity (Waller, Kojetin, Bou-

chard, Lykken, & Tellegen, 1990), vocational aptitudes (Moloney, Bouchard, & Segal, 1991), and marital status (McGue & Lykken, 1992; Turkheimer, Lovett, Robinette, & Gottesman, 1992).

It is generally assumed that the genetic and biological questions are related, especially in the sense that the heritability of behavior is taken as evidence for its biological nature. In a replication of Bailey and Pillard's (1991) study of homosexuality in male twins, for example, Whitam, Diamond, and Martin (1993) concluded, "In both studies [i.e., Whitam et al.'s and Bailey & Pillard's] the rates of concordance for MZ [monozygotic] twins is sufficiently high as to suggest a strong biological basis for sexual orientation in MZ and DZ [dizygotic] twins" (p. 203). If this inference is correct, evidence for a strong biological basis also exists across the entire range of behaviors for which heritability has been documented. Indeed, it is difficult to identify any aspect of human behavior for which some degree of heritability cannot be detected. Must one now consider a biological basis for divorce? And what is at stake if one does? An answer to such questions entails analysis of the role of biological causation in the development of behavioral outcomes and the evidential value of heritability in determining the kinds of roles played by biological factors.

Weak and Strong Biologism: The Empirical Consequences of Materialism

Today, it seems everyone is a materialist. As Dennett (1991) put it: "Materialism of one sort or another is now a received opinion approaching unanimity" (p. 106). In the most obvious sense, materialism requires one to recognize all mental and social phenomena as characteristics of the physical self, most notably of the brain. This sense of the word *biological*, which I refer to as "weak biologism," is strictly a matter of materialist

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philosophy: There is no need to conduct empirical science to locate thoughts, feelings, and beliefs in the body and brain. In contrast to weak biologism is the assertion that certain psychological phenomena are biological as a matter of empirical science. In this common sense usage, which I call "strong biologism," the word *biological* denotes behavior that is well-represented as a process at a biological level of explanation. An empirical discovery that a high-level human behavior is biological entails identification of a structurally or functionally localized biological process that explains some large part of the high-level phenomenon.

It is useful to begin the discussion of weak and strong biologism by considering computers, for which the issues are straightforward and uncontroversial. When setting out to write a computer program to accomplish some complex task, one does not begin with the first very first electronic event the computer must accomplish ("Wait for input to the keyboard. If a *p* is pressed, interpret it as . . .") and then proceed, step by tiny step, to the last ("Carry any value over 10 to the tenths column, then send the following command to the printer"). Programs written in this way would take a lifetime to complete. Instead, one divides the task into simpler modules, writes programs for the modules (which may be divided into submodules), then combines them to perform the more complex task. Indeed, this computational structure is built into the high-level programs used by most end users. The commands executed in the high-level language refer to programs written in lower-level languages, which in turn refer to machine language programs that actually produce the very complex strings of commands for the physical elements of the computer. High-level computer programs, therefore, accomplish two goals: They are a means of inducing systematic, highly complex activity without specifying each individual action of the simplest components, and they are a way of describing the behavior of highly complex computational systems.

The hierarchical structure of computer systems becomes especially important when one has to debug them, because errors can usually be fixed effectively at only one level in the programming hierarchy. If a SAS program to conduct a factor analysis requests the incorrect type of factor rotation, the error would be very difficult to fix by adjusting the machine code generated by the SAS program. Trying to fix the problem with a soldering iron would be even worse. On the other hand, if a bad Pentium chip was causing arithmetic errors, it would be ineffective to rewrite SAS programs to adjust for the error. It is worth stating the above scenario as a principle: Although high-level computer programs necessarily consist of lower-level—and ultimately electronic—elements, it does not follow that it is always best to conceptualize computer programs at the lowest available level of analysis.

This kind of distinction, which is routinely made when dealing with machines, shows the way to a meaningful understanding of biological explanation as an empirical discovery rather than as a simple assertion of materialism. Proceeding from computers to a hypothetical biomedical example, consider two mute individuals. The first is a 70-year-old man who has had a stroke in Broca's area of the left hemisphere, visible as a small dark area on a brain image. The second is a 25-year-old man who has joined a religious order requiring silence as a means of with-

drawal from the ordinary intercourse of the world. It seems natural to describe the stroke patient's muteness as biological and the monk's as psychological. What do these attributions mean? It is not simply that the aphasia is "in" the brain, because the monk's decision presumably resides there also. Instead, the difference involves the nature of the structural relationship between a neurological representation of the condition and a psychological account of it. Aphasia may be recognized behaviorally, but its cause is localized in the stroke lesion, at a neurological level of analysis. Although the neuropsychology of stroke is in fact extremely complex, it is nonetheless reasonable to say that the cause of the aphasic patient's muteness is the lesion in the left hemisphere. One would be suspicious of explanations involving the patient's motivation to remain mute. Religious devotion, although undoubtedly residing in the brain, has no localized representation there. Neurological explanations of religious devotion would seem overly reductionistic, although there is no reason to doubt that intense neuroscientific investigation might reveal neurological correlates of monkdom.

Stroke, localized neurologically, is manifested behaviorally, but there is no closed set of behavioral symptoms that is uniquely associated with neurologically defined stroke. For the monk, the situation is reversed. Religious devotion is defined at a behavioral level of analysis. Although it is undeniably related to processes in the brain, there is no closed set of neurological events that is equivalent to it. Psychobiologists may legitimately attempt to identify lower-level processes related to religious devotion, but in this biotropic era two cautions must be sounded: (a) Discovery of modest associations between behaviorally defined processes and lower-level biological structures is not cause for surprise and, in fact, it is theoretically necessary, as a consequence of weak biologism and (b) discovery of psychobiological associations need not undermine the impression that high-level variables, such as religious devotion, are indeed "psychological." What would undermine the sense of religious devotion as a psychological construct is a discovery that there was a class of silent monks whose silence was the specific result of a well-defined (structurally or functionally) brain system. Such a discovery would be an instance of strong biologism, and it would be a legitimate bombshell. Successful discrimination of these two kinds of psychobiological explanation is the key to a sensible and balanced relationship between psychology and biology in the explanation of human behavior.

One of the ironies of the contemporary fascination with the biological status of psychological phenomena is that on the rare occasions when someone pauses to consider exactly how the distinction between biological and psychological phenomena might be characterized, the most common conclusion is that it doesn't exist at all. For example, the distinction between the biological and the nonbiological has been rejected by Greenberg and Bailey (1993, 1994), who began with the observation that weak biologism is empty of scientific consequences:

The discovery that a given behavior is biologically caused (a) gives us no new information, for we already know, by hypothesis, that all behavior is biologically caused, and (b) cannot be used as a basis upon which to distinguish that behavior from any other, again because all behavior is biologically caused. (1993, p. 246)

If everything is biological, they contended, the biological ba-

sis of a behavior can hardly be used to make fine distinctions about etiology or moral responsibility (Greenberg & Bailey, 1994). Although the weak biological basis of behavioral syndromes may be irrelevant to the social, ethical, and scientific considerations about them, the question of how behavior is related to neurological or genetic processes—whether, to take Greenberg and Bailey's starting point, a homosexual disposition is determined more like an aphasia or more like a religious commitment—has real content. If a behavioral syndrome is biological in only the weak sense, then it is not simply a matter of time until neuroscientists discover the neurological structures that determine it, because there are no neurological structures that determine it, despite the fact that in the weak sense all behaviors are constituted by neurological elements. (Harre, Clarke, & DeCarlo, 1985, illustrate this point nicely by way of explaining why there is no "physics of carpets." Although carpets may have characteristic physical components, what makes something a carpet is its relationship, at a much higher level of analysis, with the world of human beings. No amount of physics would ever lead to an explanation of why some objects are carpets.)

The analogy with computers clarifies that the contrast between the biological and the psychological refers to a dimension rather than to a dichotomy (Gifford, 1990; van Praag, 1986). The choice is not simply between neurological (or machine language, biochemical, or genetic) structures and those at the highest level of analysis, like lifestyles and cultures; there are also intermediate levels of analysis that are relatively closer to the level of the wires but which are not located there in any straightforward sense. Therefore, the hypothetical reduction of monkdrom to a neurological structure is not the only possibility. It might turn out, for example, that a species of monkdrom was the cultural manifestation of a particular temperament, defined at a lower, but nonetheless suprabiological, level of analysis.

It is unfortunate that the strong and weak senses of biological explanation are often confounded, usually to the detriment of psychological explanation. Approaches to biological explanation that do not maintain the distinction between weak and strong biologism usually founder on the weak sense, which quickly leads to the conclusion that biological causation of behavior is universal. If weak biologism is accepted uncritically as the basis for theorizing about biological explanation of behavior, it can quickly lead to unjustified and all-encompassing reductionism in the explanation of complex behavior, as is illustrated below.

Weak and Strong Biologism in Psychiatry

It has been said that psychiatry is the major contemporary arena of applied philosophy (Toulmin, 1988). In the meantime, biological psychiatry is in its heyday. Biological psychiatry is so widely accepted today that it is difficult to find justifications of it: Even its flagship texts, like Nancy Andreasen's (1984) *The Broken Brain*, contain little analysis of the theoretical underpinnings of the approach. An exception, and probably the strongest theoretical endorsement of the school of biological psychiatry, is an article by Samuel Guze (1989) titled "Biological Psychiatry: Is There Any Other Kind?" In it, Guze defended the assertion that "there is no such thing as a psychiatry that

is too biological" (p. 315). Psychiatry might reasonably be called too biological, Guze suggested, if it could be shown that "few if any of the states and conditions that constitute the focus of psychiatry are the result of differences in the development or physiology of the brain" (p. 316) or, conversely, if "all or most of our patients develop their disorders primarily, if not exclusively, through normal learning processes that are independent of brain variability" (p. 316). Guze asserted that neither proposition has found any empirical support and so concluded, "psychopathology is the manifestation of disordered processes in various brain systems that mediate psychological function" (p. 317).

It is important to consider the nature of the empirical findings Guze (1989) marshalled for his argument. It is true that psychiatric disorders have not been shown to be independent of brain variability in the sense that some statistically significant associations are generally found between manifestations of psychopathology and brain variables. Such findings might be called "psychobiological associations." But these same studies have just as decisively *not* shown that biological variables are exclusively or specifically implicated in the development of psychopathology. Schizophrenic patients, on average, have larger cerebral ventricles than normal people, but not all schizophrenic patients have large ventricles, and some mentally healthy people have them.

Much rests on this distinction. What did Guze (1989) mean when he said that psychopathology is a "manifestation" of disordered processes in the brain? His next sentence states, "Psychopathology thus involves biology" (p. 317). In the sense of weak biologism, this statement is undeniable. But if the assertion is that the best way to understand humans is always to lift the cover off and study the wires, it is a radical proposal indeed. It is one thing to use psychobiological associations as a justification for psychobiological research or as an argument against what Guze called the tabula rasa assumption of radical environmentalism. It is quite another to proceed from psychobiological associations to an assertion of the supremacy of biological methods over higher-level scientific (Guze characterized them broadly as cultural anthropology, sociology, and social psychology) and clinical methods, such as psychotherapy.

The software–hardware analogy is worth pursuing a moment longer in this context. Although it is possible to be an adequate programmer without detailed knowledge of the physical workings of computers, good programmers understand the technical aspects of their medium, because hardware constrains the performance of software and because software can be optimized for particular machines. Had Guze titled his article, "Why Psychiatrists Should Know Something About Neuroscience," there would be no basis for argument. It is nevertheless necessary to insist that it is entirely possible for psychiatry to be too biological. What would you say to a programmer, assigned to correct a factor analysis program that was misestimating the communalities of the observed variables, who started out with a pile of chips and a soldering iron?

Specific Genetic Etiology

Meehl (1972b, 1977) borrowed the term *specific etiology* from medicine to describe a particular species of biological—

and sometimes genetic—causation. According to Meehl, a specific genetic etiology (Meehl uses the term *genetic* in its more general etiological sense) of a syndrome is a causal factor that exerts some form of strong influence on the determination of the syndrome. Meehl's articles on the subject were concerned with two major themes. First, in opposition to the operationalist philosophy of science that was still prevalent in psychology at the time, Meehl showed that investigation of the etiology of a syndrome could be conducted prior to, or at least in conjunction with, definitional work on its observable configuration: It is mostly thanks to Meehl that this point seems uncontroversial today. Second, Meehl specified exactly the forms of causation he counted as specific etiology. In the strongest sense, a specific etiology takes the form of a pathognomonic sign, or a necessary and sufficient cause; in some of the weaker senses, a factor may be considered a specific etiology if it potentiates all other causes or potentiates a single cause regardless of the values of other factors.

Meehl's formal accounting of criteria for the determination of specific etiology can be seen as an attempt to formulate a set of rules for determining when behavioral syndromes would be better addressed at a lower level of analysis. Meehl's conclusion was essentially that the association of the high-level process with the lower-level one must be strong and theoretically based. It is important to note that Meehl specifically excluded simple psychobiological associations, that is, cases in which the probability of a syndrome given the presence a explanatory factor is simply greater than the probability of the syndrome when the candidate explanation is absent. If the candidate factor was more easily observable than the phenomenon it was seeking to explain, Meehl called it a fallible indicator—potentially useful in further specification of the syndrome but not a form of specific etiology.

Meehl also emphasized that even in a thoroughly deterministic world, not all events have specific etiologies. Referring to work on the philosophy of causation by Mackie (1965), Meehl showed that no single event can be taken as the specific cause of an event, like a fire in a factory. Although it might be said that the fire was caused by a short circuit, it is neither the case that all short circuits lead to fires nor that all fires are caused by short circuits: rather, what is meant is something like the following:

The event we focus on as "the cause" of the factory fire, here the particular event of the short-circuit in the particular fuse box in which it happened, is part of a complex of circumstances prevailing in the factory at that time, both affirmative and negative, such that, if the other elements of the complex are imagined to be held fixed . . . then the fire would not have occurred if there had not been a fire in the fuse box. (Meehl, 1977, p. 48)

Describing this type of explanation as "perhaps the modal type of both medical and social science," Meehl (1977, p. 48) concluded that it is not a form of specific etiology.

This sensible view is in opposition to the opinion that most of the syndromes studied by psychologists or psychiatrists will turn out to have big causes (Goodwin, 1984). The causes of divorce or monkdrom are almost certainly like the causes of factory fires: diverse, nonspecific, and highly contextual. Nevertheless, just as a comparison of mean levels of electrical deterior-

ation in factories with and without fires would probably yield a significant difference, it would certainly be possible to demonstrate differences between the brains of divorced individuals and the happily married. The proper conclusion from such studies would not be that fires have an electrical etiology or divorces a biological one; rather, one could conclude that electrical and biological problems are nodes in highly complex systems that sometimes result in fire or marital disruption.

Conclusion

The contemporary fashion for biological explanation bears many similarities to the behaviorism of the previous generation, with one important difference. From the outset, behaviorism was proposed and defended on the basis of philosophy of science and mind (Ryle, 1949). It has always been obvious that behavior could be modified by learning and reinforcement; the point of behaviorism is that it is possible to build a complete science of behavior in terms of learning, and furthermore that it would be desirable to do so (Rorty, 1982). Biologism, in contrast, is more often proposed as a contingent empirical result than as a philosophical doctrine. Proponents of biological explanation do not generally defend the claim that one can or ought to reduce complex behavior to biological terms. Instead, they report the results of experiments that purport to demonstrate that behavior has turned out to be biologically based. However, to the extent these experimental results are necessary consequences of the physical instantiation of psychological processes, they provide only the weakest support, analogous to the support a behaviorist could claim on the basis of an experiment showing that behavior is "influenced" by schedules of reinforcement. This confounding of the philosophical and empirical underpinnings of biological explanation has provided biologism's adherents with a nearly impregnable defense: They claim biological explanation as a strong empirical result on the basis of experiments that depend solely on the necessary instantiation of behavior in the body and brain.

Properly analyzed, determining whether a syndrome is biological is more than an exercise in establishing tautological psychobiological associations; it is a matter of determining whether or not a syndrome has a specific genetic etiology. Much controversy in theoretical clinical psychology and psychiatry involves disagreement about where to locate schizophrenia, affective disorder, homosexuality, temperament, and personality disorder on the dimension running between aphasia and marital status. A complete evaluation of all the indicators that might be brought to bear—medication response, positron-emission tomography (PET) results, psychotherapy outcome, cross-cultural studies, Meehlian taxonomies—would be an interesting topic for another article. In the second part of this article, I attempt to give an account of one indicator of biological status, heritability, as it is represented in contemporary human behavioral genetics.

Heritability

What It Means to Be Genetic: The Empirical Consequences of Evolution

Everything is heritable. This astounding fact—at odds with the predictions of early opponents of behavior genetics, who

held that most important human functional characteristics would not be heritable, as well as of the behavior geneticists themselves, who had set out to divide the domain of human behavior into the heritable and the nonheritable—represents one of the greatest challenges for behavioral scientists of all persuasions. Irving Gottesman and I have suggested that $h^2 \neq 0$ be enshrined as the first law of behavior genetics (Turkheimer & Gottesman, 1991). Although it is possible to think of exceptions to the universality of heritability, the exceptions do not form a class of behaviors for which genes have no relevance. In fact, quite the opposite is true. Most low-heritability behavioral traits—the use of speech, for example—have been so strongly selected by evolution that they no longer show significant variation among individuals, but their basis in the genotype is uncontroversial. There are few traits that are nongenetic in the more commonplace sense that they are determined exclusively by environmental causes. Religious affiliation comes to mind, but religiosity, as opposed to affiliation, is heritable (Waller et al., 1990).

Evolutionary theory is an even more universal basis for the life sciences than materialism is for contemporary philosophy, and in hindsight it may be difficult to understand why the idea that genotype covaries with behavior evoked such bitter controversy. Just as materialist philosophy guarantees the presence of psychobiological associations, evolution and Mendelian theory guarantee heritability, unless one is willing to protect some aspects of humanity from the universal influence of evolution. The second half of this article focuses on one important but unnecessary source of disagreement: the tendency of both behavior geneticists and their opponents to mistake heritability as an instance of strong biological explanation and as evidence that the complexity of human behavior is in danger of being reduced to relatively simple genetic mechanisms.

Weak and Strong Genetic Explanation

The vast majority of human behavioral characteristics that are related to genes do not fit straightforward Mendelian models of transmission, which describe the transmission of traits determined by single or, at most, several genes, following patterns determined by the familiar concepts of segregation and dominance. Therefore, the investigator studying the genetics of depression, extroversion, or divorce must solve two fundamental problems. The first is a familiar one: separating genetic and cultural transmission, which is usually accomplished through the study of twins or adoptees. Second, the genetic models must account for the fact that the genetic transmission of psychological traits does not follow any obvious Mendelian pattern. The offspring of an extroverted father and an introverted mother do not segregate into introverted and extroverted types but are continuously distributed around an intermediate value.

The well-known genetic models of twin and adoption research apply the principles of quantitative genetics to solve the latter problem (Falconer, 1960). The key assumption of these models is that not one, but many genes are contributing to the genetic transmission of the trait in question. Each of these many genes contributes a small amount of variance according to unobserved classical Mendelian laws. Summing over these many contributions results in a binomial process, which, as the number of

genes becomes large, produces a normally distributed phenotype and offspring intermediate to parental values.

What does the heritability of divorce demonstrate about its etiology? According to the statistical models that were described above, it suggests that a large number of segregating genes each contributes a small effect to the quantitative value of a continuous trait that is related to the likelihood that a person will eventually become divorced. As to how the genes contribute to divorce, the model has very little to say. It could be that genes contribute to variation in impulsivity, which might make it more difficult to remain monogamous. Genes may be related to variation in alcoholism or depression, and these may be related to marital difficulties; genes might be related to homosexual inclinations that could disrupt a marriage. Of course, to one degree or another, genes are related to all of these behaviors and to a long list of other traits that might contribute to the success or failure of a marriage.

Population genetic demonstrations of heritability, therefore, are clearly incompatible with a view that the likelihood of developing a behavioral phenotype is completely unrelated to genes. But does anyone seriously hold this belief? Heritability is the genetic analog of psychobiological association: It serves only to remind us of what we should already know, that is, we cannot be completely free of our genetic endowment. It is a necessary consequence of weak biologism, of the simple fact that our psychological selves are physically embodied in the context of evolution. In and of itself, heritability should be neither surprising nor controversial. But in terms of strong biologism, the answer to the question of whether heritability reveals anything about biological or genetic etiology is an unequivocal “no”: Heritability reveals nothing about the difference between aphasic individuals and monks. Indeed, the heritability of religiosity is in all likelihood higher than the heritability of aphasia.

So, one cannot assume that discovery of heritability is a first step in the direction of discovering the specific genetic etiologies that underlie it, because just as it is possible for high-level computer programs to be based in lower-level languages without necessitating lower-level mechanisms that can be addressed by a programmer, it is possible for behavioral phenotypes to be heritable in the absence of any specific genetic etiology. This somewhat paradoxical outcome of behavior genetics—everything is heritable but transmitted by mechanisms that are largely unknown and potentially unknowable—sometimes leaves behavior genetic theorists in a difficult rhetorical corner. Just as Guze's (1989) confounding of weak and strong biologism led him to defend the assertion that psychology “involves” biology, behavioral geneticists often defend the weak assertion that genes “influence” behavior. In a summary of behavioral genetics, for example, Plomin (1991) used the word *influence* in conjunction with some form of *genetics* 17 times in the first five pages of the chapter.

It is ironic that the scientists most keenly aware of the limited etiological implications of polygenic heritability are exactly those genetically oriented psychopathologists who are committed to the discovery of the specific genetic etiologies of mental illness. Fuller and Thompson (1978) put it as follows:

Monogenic theories suggest major biochemical pathways which can be uncovered, whereas polygenic models suggest a complexity of

chemical interactions probably intractable to exact study. Thus if most behavior traits must be fit to polygenic models, we may be left only with statistical analyses of such problems as how many genes are involved and the specification of the almost infinite number of interactions between them. Such mathematical exercises seem to us to have only trivial importance and, furthermore, to be of small interest to most biologists and psychologists. (p. 438)

Faraone and Tsuang (1985), before embarking on a review decisively favoring multifactorial (MFT) models, put it this way:

The mode of inheritance has substantial implications for etiological research and clinical practice. A conclusive demonstration that a single major locus is involved in schizophrenia would hold the promise that a relatively direct biochemical pathway accounts for the psychophysiology of the disorder. If a multifactorial polygenic model describes the mode of transmission, the search for a simple biochemical pathway is likely to be less fruitful. (p. 44)

Faraone and Tsuang then cited Kidd (1981), who put it as follows: "Moreover, the [MFT] model can provide little insight into the genetic system. The very nature of the model relegates genetic factors to an amorphous pool of small indistinguishable components" (p. 374). Meehl (1972a) expressed doubt about the compatibility of polygenic models of inheritance with specific genetic etiology:

One understands fairly clearly what it means to conjecture that a "big-effect monogene" is the specific etiology of a disease But once we have excluded that simple situation, the *very meaning* of the phrase "specific etiology" begins to "fuzz up." (p. 376)

Statistical methodologies do exist for discriminating among single major locus, polygenic, and mixed models (Kendler & Kidd, 1986; McGue, Gottesman, & Rao, 1983; Morton & MacLean, 1974; Risch, 1990b; Risch & Baron, 1984). The literature on this topic is technical and, to a large extent, focused on animal characteristics for which the problem is to discriminate between transmission controlled by one gene as opposed to several. When applied to more complex human characteristics, however, it quickly becomes apparent that the various errors of measurement inherent in the quantification of complex human phenotypes threaten to overwhelm the subtle signals that the statistical methods have been designed to detect. Thus, there is (a) Eaves's (1983) demonstration that the difference between scoring a test by counting the number of correct responses and more sophisticated methods based on item response theory are larger than the differences between single gene and polygenic modes of inheritance, (b) McGuffin and Huckle's (1990) tongue-in-cheek demonstration of a recessive gene for attending medical school in Great Britain, (c) Weiss's (1992) dead-serious and well-documented assertion of a major gene for general intelligence, and (d) Kurnit, Layton, and Matthyse's (1987) demonstration that a single major locus combined with stochastic processes of development can closely mimic a polygenic system, and so forth. This state of affairs is unfortunate, because according to the analysis presented here, determining how many genes contribute to a phenotype is much more important than estimating the percentage of phenotypic variance for which they collectively account.

An Example: Criminality

Distinguishing between weak and strong genetic influence reduces much of the heat generated by the nature–nurture debate. Consider an example: The tension between genetic influence and psychological explanation is at its most dramatic and controversial when it comes to violence, antisocial personality, and other forms of behavior that may be collectively referred to as *criminality* (Goldsmith & Gottesman, 1996). Is criminality biological? Are some people born with a genetic predisposition to become criminals? Should one imagine a world in which the criminally prone are treated for their condition with drugs or surgery? Or is the very notion of a biology of criminality racist, reductionist, and antidemocratic?

The fractious scientific and moral issues inherent in the biogenetics of criminality are far easier to discuss sensibly if one distinguishes between weak and strong biologism. Consider the scientific and ethical consequences of two propositions:

Proposition A: The likelihood that a person will become a criminal is correlated, through unknown but certainly multifarious, nonlinear, and interactive biological and social pathways, with large numbers of genetic loci.

Proposition B: Some people are compelled to commit violent criminal acts because of the specific actions of a small number of genes that produce identifiable brain damage.

The literature on the genetics and biology of violence can be summed up by stating that there is a substantial and well-replicated body of empirical evidence for Proposition A and practically none for Proposition B. Behavior geneticists have repeatedly asserted a substantial genetic component to most forms of criminal behavior (Goldsmith & Gottesman, 1996; the empirical data have been meta-analyzed by Mason & Frick, 1994, who found moderate heritabilities, and Walters, 1992, who suggested that if findings are properly summarized, heritabilities are much smaller; my own view is that the heritability of adult criminality is very unlikely to be zero, and other than that its magnitude is indeterminate and of little theoretical interest). Neurobiologists (Dolan, 1994) catalog a host of psychobiological associations of antisocial behavior with electroencephalogram (EEG) abnormalities, autonomic functioning, and neurological testing. The only evidence relevant to Proposition B follows from a report of an association between aggressive behavior and a mutation related to monoamine oxidase (MAO) metabolism in a single family (Brunner, Nelen, Breakfield, Ropers, & van Oost, 1993). But as is usually the case with molecular genetic associations with complex behavior, when examined closely, the finding is much more supportive of Proposition A than B. The mutation in question is extremely rare, too rare to account for more than a tiny fraction of violence in the population. It is also associated with low intelligence and learning disability (Alper, 1995).

Although the evidence for the biogenetics of criminality consists almost exclusively of the weak biologism variety, when the ethical and scientific consequences of the findings are anticipated, they are usually discussed as though a well-specified genetic etiology of violence were just around the corner. Brennan and Mednick (1993) concluded a review titled "Genetic Perspectives on Crime" (finding, as usual, moderate heritabilities

for most forms of criminal behavior) with these somewhat chilling words:

This review has demonstrated that genetic factors can and do influence certain types of criminal behavior What are the implications of this knowledge? First, biological factors must be added to the list of causes of crime; it is through heritable biological structures and processes that the genes exert their influence. Second, we must try to identify the specific biological mechanisms through which heritable predispositions toward criminal behavior are expressed. By identifying these mechanisms, we can learn how to successfully treat and prevent criminal behavior. (p. 25)

This paragraph crystallizes the unhappy consequences of confounding heritability with strong biologism. Criminality, like pretty much everything else, is heritable. This means that, on the day people are born, they do not all have an equal probability of becoming criminals. It means that if a deacon adopts the son of a gangster, the child will have a greater chance of following a life of crime than his adoptive sibling sprung from the deacon's loins, and it means that if you are studying the effects of child-rearing practices on adult criminality, you had better include adoptees in your research agenda. However, it does not mean that there are "specific biological mechanisms" underlying criminality any more than there are specific machine language structures and processes underlying malfunctioning factor analysis programs, or specific brain structures and processes underlying the silence of ascetic monks.

Does anyone really suppose that unintelligent, unattractive, greedy, impulsive, emotionally unstable, or alcoholic people are no more likely than anyone else to become criminals or that any of these characteristics could be completely independent of genetic endowment? There is nothing surprising, much less morally repugnant, in the notion that the likelihood of becoming a criminal is not completely independent of the dynamic, interactive multitude of ways in which our genotype influences our behavior. But the notion of "specific biological mechanisms" for criminality—with its ominous implications of genetically defective criminals, compulsively violent, who can be treated with chemical or surgical interventions—is deeply troubling. It is troubling not because it assumes that biological endowment bears some distant relationship to eventual manifestations of criminality but, rather, because it violates one's fundamental sense that the causal paths leading to criminality—biological or otherwise—are more complicated than that; and there is good reason, as the reader will see, to insist on the complexity of the genesis of human behavior.

Molecular Genetics and Specific Genetic Etiology

The Human Genome Project will be crucial to the future of behavioral genetics. Linkage- and association-based molecular genetic methods can detect specific alleles associated with behavioral outcomes (Lander & Schork, 1994). Early molecular and statistical methods assumed that the relevant alleles were genes of large effect; more modern methods offer the power to detect quantitative trait loci (QTLs) accounting for less than 10% of the variability in outcome. Gottesman (1997) sees QTLs as the crucial missing link between population genetics and specification of developmental models of behavior.

Data from twin studies for complex traits can be used to screen for "candidate traits" that can become the focus for connecting to candidate genes. The high heritabilities of traits such as general cognitive abilities and diseases such as schizophrenia tell researchers where to invest their efforts first. (p. 152)

But do they? MFT threshold models of heritability, with their assumption that genes of large effect do not contribute to behavioral phenotypes and their crucial dependence on genetic and environmental variability, seem to offer little reason to expect that there will be useful information about the etiology of complex behavior in the outcome of the Human Genome Project. Plomin (1990) has compared the task of molecular genetics to finding "many tiny needles in the haystacks" (p. 187) but notwithstanding the pessimistic connotations of the needle-in-the-haystack metaphor, Plomin remains confident that molecular genetics will make important contributions to the study of high-level behavior (cf. the much more cautious expectations expressed in Risch, 1990a): Plomin summarized his book *Genetics and experience* (1994) with seven hypotheses, of which the seventh is, "Specific genes that affect experience will be identified" (p. 163).

The distinction between weak and strong genetic explanation is crucial to an evaluation of Plomin's conjecture. It is important to be very clear about the basis for skepticism about its outcome: It is not that linkages and associations with complex behaviors are unlikely to be discovered—quite the opposite—but that there is no theoretical reason to assume that linkages and associations will form a basis for a coherent genetic theory of complex behavior. If one accepts fundamental genetic theory and realizes that genes are a necessary component of all development, and if one reinforces the confidence in this conclusion by observing the ubiquity of heritability for variation in behavioral phenotypes, the existence of small covariations between alleles and behavior becomes a theoretical certainty, not a risky empirical prediction. As before, it is important to maintain a distinction between a theoretical assertion that individual genes somehow contribute to complex outcomes, which is undeniable but uninteresting, and the practical question of whether there is anything worthwhile to be learned about the etiology of divorce by studying the remote molecular genetics of marital status (it passes Gottesman's test of substantial heritability), which would be amazing if true but is very unlikely to be so.

Needless to say, opponents of behavioral genetics have taken a somewhat different outlook on the prospects of the Human Genome Project for the study of human behavior. If Plomin is somewhat unduly optimistic about the prospects for the molecular genetics of complex behavior, theorists on the other side of the issue have been unable to contain their *schadenfreude* over repeated failures to replicate early reports of genetic linkages and associations with major psychopathology. These new challenges were highlighted by John Horgan (1993; see also Alper & Natowicz, 1992; Billings, Beckwith, & Alper, 1992). Although Horgan's foremost theoretical concerns appear to lie with opposition to biological determinism and eugenic breeding programs in humans, the bulk of the article is concerned with two themes: discrediting the empirical results of twin studies, especially the Minnesota study of twins reared apart (Bouchard, 1994) and celebrating the notable failures of molecular geneticists to repli-

cate preliminary reports of linkages and associations with complex human phenotypes.

Horgan's (1993) error was essentially the converse of Plomin's: Where Plomin used the success of population-based behavioral genetics to promote an unrealistic optimism about the molecular genetics of complex human behaviors, Horgan used the failure of molecular genetic studies of behavior to discredit the validity of twin and adoption results. In a table, Horgan compiled a "Lack-of-Progress" report, including some of the typical targets of behavior genetic research: crime, manic depression, schizophrenia, alcoholism, intelligence, and sexuality. For each, he paired substantial heritabilities estimated from twin studies with the well-known failures to replicate molecular genetic findings. This contrast is interesting and should lead to the important conclusion that human phenotypes such as these are unlikely to be influenced by genes of large or even moderate effect, but it does not demonstrate that there was something wrong with the twin studies.

The distinction between weak and strong biogenetic explanation of complex behavior provides a sensible middle ground for expectations about the contributions of molecular genetics to the study of complex human behavior. Population-based behavioral genetics has demonstrated that genotype and behavior can be expected to covary. Although the epigenetic developmental pathways linking gene products to complex behavior will in general be almost unimaginably complex, modern molecular genetics has made it possible to detect small covariations between alleles and behavior that span the complexity of the causal network, much as modern brain imaging has made it possible to detect small psychobiological associations that span the vast gap between neuronal action and complex cognitions. Such associations are real and potentially interesting, but they remain correlations—and small ones—not evidence of substantial causal pathways between individual alleles and complex behavior or evidence of genes for extroversion or intelligence or evidence that future scientific efforts will be most productively applied at a genetic level of analysis (Strohman, 1997). If the history of empirical psychology has taught researchers anything, it is that correlations between causally distant variables cannot be counted on to lead to coherent etiological models.

Conclusion: The Way Out of Nature–Nurture

Everything is biological; everything is genetic. Some behaviors are biological in a stronger sense in that they are manifestations of a circumscribed process at a lower level of analysis; some behaviors are genetic in a stronger sense in that they are the consequence of well-specified genetic mechanisms involving relatively small numbers of genetic loci. Two of the most important contemporary developments in the behavioral sciences—the discovery of nearly universal genetic influence on behavior and the assertion of equally pervasive biological causation in the same domain—are linked in their prodigious empirical successes and notable theoretical shortcomings. Materialist philosophies of mind and evolutionary biology have demonstrated that human psychology is not exempt from biological instantiation, thus guaranteeing two empirical consequences: If studied hard enough, all behavior and mentation will turn out

to be constrained by genetic endowment and correlated with biological variables.

Experiments with guaranteed outcomes tempt scientists to conduct them again and again, especially when each instance of the guaranteed result appears to hammer another nail in the coffin of a long-standing intellectual opponent. Genetic and biological reductionists, in their eagerness to win ancient debates with environmentalists, sociologists, psychoanalysts, and other top-down theorists, have used to great advantage an incorrect account of the nature–nurture problem, according to which the important issue is whether psychological phenomena are, more or less equivalently, genetic or biological. If all that needs to be demonstrated is that psychology involves biology or that genes influence behavior, the reductionists will win every time. But the very one-sidedness of the reductionists' victory may prove their undoing if they are not careful because now that it is known that everything is heritable and biological, it is also known that there is little to learn about a trait by establishing its heritability or biological basis: It simply confirms what materialist philosophers have already explained.

Complex human behaviors of the kind that have interested psychologists—beliefs, intentions, emotions, personalities—do not have localized biological or genetic causes in the sense that stroke lesions cause aphasia or a single gene causes phenylketonuria. What troubles social scientists opposed to biological reductionism in psychology is not simply that biogenetic theories associate behavior with genotypic or neurological variation, but rather that the biological explanations are oversimplified in that they try to explain complex behaviors as if they were aphasias. Divorce *per se* is unlikely to yield to neurological or genetic analysis. The difficulty is not that divorced individuals are not constituted by neurological or genetic processes, because obviously they are; the problem is that too many levels are being skipped between marital behavior and neurons or genes. Divorce is a coherent process at a social level of analysis, but it is utterly incoherent neurologically and furthermore depends for its definition on relationships with other people and institutions that do not reside in the brain at all.

When the nature–nurture debate is reformulated in terms of determining the most appropriate level of analysis for complex phenomena, its outcome is once more in doubt, and the task of characterizing complex phenomena on a dimension running from biological to psychological can resume on a sound theoretical basis. I will close with two recommendations, one very practical and the other theoretical, for future discussions of nature–nurture. The practical suggestion is that all sides of the issue should stipulate the first law of behavioral genetics and refrain from further discussion of whether or not the heritability of anything is equal to zero. As a reader of behavioral genetics, keep a pencil by your side and lightly excise everything that either asserts nonzero heritability or attempts to explain it away: Much space could be saved in our journals (even those containing sophisticated multivariate genetics or well-informed opposition) if this recommendation were put into effect.

The central issue addressed in this article—whether the empirical facts of psychobiological association and heritability can be squared with the relevance of top-down scientific methods for generating psychological explanations of complex human behavior—has been addressed in diverse areas of applied phi-

losophy ever since it became clear that extraphysical principles were unlikely to provide plausible accounts of the behavior of complex systems. Philosophers of biology, for example, have wondered whether traditional whole animal biology must inevitably be reduced to molecular biology. Philosophers of mind have struggled to explain how the phenomenal qualities of mind can derive from the physical properties of brains, and those concerned with the ancient problem of free will have confronted the implications of human physical instantiation for the intuition that people are free and self-determining (Dennett, 1984).

Philosophers are less likely to agree than even social scientists, but a broad area of consensus has emerged about how these questions might be answered. In the free-will literature, this point of view is known as *compatibilism*. Compatibilist accounts of complex systems accept that complex phenomena are composed of simpler physical elements but insist that physical instantiation need not threaten the aspects of human psychology people hold dear. Human complexity is the key to compatibilist accounts of behavior. Although people may be composed of deterministic physical units in some ultimate theoretical sense (the sense of weak biologism), the causal chain linking the physical units to complex human behavior is, for most practical purposes, infinitely complex, and it is that complexity that humans experience as the psychological determination of our behavior and that necessitates psychological scientific methods to study it. So, whereas human dignity may be justly threatened by oversimplified biological accounts of behavior, there is no need to fear the generic assertion that people are ultimately biological or genetic: We knew it all along.

Here, for example, is Dennett's (1984) summary of the relationship between biology and free will:

What we want when we want free will is the power to decide our courses of action, and to decide them wisely, in the light of our expectations and desires. We want to be in control of ourselves, and not under the control of others. We want to be agents, capable of initiating, and taking responsibility for, projects and deeds. All this is ours . . . as a natural product of our biological endowment, extended and enhanced by our initiation in society. (p. 169)

When Dennett specified what would constitute a real threat to human freedom, he limited himself to interpersonal coercion and political bondage; however, one can recognize that neurological and genetic diseases pose similar threats in that they replace the exquisite complexity and sensitivity of normal human causation with a short-circuited and linear compulsion.

Nature and nurture are compatible and not only in the commonplace sense that genes and environment are both necessary for development. Genes and other biological structures constitute complex behavior, but the behavior of complex organisms cannot be derived from the biogenetic atoms of which it is composed. If radical behaviorism temporarily obscured the fact that all behavior shows genetic influence, behavioral genetics has decisively revealed the truth. Researchers have also discovered that a few behaviors that were once thought to be most succinctly described as psychological or sociocultural phenomena, like paresis and infantile autism, turn out to be better described at a lower, and more biological, level of analysis. These rare instances of strong biologism have not been founded on broad assertions of heritability or psychobiological association

but rather on the specification of the particular processes linking specific genetic etiologies to their behavioral manifestations. In the meantime, the great preponderance of human behavior—heritability and psychobiological associations notwithstanding—has resisted reductionistic biological and genetic analysis. If I am correct, it will continue to do so.

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