THE UNDERTHEORIZED ENVIRONMENT: SOCIOLOGICAL THEORY AND THE ONTOLOGY OF BEHAVIORAL GENETICS

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ABSTRACT: *Growing interest in the genetic contribution to human behaviors has led to the growth of the field of behavioral genetics. The authors consider the concept of "environment" in behavioral genetics and argue that sociology is in a unique position to evaluate and specify a theoretically robust concept of environment. Building on insights from classical and contemporary social theory, the authors argue that the study of genetic influence on behavior needs to incorporate sociological understandings of contextual effects. The authors propose five points for evaluating behavioral genetic studies' conceptualization of environment and use those points to evaluate three exemplary recent studies.* Keywords: genetics; environment; behavior; ontology

When, in June 2005, Oprah Winfrey told a Johannesburg crowd that she felt "so at home" there because "I went in search of my roots and had my DNA tested, and I am Zulu," observers had every right to be skeptical, as some were (BBC News, June 15, 2005). Although the BBC and the South African *Mail and Guardian* (Lemmer 2005) pilloried the unlikely specificity with which Oprah claimed her roots,[1] most American news outlets accepted the genetic basis of her contemporary comfort (not to mention the accent: "'I'm crazy about the South African accent,' she said. 'I wish I had been born here.'") without serious question. Although DNA probably plays some role in her comfortable experience in South Africa, social and historical factors are presumably much more important in this case.

Winfrey's pronouncement is only a particularly visible example of the increasing attention potential genetic origins for behavioral outcomes are enjoying in the popular press (Finkler 2000). Of particular recent importance is the likelihood that many human behavioral traits are, in part, genetically based. This possibility is the purview of the emerging discipline of behavioral genetics (Burgess 2005).

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A recent article, for example (Cochran, Hardy, and Harpending forthcoming), argues that higher-than-average intelligence among Ashkenazi Jews may be the result of selective pressures toward intellectually demanding work at key historical moments. Interviewed by *The New York Times*, behavioral geneticist Steven Pinker expressed concern that the research is "politically incorrect" (Wade 2005) but is not quoted as interrogating its theoretical assumptions, several of which seem to contradict important sociological and even evolutionary principles, such as "overestimat[ing] . . . the speed of genetic change within a Darwinian framework—where it is measured in many millennia" (Floud, Wachter, and Gregory 1990: 4–5).

Genetics is one of the major scientific accomplishments of the twentieth century. The growing evidence of the importance of genes in influencing individual variation in disorders from depression to schizophrenia and attributes from IQ to obesity and risk-taking behavior (Plomin et al. 2001) has created fascination and increasing interdisciplinary interest in the role of genes in determining behavior. Biologically influenced analyses of human behavior have taken two distinctive but related approaches. The first approach attempts to explain panhuman traits using evolutionary theory. Panhuman traits are behaviors shared by all humans, such as language and violence. The second approach, behavior genetics, concentrates on the role of genetic diversity in creating individual differences. In this approach, the focus is on the 15 percent to 30 percent of the genes that vary between individuals. For further discussion of this approach, see Burgess (2005: 1–19).[2] The general public has also not missed this trend, as popular media eagerly cover the possibility that gender, race, and differences in behavior ranging from aggressiveness to appearance, sexuality, and aesthetic taste may be "hard wired" in individuals' genetic code.

Although behavioral genetics routinely acknowledges and studies the role of environmental influences on the expression of genetic tendencies, its conceptualization of "environment" is usually relatively cursory. Unfortunately, sociology, the disciplinary voice best positioned to provide insight about the importance and specification of environment in behavioral genetics research, remains mostly silent or, worse, cedes the intellectual ground to genetics' undersocialized conception of human behavior. As a discipline, sociology seeks to understand and explain social context and its influence on human behavior. It is therefore imperative to include the insights of sociological theory in understanding the concept of the environment in behavioral genetics research.

In this article, we argue for a sociologically robust understanding of the environment in behavioral genetics. We present challenges posed by extant sociological work to the current practice of behavioral genetics. Our hope is that future research in behavioral genetics will seek to conceptualize environment in a more sociologically appropriate way, thereby better delineating the relationships between genetic and environmental influences on human behavior. This task benefits sociologists and geneticists alike by offering each the insights of the other. We approach this project as interested skeptics, cautious about the increasingly large claims made for the genetic basis of human behavior but intrigued by the possibilities genetic data and principles could lend to our understanding of

social behavior. We seek to develop a theoretical approach that allows for future progress on the shared projects of sociology and behavior genetics while recognizing the uncertainty demanded by the standards of sociological theory.

We begin with a discussion of the concept of environment and a sociological definition of the term. We then review the state of the art in behavioral genetics. We continue with a discussion of principles drawn from sociological theory that motivate our claims about environment. We use these principles to develop five key points we consider essential to consider in employing a sociologically robust theory of the environment in behavioral genetics. We then consider three recent exemplary studies' inclusion of environmental elements and evaluate their conceptualizations. Finally, we examine the threats to construct validity and the sources of remaining uncertainty in current behavioral genetics research.

We begin with the assumption, shared by most behavioral geneticists, that most behaviors are the result of complex, mostly unobserved, combinations of genetic and environmental causes (Turkheimer 2000). Our concern is that, through undertheorization, the environmental elements of these combinations have been shortchanged by the emerging research tradition. We hope that this article will illuminate and guide the small and growing stake of sociology in behavioral genetics research, both by stimulating sociologists to participate in the field and by encouraging geneticists to learn from sociology's ideas of social context.

ENVIRONMENT: A SOCIOLOGICAL APPROACH

Sociology's greatest contribution to understanding the environment of genetic expression may be its sensitivity to the hierarchical and contextual nature of human behavior. The concept is brought home in Marx's famous polemic (the point is apt even if the language is anachronistic):

> A Negro is a Negro. He only becomes a slave in certain relations. A cotton-spinning jenny is a machine for spinning cotton. It becomes *capital* only in certain relations. Torn from these relationships it is no more capital than gold in itself is *money* or sugar the price of sugar. (Marx 1891: 207; see also Rubin 1975 for an extended analysis)

Fundamental to this theoretical position is the claim that phenomena—behaviors, meanings, arguments, feelings, and so forth—gain definition and meaning only within contexts.[3] Thus, our first sociological contribution to behavioral genetics must be in understanding that "disorders," behaviors, and traits are themselves the product of social experience (see Durkheim and Mauss 1903/1963).

Second, more than a century of sociological theory has established that social systems characterized by more "freedom" may not actually provide greater individual autonomy, at least in direct correspondence. This insight—which we characterize as the "paradox of reform"[4]—appears in numerous contexts, from Lewis Coser's (1956) discussion of dissidents' strengthening of the core community to Weber's (1978) concern with increased democracy underwriting stifling bureaucracy, Merton's (1949) distinction between manifest and latent functions,

Foucault's (1977) examination of the irony of prison reform, and Lessig's (1999: 97) discussion of the durability of restrictive covenants (for more examples, see Freeman 1972; Meyer 1956; Sewell 1992; Swidler 1986). In the general case, the paradox of reform states that decreased social control in one domain may induce increased social control in other domains. This has two implications for behavioral genetics. First, researchers should not assume that "freer" societies are characterized by greater expression of genetic predispositions. Second, and more generally, a given cause may have paradoxical (that is, contradictory) effects; researchers should not assume that a genetic (or environmental) influence affects behavior in a direct, linear fashion.

Third, across several sociological domains, we have examples of "small differences that matter" (Card and Freeman 1993, 1994). Once more, this insight comes from diverse theoretical sources. In economic sociology, Card and Freeman (1993) show that early small differences between Canadian and U.S. labor law set in motion an iterative process that produced large differences in union membership years later. The life course literature (see, e.g., Elder, George, and Shanahan 1996; Shanahan and Hofer 2005) demonstrates that relatively "small" experiences in individuals' lives may similarly grow in importance as time continues. Risk factors, from diet and exercise to loneliness and social isolation, have been shown to have effects on major health outcomes decades later. Unlike the previous two points, this is a relatively new tenet in sociological theory; indeed, sociological classics often violate the principle, as Durkheim (1893/1984: 12–19) did in *The Division of Labor in Society*.

WHAT IS ENVIRONMENT?

In the historical, evolutionary sense, genes and environment are expressions of one another. That is, because of the evolutionary mechanism theorized to produce variations in genotype, an individual's genetic makeup is the result of environments experienced many generations before. DNA, in this sense, is a recording mechanism capable of carrying information gleaned at one historical period and reporting its lessons at another. Similarly, environments are produced and selected, in part, by people with genetic predispositions. The question, therefore, of which influence predates the other is neither answerable nor helpful (Freese and Powell 2003; Horwitz et al. 2003).

In the more immediate sense, though, we can think of environment as the set of outside stimuli available to influence an individual through any mechanism, whether conscious or unconscious, whether immediate or delayed. This is an extremely intricate construct—indeed, it necessarily encompasses the entire corpus of sociology. Because of that intricacy, the temptation to avoid the complexity by substituting very specific environmental influences—severe childhood maltreatment (Caspi et al. 2002), for example—is great. But sociological research has demonstrated that current responses to stimuli may be conditioned by a host of other stimuli, from cultural norms to family background, childhood memories, and more. Indeed, these environments are nested within one another: the physical environment in which a gene or quantitative trait locus (QTL) is set is, itself, set

within additional environmental levels, all of which may condition that gene's effects. Substituting specific environmental exposures for a general consideration of the environment therefore makes it very likely that environmental influences will be missed.

A suitable consideration of environment encompasses the social environment and, therefore, includes the concepts of society and social system and the multiple ways these terms can be operationalized. The "environment" for genes and QTLs is typically defined in ways entirely different from—and far narrower than—the social environment. However, when attempting to understand individual development, or the different ways in which individuals can respond to varying environmental situations (Burgess 2005), the "environment" in which genes are expressed should not be limited to measurements of maternal nutrition or the experiences of twins. The environment must be conceptualized as interactive, multidimensional, and nested. For example, in their analysis of the causes of child maltreatment, Burgess and Drais-Parrillo (2005: 305–30) show how different levels of environmental factors interact to influence behavior.

THE GENE/ENVIRONMENT INTERACTION VIEW

The canonical view of genetic expression—whether in behavioral genetics or in other genetic research—is that individuals' genetic code (the genotype) provides the potential for observable effects (the phenotype). When an individual reaches this potential, the gene's effect is said to have been expressed.[5] As the geneticist J. M. Thoday (1965) put it, "genotype determines the potentialities of an organism. Environment determines which or how much of those potentialities shall be realized during development" (p. 94). Colloquially, "genes load the gun, but environmental factors pull the trigger" (Bulik, as quoted in Neumark-Sztainer 2005: 29). The *environment* refers to factors outside the individual that may affect the likelihood or extent of the gene's expression.

The simplest form of this principle can be viewed in the case of genetic and environmental influences on individuals' height. Individuals are said to have a potential height, often estimated by entering the biological parents' heights into a simple formula to account for the parents' and child's gender.[6] Environmental effects such as nutrition, physical activity, and more contribute to the likelihood that an individual will achieve his or her potential height (Grilo and Pogue-Geile 1991; Plomin 1990). Although environments are important, "genes drive experience," and as such, "a good theory of the environment can only be one in which experience is guided by genotypes that both push and restrain experiences" (Scarr and McCartney 1983: 425).

Recent approaches have improved on the canonical gene-environment (GE) view, introducing a more nuanced concept of the GE interaction. In such models, we can no longer talk about the separate "contributions" of genetics and the environment to individuals' phenotypes. Rather, genes are said to moderate individuals' response to environmental inputs or the environment is said to moderate or even shape the expression of individual genotypes (Carey 2003: 294–6). These interactions are typically modeled using standard statistical interaction terms

(Jaccard and Turrisi 2003), an approach that may miss other kinds of interactions such as a confluence of necessary and sufficient conditions (see, e.g., Ragin 2000).

However, in evolutionary time, experience also drives genes, as we note above. Furthermore, and more importantly, GE interactions represent only one theoretical mode for determining behavior. There may be very simple genetic (G) or environmental (E) causes, which do not require any input from the other; genes may interact with other genes (GG) and environments with other environments (EE); and there may be causal chains of indeterminate lengths combining these elements. These chains may reach great lengths in explaining relatively complicated phenotypes such as modern human behaviors (see Bronfenbrenner and Ceci 1994; Ceci 1990). Some studies have presented evidence of such complex chains; Turkheimer et al. (2003), for example, show that family social class affects the degree to which IQ is heritable,[7] a claim we could classify as EEG, with one environmental measure moderating the degree of environmental influence on the expression of a genetic tendency. Similarly, a (hypothetical) genotype predisposing an individual to rebellion against parental guidance would be expressed in widely varied phenotypes based on its position within a long chain of genetic and environmental effects.

In theory, sociologists' use of genetics in behavior models should be more accurate because they better define and care more about environment than do behavioral geneticists and neurobiologists. However, this depends on sociologists successfully incorporating theoretical sociology in their understandings of genetics. There are very few sociological studies that have done this, and in fact, Shanahan and Hofer (2005) note that there are surprisingly few empirical examples of environment measured as social context in studies from any discipline that investigate GE interaction.

The current view of complex GE interactions driving behavioral phenotypes assumes a particular kind of relationship between individual and society. Specifically, it assumes that individuals exist presocially and either create or are constrained by social systems. This assumption is not testable—essentially, it is an issue for the philosophy of the subject—and is directly at odds with several productive and empirically fruitful lines of sociological research (see Kurzman 2004 for a discussion). One need not believe, with Durkheim (1912/1995), that individuals are essentially the product of social systems to reject the notion of a fully formed presocial individual interacting independently with its environment.

METHODS OF GENETIC RESEARCH

Although sociologists are very interested in understanding the social moderation of the level of heritability for a specific behavior and despite the many advances in modeling in the field of behavioral genetics research, they are still limited by the methodological approaches they can use to explore this relationship. This is because traits involving behavior have a complex genetic basis. Single genes do not determine most human behaviors. Genes in these multiple gene systems are called QTLs because they result in continuous (quantitative) distributions of phenotypes that lie behind susceptibility to common disorders (McGuffin, Riley, and

Plomin 2001). The problem is that we lack a full understanding of how these QTLs work. A sociologist cannot incorporate such models when trying to understand how social environment affects genetic expression when even proximate understandings of environment and their interaction with multiple genes is not completely understood by geneticists themselves. And although headway has been made in identifying many QTLs, translating these findings into explanations of human behavior is an entirely different task (McGuffin et al. 2001).

Those sociologists who do study environmental effects, like their colleagues in other fields, rely on estimates of genetic and environmental effects from twin studies for this very reason. Quantitative genetic research (e.g., twin and adoption studies) has provided the most solid genetic findings about individual differences in human behavior (Burgess 2005: 9–10; McGuffin et al. 2001). They are consistent in their conclusion that genetic variation contributes substantially to phenotype variation among individuals for all behavioral domains. Psychopathology, cognitive abilities and disabilities, and personality are the best studied areas. These all have been assessed by recent "model fitting" and have resulted in two interesting findings. One is that of the behaviors studied, almost all show moderate to high heritability—usually to a larger degree than shown by many of the common physical diseases. The other is that, notwithstanding the fact that environment does play a role, it is generally of the nonshared type. That is, instead of making people similar to their relatives, environmental factors tend to make people more different from their relatives than their genotypes would suggest (McGuffin et al. 2001).

Notwithstanding the fact that direct measurement of biological markers ("biomarkers") is the gold standard, the vast majority of sociological considerations of genetic effects has used twin modeling methods (e.g., Alford, Funk, and Hibbing 2005; Duncan, Boisjoly, and Harris 2001; Guo and Stearns 2002; Kohler, Rodgers, and Christensen 1999). Udry's (e.g., 1988, 2000) use of biosocial models for gender construction and sexual behavior may be the only exception to this rule.[8] Using the knowledge that different types of genetically related individuals such as monozygotic (MZ) and dizygotic (DZ) twins have different and known levels of genetic relatedness, variation among individuals' measured behavior can be partitioned into the parts due to heritability, shared environment, and unshared environment[9] (Plomin and Daniels 1987; Turkheimer and Waldron 2000). This approach, however, is controversial because it fails to account for GE correlations and interactions (Rutter and Silberg 2002; Turkheimer 2006; Vreeke 2000).

The twin-study approach, while attractive for its parsimony and for its ability to factor out, if not explicitly account for, environment, leaves open some important threats to validity when applied in sociological domains. The U.S. twinning rate, for example, climbed 65 percent between 1980 and 2002: a large increase in the number of DZ twin births attributable, at least in part, to environmental factors such as increasing maternal ages during pregnancy, the use of reproductive technologies, and maternal diet (Martin et al. 2003: 21ff; Steinman 2006). We know little about how families of these DZ twins may differ from the families of singleton children. Given these causes, they could be characterized by older parents;

more professional parents who delayed childbearing for career advancement (Gustafsson 2001); parents who more desired children, motivating their use of reproductive technologies; or by other differences when compared to parents of singletons and of MZ twins. Furthermore, the increasing availability of genetic information to families means they are more likely to be aware of their twins' zygosity and (consciously or unconsciously) expect greater commonality between MZ than between DZ twins.[10] These possibilities and more are theoretical variables that could be included in a model explaining degree and kind of twin variation.

Horwitz et al. (2003) present evidence of different patterns of sociability between MZ twins from those between DZ twins. They suggest that these microsocial factors constitute sufficient environmental difference to be wary of the assumption that twins grow up in common environments: an assumption crucial to the dominant twin-study design. Ironically, the theoretical logic underlying Horwitz et al.'s argument parallels closely that of an important article over a decade earlier (Plomin and Bergeman 1991), which argued that there were plausible genetic reasons for factors generally presumed to be environmental (see also Purcell and Koenen 2005)! Although there has been some controversy over the import of the Horwitz finding (Freese and Powell 2003), each of these factors (and potentially more) raises the possibility that twins' zygosity may be correlated with characteristics of their families and with their families' treatment of them. This is cause for caution in interpreting measures of heritability.

Both Horwitz et al. and their critics (Freese and Powell 2003) use the opportunity to argue that the enterprise of partitioning variance between genetic and environmental factors is theoretically bankrupt and should be replaced, although neither offers a competing theoretical framework for interpreting genetic evidence in social science. Turkheimer (2004) has similarly argued for limiting the use of twin and sibling models to testing theoretical hypotheses, as partitioned variance has proved relatively unconvincing.

We are not prepared to abandon the partitioning project entirely, although we believe the interplay between genetic and environmental factors is far more complex, iterative, and diachronous than typical studies assume. A promising direction for sociological theory would be an elaboration of action as a product of complex GE interaction chains. Unfortunately, then, most studies incorporating behavioral genetics have controlled for environment instead of conceptualizing it. This results in threats to validity in these studies.

SOCIOLOGICAL THEORY AND BEHAVIORAL GENETICS

In a useful article, Shanahan and Hofer (2005) review selected studies that report GE interactions involving social context. They organize the research into a typology that identifies four generic mechanisms by which social context moderates gene expression: contextual triggering, social context as compensation, social context as social control, and social context as enhancement.[11]

Shanahan and Hofer's critiques of gene-context interaction studies fall under four main themes: (a) the failure to consider social context from a life course perspective, (b) the inattention paid to the multifaceted nature of social

context, (c) failure to specify mediating mechanisms that link context with the immediate biological substrate, and (d) an overreliance on population-based statistical models, which require untenable assumptions to make statements about individual development. They contend that their critiques might contribute to explaining why few GE interactions have been observed despite widespread theoretical expectations to the contrary.

Based on our review of sociological theory and Shanahan and Hofer's (2005) critiques, we present a set of five criteria for evaluating measures of environmental effects in behavioral genetics. To be sociologically convincing, a theoretical understanding of an individual's "environment" should consider the following criteria:

1. It cannot simply assume that environment *constrains* genetic expression. Particular environments may very well be the "natural" or (particularly in humans) the strategic *production* of actors or groups. The strategic, agentic (i.e., as an *agent* capable of action) *production* of environment (Emirbayer and Mische 1998) makes human ecology different from, say, plant ecology, in which a plant may produce its own environment (say, shading its stem or creating water channels) but cannot be said to do so strategically. Genetic models that assume the fundamental actor is an autonomous individual simply reacting to a fully external environment must recognize that this assumption remains empirically undemonstrated and theoretically problematic.
2. Similarly, environment must be conceptualized as potentially enabling as well as constraining. Consider, for example, the concept of an individual's "genetic potential" for a trait, whether height or a less easily measured trait. The concept is endogenous to the theoretical framework, as it is impossible, even in theory, to measure such potential. Rather, the framework assumes there must be a genetic potential and develops a theory of environmental constraint. Because genetic potential is unfalsifiable, it must not be assumed in studies that evaluate environment's impact on phenotypes.
3. Both environments and genetic potentials must be understood as nested and cross-cutting in potentially complex ways. That is, environments interact with, and exist within, other environments. Furthermore, genetic potentials may exist at different levels; the genetic potential for basketball appreciation, for example, may be the complex product of potentials for aesthetic appreciation, social likeability, and so forth. The concept of "social geometry" (Black 1976; Simmel 1950) and more recently network and contextual analyses (e.g., Perrin 2005, 2006), which have enjoyed substantial empirical support, demonstrates that the interconnections between individuals and social structures can predict important variations in individual behavior without recourse to individual traits. This principle does not undermine the importance of individual traits (whether genetic or not) in predicting behavior, but it does require that analysts consider the potentially large independent effect of the context of behavior.
4. Because genetic and environmental influences may iterate over time, small differences may matter—that is, small and even immeasurable environmental

influences may have substantial influences on outcomes, particularly over multiple iterations. Even a simple trait such as height—generally considered to be among the most heritable of human traits—is the result of a GE interaction in which part of the environmental factor takes place before the measured height is achieved (Floud and Wachter 1982).

5. Because evolutionary time is very slow, change observed within historical time must be (mostly) the result of environmental change. However, precisely because of our previous point that conceptualizing GE interaction strongly means rejecting the idea of partitioning variance among genetic and environmental sources, this point does not rule out the likelihood that genotypes are necessary conditions for phenotypic changes observed in historical time.

Each of these points can be understood as increasing the level of uncertainty in the measurement and interpretation of genetic and environmental data. Although we maintain no general preference for uncertainty, the sociological research strongly supports more uncertainty than is generally expressed in behavioral genetic research. Of course, this uncertainty should not be assumed to mask only environmental origins for behaviors; the threats to validity we uncover here could plausibly be masking genetic causes as well.

APPLYING THE STANDARD: THREE CASE STUDIES

We now apply our theoretical standard to three exemplary studies that straddle the border between sociology and behavioral genetics. We undertake this exercise not as an attack on the studies or their authors but rather as substantive critique in the hope of building sociological insight into behavioral genetics. Each of the articles we select is innovative, applying behavioral genetic knowledge in new ways, to new questions, or to new disciplines; this work is of vital importance. Our critiques highlight these exemplary studies.

Our first case study is Guo and Stearns (2002; see particularly Table 8, p. 904), which seeks to estimate the environmental impact on children's likelihood of achieving their "genetic potential for intellectual development." This innovative article reconceptualizes the study of social inequality by understanding it as differential opportunities for individuals to achieve their genetic potential. Including a measure for the heritability of intellectual propensity, the authors reason, allows for a more direct measure of social systems' success at unleashing that genetic potential. Our concerns about the article do not extend to its innovation in the study of inequality. Rather, we concentrate on the theoretical underpinnings of the article's partitioning genetic from environmental factors to illustrate the assumptions inherent in behavioral genetic research.

The article hypothesizes that family social class constitutes children's environment and that poor environment restrains children from achieving their genetically based potential. By partitioning variance in children's achievement into hereditary and environmental factors, they seek to measure the effect of social policy on inequality in a more direct way than have previous sociological considerations. As indicators of family socioeconomic status (a presumed environmental

effect), they include measures of mother's education, the presence in the home of the biological father, and the child's race.

The analysis simplifies the environmental side of the proposed GE interaction by assigning children to either "good" or "bad" environments. The article thereby potentially increases its estimate of the genetic factor. Environments are rarely "good" or "bad," as individuals may exhibit resilience to "bad" environments because of protective traits or contexts (Luthar and Cicchetti 2000; Luthar, Cicchetti, and Becker 2000). Furthermore, some scholars have suggested that a small introduction to a "bad" environment may induce resilience (Wheaton 1990; see also Fergus and Zimmerman 2005 for a review). This is not simply a matter of statistical noise. Rather, it suggests that there may be GE or EE interaction chains that could alter the direction of an environmental effect. Environments that are "good" for individuals carrying one environmental or genetic trait may be "bad" for those carrying other such traits.

Guo and Stearns (2002) assume that the twins they study grew up in common environments—that is, that environment is therefore held constant while genetic similarity varies. They reason that children who grow up in a "good" environment are more likely to realize their genetic potential for intellectual development and that genetic potential for intellectual development is suppressed for those children who live in a "bad" environment. They assume not only that genetic potential is suppressed in a bad environment but that shared environmental influences would play a larger role in the intellectual development of the children. Therefore, the within-pair correlations due to shared environmental influences with respect to intellectual development would tend to be higher for those children in "bad" environments and the within-pair correlation due to genetic influences would be lower for children in the "bad" environment than for children in the "good" environment.

Although Guo and Stearns (2002) conceptualize children's environments as both potentially enabling or constraining of genetic potential (i.e., dichotomizing environments as either "good" or bad" for realization of genetic potential), their characterization of environments is oversimplified. Furthermore, their analysis is based on the ability to measure the degree of expression of genetic potential. Although they distinguish between level of genetic potential for intellectual development, which they do not measure, and realization of genetic potential for intellectual development, which they do, there remains a basic assumption that genetic potential is a measurable concept. They recognize possible endogeneity between genetic potential and environment when they cite work of Scarr and McCartney (1983), who argue that children tend to actively seek the right environment that match their genetic tendencies. They note that it is much easier for children living in affluent neighborhoods to find the right environmental niches to fill than it is for those children living in urban ghettos.

An important strength of the article is its recognition that environments and genetic potentials must be understood as nested and cross-cutting in potentially complex ways. This is exemplified in their complex twin design to measure genetic potential as well as their extensive measures of environment ranging from race of respondent to region of the country.

Guo and Stearns (2002) also note that because evolutionary time is very slow and that genes do not change over a relatively short period of time, change

observed within historical time must be (mostly) the result of environmental change. However, they see large changes in environment enabling the full expression of genetic potential that may have previously been constrained.

> Behavior that changes over a historical period seems to defy the explanation of genetic influences because genes do not change over a relatively short period of time. Although genetic potential does not change, the social conditions that regulate genetic potential do. When the social conditions change markedly over time, the amount of genetic potential realized could change markedly over the same period. For this reason, the remarkable historical changes in fertility and sexual behavior could well have an important genetic explanation even though social forces are still the driving forces. (p. 885)

No direct attention is paid to the idea that small environmental influences may have substantial influences on outcomes, particularly over multiple iterations, although such a conclusion could follow from the thinking evidenced in the paragraph quoted above.

Second, we consider Alford et al.'s (2005) recent study of the heritability of political orientations. Having argued previously that "[political] preferences and behaviors are at least partially shaped by evolutionary forces and therefore by genetic heritage" (Alford and Hibbing 2004: 707), Alford et al. present a classical twin study based on a large sample of MZ and DZ twins (the VA30K study; see Lake et al. 2000). Essentially, they argue that two persistent types of political orientations—"absolutist" and "contextualist"—are the product of largely stable personality traits that are, in turn, partially the expressions of genetic predispositions. The argument is attractive, as the authors point out, for its ability to answer some persistently vexing problems in political science: "absolutists and contextualists simply do not connect, and the result is frustration" (p. 166). Even more basic, "Why is a reasonably standard right-left spectrum so widely applicable cross-culturally and over time?" (p. 153).

A plausible answer to the latter question, of course, is genealogical instead of genetic: the contours of contemporary world politics, with remarkably few exceptions, have been set in the terms of classical political and moral philosophy, recast through generations of debates over values, procedures, and structures. One need not resort to presocial genetic predispositions to explain belief structures that match the modes of evaluation available to individuals.

Alford et al.'s study uses a classical twin design, partitioning variance in twenty-eight political attitudes among genetic, shared environmental, and nonshared environmental origins. As such, its validity rests on the same supports as do other twin studies. More than others, though, this study is forthright about assuming that genetic influences precede, and are then filtered through, environmental constraints: "To the extent that political ideologies are inherited and not learned, they become more difficult to manipulate. Conservative parents who try to make their children conservative by carefully controlling their children's environment are probably overestimating the importance of those environments" (p. 164).

Our final case study is Caspi et al.'s (2002) groundbreaking study of the relationship between genes and environment in producing youth violence. Because this

study received world-wide attention and is viewed as a scientific exemplar of the best ways to test GE interactions, we examine how closely it follows our five criteria for testing GE effects. Given the importance of this study to the scientific community and the fact that few studies have done similar research, evaluating this research on the bases of five of our criteria will provide a good evaluation of the state of behavioral genetics' conceptualization of environment.

Caspi et al. (2002) followed a large sample of male children from birth to adulthood (members of the Dunedin Multidisciplinary Health and Development Study[12]) to determine why only a portion of those children who were maltreated as children developed antisocial behavior as adults. They established that a functional polymorphism in the gene encoding the neurotransmitter-metabolizing enzyme monoamine oxidase A (MAOA) moderated the effect of maltreatment on the development of conduct disorders in adulthood. The authors asserted that these findings provided initial evidence that a functional polymorphism in the MAOA gene moderates the impact of early childhood maltreatment on the development of antisocial behavior in males. But, more importantly for social scientists, the study demonstrates the dependence of gene expression on a measured environmental factor in a behavioral domain. Males with low levels of MAOA who were not maltreated, though prone to being more aggressive, showed less aggressive behavior than those who were maltreated.

Applying our five criteria to these three studies (Table 1), we find that Caspi et al. satisfy the most criteria (three of the five). Two of the three studies (Caspi et al. 2002; Guo and Stearns 2002) succeed in conceptualizing social environments as

TABLE 1

Conceptualization of Environment in Three Studies

Criteria	*Guo and Stearns (2002)*	*Alford, Funk, and Hibbing (2005)*	*Caspi et al. (2002)*
Measure or control for environment?	Measure	Control	Measure
No assumption that environment *constrains* genetic expression			Yes
Environment is conceptualized as potentially enabling as well as constraining	Yes		Yes
Both environments and genetic potentials understood as nested and cross-cutting in potentially complex ways	Yes		Yes
Recognition that small environmental influences may have substantial influences on outcomes, particularly over multiple iterations		Yes	
Acknowledgement that change observed within historical time is mostly the result of environmental change	Yes	na	na

nested. However, Guo and Stearns (2002) include the strong assumption that environment can only constrain genetic expression. Only Alford et al. (2005) consider the possibility of small influences gaining power through multiple iterations over time. They do so because of existing theory of political socialization, which hypothesizes that early childhood experiences help to shape later political attitudes. None of the articles addresses the question of evolutionary time, although Alford and Hibbing (2004) offer a parallel theory of genetic dispositions based on human evolution.

This is by no means a systematic review of the field of behavioral genetics. Rather, we present these cases as examples of contemporary research investigating genetic influences on behavior. It is ironic that of our three exemplary studies, the two by social scientists offer less complete considerations of environment than does the one by a behavioral genetics team.

CONCLUSION

More than forty years ago, Dennis Wrong (1961) took sociological theory to task for its "oversocialized conception of man [sic]" (p. 192–3). By assuming individuals' actions fit with social needs, he argued, the then-dominant structural-functionalist paradigm avoided the question of how individual decisions might emanate from sources other than macrosocial ones. Instead of demonstrating behaviors' social origins, structural-functionalism assumed them, offering little source of an active self.[13]

Implicit in contemporary behavioral genetic research is the opposite error: an undersocialized conception of the individual that emerges through an unduly thin theoretical conception of the environment. The error is structurally similar to the one Wrong identified: this conception of the individual is implicit in, and assumed as a part of, the methodological approach used to establish the heritability of individual traits. It remains theoretically possible that such a conception of the individual is correct, but in the face of substantial sociological research suggesting that it is not, it should not be the strong assumption on which claims about the bases of human action are based.

The increasing behavioral genetics knowledge slowly being incorporated into sociological research highlights the complex relationship between genes and environment in predicting human behaviors. The incorporation of genetics into sociological models that attempt to explain human behavior both enriches and complicates them. Although sociologists are slowly embracing the possibility of the existence of genetic predispositions to types of human behavior, they must still place these new concepts of genetic predispositions within a sociological paradigm. Most critically, we should not discard the insights of generations of sociological theory, which teach us that social systems are complex, multifaceted, and often even self-contradictory.

Behavioral genetics needs theoretical sociology to incorporate a convincing conception of environment. Creating and finding data for models that both incorporate genetics and accommodate for complex interactions between genes and environment is a difficult task. In this article, we have identified key principles in

current sociological theory, attention to which would greatly benefit the work of behavioral geneticists. Sociologists must be able to create models that incorporate genetics and, at the same time, do not compromise their dedication to fully incorporating aspects of social environment that are imperative in upholding sociological standards. Some sociologists have already made attempts to do so. Acknowledging the role of biological predispositions does not require abandoning or denying the influence of social context on behavior.

Sociological theory provides a richer way of conceptualizing environment, where environment is not defined a priori as secondary to genetics. It provides a platform for better understanding GE interactions in our world. It helps to prove that genetic information cannot be understood or interpreted in the absence of social context; racial, ethnic, and socioeconomic factors effect the interpretation of genetic information because they are social constructions in and of themselves.

The issues we discuss here are important to scientific validity, but they have crucial ethical implications as well. Popular and academic considerations alike often assume genetic influences to precede, and even to be more "real" than, environmental ones. This conception has led to policy prescriptions that abandon efforts to ameliorate race, sex, and economic inequality, not for reasons of political philosophy but because of the erroneous assumption that such policies are doomed to fail because they violate a presumed "natural" order. To be clear, our objections are to the specific threats to scientific validity we outline here; our concerns are that the undersocialized self promoted by the undertheorized environment has potentially very negative outcomes.

For example, how are we to interpret the recent finding of genetically linked differences in "novelty-seeking" between Whites and Blacks (Benjamin et al. 1996)? If African Americans are more likely to become addicted to certain drugs because of a certain allele that they carry that Whites do not, can we confidently claim that the difference is caused by the presence of that allele? If—as generations of first-class sociological research has shown—race is a social category that stems from historical patterns of interpersonal and institutional domination (Conley 1999; Henderson 2004; Hirschman 2003; Omi and Winant 1986; Zuberi 2001) and addiction itself is a social issue whose distribution and prevalence has changed over short periods of historical time, what role, exactly, does the "allele" or "gene" play? We maintain that a primarily genetic interpretation would be both incorrect—empirically and theoretically as well as ethically. A robust understanding of the social environment is a necessary condition for addressing these very difficult but important questions.

Our discussion has focused on the shortcomings of oversimplified conceptualizations of environment. A necessary outcome of that focus is that we call for an added dose of methodological humility in attributing shares of biological and environmental genesis to behavioral traits. We believe that any study seeking to investigate genetic sources of human behavior should seriously consider the five criteria we have presented here. As social scientists, we find the simplicity and elegance of current behavioral genetic models compelling, and we expect sociologists to benefit greatly from incorporating their insights. However, we are quite concerned with the unmeasured threats to validity they contain and by the empirical, ethical, and scientific implications they convey.

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NOTES

1. With some exceptions, DNA testing can establish that an individual is related to one of three major population groups native to Africa (L1, L2, and L3) but cannot nearly approximate identification with a specific, modern tribe such as the Zulu.
2. We are grateful to an anonymous reviewer for pointing out the importance of this distinction.
3. See Norton 2004 (p. 33) for a provocative defense of this claim.
4. In separate work, Perrin is in the process of identifying the history and persistence of the paradox of reform in sociological theory. The term itself has been used only in a few specific instances, but the concept is remarkably recurrent across sociological domains.
5. This is one of two distinct uses of the term *expression* in genetics. At the cellular level, a gene is considered expressed when the protein for which it codes is present in a given cell (Lewin 2004: 25–6).
6. Calculating potential height based on parental heights introduces a new source of error, as parents' heights must also be affected by environmental factors. This concern, though, is separate from the theoretical question of whether individuals have a pre-environmental height potential whose expression environments can constrain or allow.
7. In behavioral genetics, heritability is defined as "the genetic contribution to individual differences (variance)" in a trait (Plomin et al. 2001: 87).
8. However, this research has more of an evolutionary biology focus than a behavioral genetic focus.
9. Shared environment refers to environmental influences experienced by both subjects in a family comparison, whereas nonshared environment refers to environmental influences experienced by one subject but not the other.
10. This hypothesis would parallel Callon's (1998) finding about economic actors "performing" the economic theory that was designed to explain their behavior.
11. Shanahan and Hofer do not consider gene-environment correlations, which may occur as a result of natural selection. These are "genetic effects on individual differences in liability to exposure to particular environmental circumstances" (Rutter and Silberg 2002: 464).
12. This is a birth cohort of 1,037 children (52 percent male) that has been assessed at ages three, five, seven, nine, eleven, thirteen, fifteen, eighteen, and twenty-one. Ninety-six percent of the sample was intact at age twenty-six.
13. Wrong went on to argue for a theory of the self-based in Freudian theory that offered a social basis for individuality without subsuming that individuality under social forces. This is an early exemplar of the theoretical problem of the structure/agency dialectic (see Emirbayer and Mische 1998; Sewell 1992).

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