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# Genetics and Social Inquiry

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## Abstract

Social science and genetic science still have fairly little engagement with one another, but the continued swift development of genetic science has certainly gained social scientists' attention. First, some social scientists are incorporating techniques from quantitative and molecular genetics into their work. Genetic data is increasingly recognized as providing valuable leverage even for research animated by strict interest in social environmental causes. Second, social scientists have been interested in understanding aspects of genetic science as a social phenomenon. This literature identifies possible noxious effects of uncritical public acceptance of genetic science, although how consistent these speculations are with public opinion and other available data is less clear. Because public understanding of genetics can influence behavior and social policies in ways that affect the ultimate causal potency of genes themselves, adequately theorizing genes as causes requires integration of these two lines of inquiry.

The price to assay a basic bit of genetic information—the single nucleotide polymorphism (or SNP)—is presently declining at rates faster than the fabled decline in the price per bit of computer memory. Consequently, we are at the beginning of what will be a radical increase in the data available to researchers for drawing connections among genetic endowments, life experiences, and observable life outcomes. Direct study has so far been broadly consistent with what indirect study has suggested for decades: that genetic differences are pervasively associated with outcomes of longtime social science interest (Udry 1995, Hernandez & Blazer 2006, Benjamin et al. 2007). Twenty-five years ago, serious scholars could credibly speculate that genetic differences might prove of only trifling importance for understanding individual differences in psychological characteristics, behaviors, and life attainments (Lewontin et al. 1984). Now, the terrain has shifted to arguments that genetic influence should not be overstated and can only be understood in a context of extensive causal interdependence with the social environment (Pescosolido 2006, Rutter 2006). Even though no one can foresee the particulars ahead, every indication is that advances in genetic science will continue to reveal how DNA differences combine with environmental differences to yield different life course trajectories and outcomes.

Some sociologists see nothing so opposed to the spirit of their craft as genetics. Deterministic genetic explanations have long served as textbook staples for illustrating what a sociological imagination is not. A common stance has been first to doubt that genetic differences actually are important for outcomes that sociologists study, but then also to assert that, if genetic differences do matter, they would still be irrelevant to the sociological study of those outcomes (Anderson 1967). The dramatic rise of human genetic science has happened despite the skepticism, hostility, and studied indifference of many sociologists. Sociologists have been thinking much more about genetics lately, however, as genetic science has become not just theory and findings but an ascendant social phenomenon. The rapid maturation of this science and its human consequences seem likely to stand as one of the most important developments of our age. As demonstrated by this review, the ways sociologists are now focused on genetics reflect the vast intellectual diversity of the discipline. Some are working with genotypic data, some are examining public beliefs and attitudes about genetics, some are interviewing the scientists, and some are forecasting the long-run implications of genetic innovation for culture.

The diversity of sociology's engagement with genetics also reflects the complex and dynamic character of genetic causes. The classic caricature is one of genetic differences unfolding into phenotypic differences, augmented perhaps by appreciation that this unfolding may be highly contingent upon characteristics of an organism's environment. Increasingly, social scientists are appreciating that genetics also provide a vital resource for understanding the consequences of environmental differences, and, indeed, genetic and environmental causes likely interpenetrate in ways that thoroughly defy our current capability for causal description. More than this, however, human genomic causality is not like causality in physics or even genomic causality in other species. What people believe about genetics and about their own genomes can influence their decisions and behaviors. As we develop more nuanced understandings of the biochemical causality of genetics, we create new opportunities for the psychosocial causality of genetic information (Freese 2006). Today, we have healthy individuals with genetic diagnoses lobbying the state to fund scientists to discover knowledge that can be translated into new technologies that can be used to prevent their genes from causing pathological consequences. Such possibilities are at the center of much of sociology's enduring unease with genetics, which reflects not just skepticism about the actual importance of genetic differences but also concern about deleterious social consequences that might follow from transformations in how people think about genes as causes of human difference. Prominent among these concerns has been that

the rising public enthusiasm for genetics will divert resources from research and policy oriented toward social structural causes of difference (Duster 2003).

Our review is divided into two complementary parts. First, we examine the possibilities for genetic information to elaborate and reshape how social scientists understand how social environments affect individuals. Second, we examine social science research on the possibilities for genetic information to elaborate and reshape individual beliefs and social relations. In both cases, we find that existing work is highly suggestive but also sharply limited, and the extent to which either social science or society will be reshaped by the increased availability of molecular genetic information remains unknown.

## GENETICS AND THE STUDY OF SOCIAL ENVIRONMENT

As noted, sociologists who acknowledge the possible importance of genetic influence for outcomes they study might nonetheless regard such influences as simply outside their discipline's purview. Although much could be said about the historical "boundary work" of sociologists in regard to their relationship with the biological sciences (Gieryn 1999, Pescosolido 2006), it is clear today that genetic information has much potential value for research oriented toward understanding social environmental causes. First, given accumulating evidence that genetic differences may be a source of pervasive confounding in estimating effects of social environmental causes on outcomes, genetic information is being used to try to strengthen causal inference. Second, heritability estimates attempt to measure what percentage of population variation in an outcome is accounted for by genetic differences. Heritability estimates differ across populations, and probing these differences may provide unique information for assessing macroscopic characteristics of societies, especially regarding the intergenerational dynamics of social stratification. Third, a ubiquitous problem in studying social environmental causes has been why such causes almost invariably affect similar people very differently, and studies of gene-environment interaction and correlation may help illuminate some of this heterogeneity.

### Improving Estimates of Effects of Environmental Causes

Findings of the substantial heritability of traits have been used to argue against any presumption that observed differences among persons can be wholly explained by divergent experiences. For example, the heritability of political attitudes contradict preceding literature on political socialization in which the possible importance of genetic influences on attitudes went virtually unmentioned (Alford et al. 2005). If an environmental cause and outcome each have substantial heritability, then it seems tenuous to presume that an ordinary observational research design will accurately estimate the true effect of the environmental cause.[1] For outcomes of interest to social scientists, genes may often be best conceptualized not as confounders per se but as causes of confounding phenotypic characteristics (Freese 2008). That is, the relationship between genetic differences and life circumstances seem likely to be mediated by more immediate individual characteristics. For example, genes may influence personality traits that affect choices (e.g., impulsivity and school continuation), early displays of aptitude that affect opportunities for development (e.g., early cognitive ability and tracking), and characteristics of appearance that affect treatment by others (e.g., skin tone variation within ethnic groups and discrimination).

Substantial heritability estimates have been observed for a wide enough range of outcomes so as to prompt otherwise moderate behavioral geneticists to declare that virtually

[1] In this scenario, for an ordinary observational study to yield an unbiased estimate, either the genetic influences on the environmental cause and on the outcome would need to be entirely independent of one another, or the intervening phenotypic measures would need to be measured fully and without error.

"everything is heritable" (Turkheimer 2000). These include such outcomes as educational attainment, earnings, divorce, delinquent behavior, and voter turnout (Behrman et al. 1980, McGue & Lykken 1992, Fowler et al. 2008). Even though specific heritability estimates are easily criticized, they nonetheless provide a diagnostic for the possibility of confounding by genetically influenced characteristics (Freese et al. 2003). If the received findings from behavioral genetics are even approximately correct, then conventional sociological techniques for estimating effects of social environmental causes likely yield pervasively biased results. We may expect this bias usually to overstate the consequences that would result from a targeted change of the specific social condition in question, although effects of environmental changes may also be underestimated (Martin 2008).

Longitudinal data have provided the traditional first line of defense against confounding from unmeasured differences among individuals. Longitudinal data work best for causes that are specific events, and even then the limitations of inference are well documented (e.g., Winship & Morgan 1999). In contrast, attempts specifically to reduce confounding owing to genetically influenced causes have long relied upon constructing samples of sets of persons with particular genetic relationships, most importantly twins and adoptees. For instance, J. Schnittker & J. Behrman (unpublished manuscript) raise the possibility that estimates of the strong positive effect of schooling on social connectedness may be confounded by unobserved characteristics that cause differences in educational attainment. They find greatly diminished and even sometimes negative effects when attention is restricted to monozygotic (MZ, identical) twins. Because MZ twins share not only genetic endowments but also generic features of family background and cohort, the strategy of estimating effects by comparing MZ twins eliminates a wide variety of potential confounders. Even so, MZ twins do not differ in educational attainment randomly, and estimates following this strategy may still be confounded by psychological and other characteristics that are not fully determined by genes and shared family environment. In other words, if one believes that cognitive ability confounds an estimated effect of educational attainment and also that genes are only one determinant of cognitive ability, then genetically informed designs still do not account fully for cognitive ability as a confound.

When samples include pairs with different genetic relatedness [e.g., MZ versus dizygotic (DZ, fraternal) twins, or adopted versus biological siblings], structural equation models can be used to estimate the relationship between an environmental cause and outcome net of overlapping genetic and shared family ætiology.[2] Using such models, Prescott & Kendler (1999) report that genetic and other confounding factors account for all of the strong association between age at first birth and alcoholism. Schnittker (2008) reports that genetic endowments account for most of the relationship between socioeconomic success and self-reported happiness. Rodgers et al. (2008) report that the apparent effect of education on age at first birth is accounted for by environmental causes shared by siblings in the same family. Thus, studies using genetically informed designs to assess confounders can yield much different conclusions than the samples of unrelated respondents more commonly used within sociology. Sociological research has invested heavily in data resources that are excellent for descriptive inference about populations (e.g., the General Social Survey); sociology has been slower to recognize how limited such data are for many kinds of causal inference.

The assumptions of behavioral genetics models have been heavily scrutinized (Goldberger 1979, Lewontin et al. 1984, Freese et al. 2003, Schaffner 2006). Unfortunately, however, such scrutiny has focused predominantly just on whether violations could result in heritability estimates being so overstated as to find consistently high heritabilities even

[2] Bayesian techniques are also being increasingly used in quantitative behavioral genetics (see Fowler et al. 2008).

when none exist. That possibility as grounds for generalized skepticism toward twin studies now seems quite unlikely (e.g., Kendler & Prescott 2006, Rutter 2006). Far less consideration has been given to assessing the consequences of violations of model assumptions for assessing multivariate relationships, such as assessing the relationship between a measured environmental cause on an outcome (like educational attainment and age at first birth). The model-fitting strategies that are used often imply relatively lower statistical power for detecting such effects. This can make conclusions that genetic overlap entirely accounts for the difference between two observed variables premature (Rutter 2006). Despite their advantages, then, multivariate behavioral genetics models surely should not be mistaken for a panacea for causal inference problems. More work is needed to understand how to combine findings from twin and adoption studies with those from other designs.

Compared with conventional models from twin and adoption studies, molecular genetic information allows for the possibility of more direct and more convincing inferences. As we discuss shortly, direct genotypic measures have enormous utility for assessing specific gene-environment interactions. As control variables in attempts to assess the importance of environmental causes, however, molecular genetic measures are limited in that measuring selected genes does not equate to measuring the whole genome [all genes and gene $\times$ gene ($\times$ gene) interactions]. Indeed, a humbling finding for behavioral genetic studies has been the small, often hard-to-replicate nature of estimated specific gene effects for outcomes with substantial heritability (Balaban 2001). Some speculate that one of the strongest ultimate lessons of the new genetics will be greater appreciation of how human development is dynamic and highly contingent in ways that contradict a regression-analysis-oriented conceptualization of either genetic or social environmental causes (Turkheimer 2006, Freese 2008).

Even so, molecular genetic information may help illuminate complex causal relationships among variables of enduring social science interest because the genome represents perhaps the primordial natural experiment: For each gene, which of the two copies we receive from each parent is effectively random and independent except for proximal locations on the genome. If a mother has copies of the $\varepsilon 2$ and $\varepsilon 4$ allele of the APOE gene, the latter of which is associated with increased risk of Alzheimer's disease, then it is a (quasi-)random matter whether the child inherits the $\varepsilon 2$ or $\varepsilon 4$ copy, and siblings have an equal chance of being concordant or discordant. Consequently, for example, if particular genes could be identified that have strong effects on lifetime health but did not affect socioeconomic status (SES)—except indirectly, through whatever their effect on health—then variation on these genes among full siblings potentially offers a powerful approach to disentangling the effect of health on SES from the effect of SES on health (Ding et al. 2006; see also Benjamin et al. 2007, p. 305; Ebrahim & Davey Smith 2008). Comparing genetically discordant full siblings using this design also eliminates the possibility of confounders from consequences of parental genes on parental health and subsequent parental SES (Fletcher & Lehrer 2008). This approach depends not just on identifying genes associated with the cause in question (e.g., health), but also establishing that these genes only influence the outcome (e.g., SES) through their influence on health. In sum, genetic information offers many possibilities for strengthening the work of even those researchers who are exclusively interested in social environmental causes. However, the strategies for doing so require both better understanding of the assumptions of the statistical models and further advances in our understanding of genetics.

## Heritability and Macrostructure

The heritability of traits, often presented as statements that $x$% of variation in a trait is genetic, are not transcendent facts of nature but population-dependent statistics. As such, when heritability estimates vary across populations,

that variation may indicate something meaningful about the populations. Boardman (2009) finds that the heritability of smoking is reduced in states with more aggressive public health measures against smoking. Guo & Stearns (2002) find that the heritability of cognitive ability is higher among children from higher status families (see also Rowe et al. 1999, Turkheimer et al. 2003). Guo & Stearns interpret the finding as indicating that socioeconomic advantage facilitates children reaching their genetic potential (for critique of this view, see Perrin & Lee 2007). Put differently, the results strongly suggest gene-environment interactions whereby socioeconomic advantage amplifies the positive effects of some genetic variants on cognitive ability. Nielsen (2006) follows similar reasoning to argue that cross-societal comparisons of heritability estimates of status attainments can be used to infer variation in opportunity for achievement (versus ascription) in a society. After all, if the only systematic determinant of status was the status of one's parents, then the correlation of status among MZ twins would be the same as that among DZ twins, and the heritability would be zero.

At the same time, inferences based on differences across populations in heritability estimates can easily conflate the influence of social environments with the amount of overall variation of environments. Again, heritability estimates are based on the premise that observed variation in an outcome can be partitioned into the percentage accounted for by genetic differences and the percentage accounted for by environmental differences. If the amount of overall pertinent environmental variation decreases, the extent to which the environment can explain variation in an outcome generally decreases, and estimates of heritability increase. In addition, traits like height and skin color variation are heritable, but we would not necessarily think of their contribution to the overall heritability of status attainment as indicative of the relative importance of achievement versus ascription in society (Freese 2008). That is, linking heritability to meritocracy can be misleading insofar as genetic differences influence some traits that may affect attainment in ways typically not recognized as merit. More generally, heritability estimates may provide an intriguing structural diagnostic, but these estimates are strongly limited in what can be inferred from them. Molecular genetic studies of specific gene-environment interactions may ultimately prove much more illuminating for the structural questions that heritability estimates have been used to address.

## Genetic Moderation of Environmental Causes

Social environmental causes commonly explain only a small portion of observed variation among individuals. A corollary is that outcomes typically differ widely among individuals who experience similar events. The same stressor, for instance, can have a strongly negative effect on one person and no discernible impact on another. Despite all the attention from sociologists about the advantages parents confer to children, about three-fourths of the income inequality in the United States is within sibships (Conley 2004). Genetic differences may be implicated in the different ways that two individuals respond to the same cause. The field of quantitative behavioral genetics was once skeptical of the importance of gene-environment interaction (Rutter 2007, pp. 15–16). Now, gene-environment interaction is a hot topic in behavioral genetics, biomedical science, and social science (Shostak 2003, Shanahan & Hofer 2005, Hernandez & Blazer 2006, North & Martin 2008). For sociology, the moderating effect of genetic differences may be key to understanding why individuals exhibit so much more heterogeneity in response to similar environments than would be predicted if they were uniform, passive automatons of structure and culture.

Shanahan & Hofer (2005) develop a typology of four common varieties of gene-environment interactions (see also Ottman 1996, Boardman et al. 2008). First, an environmental cause may trigger a genetic difference in an outcome for which little or no genetic

difference would be observed in the absence of the cause. In the most famous study of gene-environment interaction in recent behavioral science, Caspi et al. (2003) find that a genetic variant (MAOA) is associated with antisocial behavior in boys, but only among those who experienced abuse in childhood. Second, a positive environmental cause may compensate for a genetic liability, resulting in a more positive outcome than would otherwise be observed for those with the liability (and thus less effect of genetic difference overall). Shanahan et al. (2008) find that a dopamine-related genetic variant (DRD2) has a substantial relationship with school continuation, but this relationship is much reduced among those with high social capital. Similarly, Pescosolido et al. (2008) find that family support reduces the association between a genetic variant (GABRA2) and alcoholism. Third, social norms and limitations of opportunity can inhibit the relevance of psychological variation for outcomes and thus reduce the effect of genetic differences. Guo et al. (2008) offer a social control interpretation to the finding that a relationship between a DRD2 variant on delinquent behavior is not observed for respondents who have daily meals with their parents. Fourth, positive social contexts can increase genetic differences by providing disproportionate benefit to those whose genetic endowment already dispose them toward a more favorable outcome. As already mentioned, several studies find that genetic differences matter most for cognitive skills in advantaged environments.

Studies of gene-environment interaction will undoubtedly be central to future social science study of the environment, but challenges to positively identifying interaction temper enthusiasm for single studies. Entirely apart from genetics, interaction effects are notorious for replication failure (Benjamin et al. 2007). In gene-environment interactions, the possible genes and environments to be tested can multiply quickly. Additionally, some genetic variants may be rare, and observing a sizable effect in a rare subgroup relative to a much larger subgroup may prompt especial concern about replicability. Guo et al. (2008) find an interaction in which the relationship between the proportion of sexually active teens in the respondent's high school and the respondent's number of sexual partners is moderated by a dopamine-related genetic variant (DAT1). While certainly intriguing, the genetic variant found to be more responsive to the environment was observed for only 5% of the 674 individuals in their study. Moffitt et al. (2005, p. 478) emphasize the importance of theoretical justification for a candidate gene-environment interaction, and even then they extol the "wisdom of awaiting the meta-analysis, while not overreacting to any single study." Unfortunately, awaiting the meta-analysis is not easy to do in social science, where multiple studies on independent samples do not accumulate with the speed that they do in medicine. Beyond this, what constitutes a statistical interaction is more conceptually complicated than is commonly recognized within sociology, especially when guidance from theoretical models is vague at best (Ai & Norton 2003, Campbell et al. 2005).

Additionally, gene-environment interaction is sometimes used to characterize what is better termed gene-environment interdependence, encompassing not just interactions but also gene-environment correlations. In gene-environment interaction, genetic differences moderate effects for individuals who experience the same environments; in gene-environment correlation, genetic differences cause individuals to experience different environments. A poster child of gene-environment correlation would be a tall girl whose height prompts encouragement to play sports and who consequently ends up with more athletic skills than she would otherwise.

The signal contribution of sociologists to studies of gene-environment interdependence may be to elaborate how environment is understood and measured (Perrin & Lee 2007). Shanahan et al. (2007, 2008) employ methods borrowed from comparative-historical analysis to evaluate how the effect of genetic differences on school continuation may be moderated by configurations of multiple indicators of

social capital. More than this, behavioral genetics sorely needs a more sociological imagination of how the experience of environmental causes is patterned and often self-reinforcing over time. Elaboration of work on cumulative disadvantage and other tools from life course research can further our understanding of how selection into different environments widen initial individual differences, so that genetic and environmental causes may often be mutually reinforcing (Dannefer 2003, DiPrete & Eirich 2006). Similarly, work on fundamental causality in medical sociology can increase appreciation of how basic conditions like SES manifest as a very broad array of specific environmental causes (Link & Phelan 1995, Freese & Lutfey 2009). This work also highlights how advances in knowledge often benefit different groups differently, underscoring the potential for technologies like genetic testing and therapies to affect health disparities. Theories of culture and symbolic interaction can help articulate how the effects of environments are contingent on the meanings attached to them, implying among other things that improving measures of environment requires improving measurement of their subjective as well as objective aspects (Schulz & Lempert 2004, Kaufman 2004). In sum, the sociological imagination seems to have much to contribute to improving the environment side of the burgeoning literature on gene-environment interdependence, but to do so, sociologists must be willing to give genetics serious attention.

## GENETICS AND THE STUDY OF ITS SOCIAL IMPLICATIONS

Human genetics has been centrally concerned with understanding how genes work as causes of development and of disease. As already noted, however, genetic advances are not passive, but rather new knowledge claims diffuse, serve as warrants for individual action, and potentially transform social arrangements. Sociologists have done at least as much work trying to identify the consequences of genetic science for society as they have trying to make use of genetic information in their own studies. This work has expressed much concern for the possibility of genetic science remaking the public consciousness through a process of geneticization. As we discuss below, writing on geneticization has to date not made much use of studies of beliefs and attitudes regarding genetics among the general populace. In addition, work on geneticization has also been only loosely associated with a burgeoning separate literature on biosociality that emphasizes new collective identities based on genetic or other biological information.

### Geneticization

Geneticization has been central to sociology's critical analytic stance toward new developments in genetics. As introduced by Lippman (1991, p. 19), geneticization refers to "an ongoing process by which differences between individuals are reduced to their DNA codes, with most disorders, behaviours, and physiological variations defined, at least in part, as genetic in origin." Geneticization is understood to encompass both what people think and what is done in clinical and other settings. Thus, for example, the identification of the BrCa1 and BrCa2 genes, which significantly increase a woman's risk for breast cancer, occasioned predictions of the geneticization of breast cancer, even though hereditary breast cancers account for no more than 10% of all breast cancer diagnoses in the United States (Sherwin & Simpson 1999). Of particular concern was the possibility that individualized clinical management strategies based on genetic testing would replace public health approaches to cancer prevention (Sherwin & Simpson 1999) and mitigate attention to environmental risk factors (Conrad 1999, p. 235).

Lippman's (1991, p. 18) animating paper on geneticization emphasized how genetics as a "dominant discourse" has numerous potential negative social implications. Geneticization thereby has sometimes served as a connotative shorthand for various frequently raised concerns about how new genetic findings may be

understood and used. First is genetic reductionism, in which a complex and ecumenical understanding of the causes of human development is supplanted by one in which genes are perceived as the "true cause" of difference (Sloan 2000, p. 17).[3] Second is genetic determinism, in which genes are taken as inevitably implying traits and behaviors (Lippman 1992, Alper & Beckwith 1993, Rothman 2001, Nelkin & Lindee 2004). Third is genetic essentialism, in which genetics becomes a dominant way "to explore fundamental questions about human life" and "to talk about guilt and responsibility, power and privilege, intellectual or emotional status" (Nelkin & Lindee 2004, p. 16). Social scientists are concerned that these phenomena contribute to individualizing social problems and to relegating social, political, and economic explanations of health and other outcomes to secondary status (Lippman 1991; Duster 2003, 2006). For example, Duster (2003) argues that extensive public sector investment in genetic research will disproportionately and negatively impact blacks by diverting attention and resources away from social environmental factors that contribute to increasing rates of lung cancer and cardiovascular disease in the African American population. Bourdieu (2003, p. viii) asks, "How can we deny the link between the forceful return of conservative thinking and the favorable climate offered by progress in genetics?"

As with use of the term "medicalization" (Conrad 1992), there has been disagreement about whether geneticization implies critique or whether it can be used to characterize empirical phenomena neutrally. At issue is whether geneticization is now too ethically loaded or morally circular to serve empirical research (Hedgecoe 1998), such that instead it should it be understood as "a heuristic tool" in a moral debate (ten Have 2001). Sociologists have countered that the literature on geneticization is replete with empirical claims that could be evaluated with the tools of social scientific research (Hedgecoe 2001). Toward this end, Hedgecoe (2001, p. 307) suggests a "stripped down" definition whereby geneticization "takes place when an explicit link is made between a condition and a stretch of DNA."[4]

Variously defined, geneticization has been at the center of empirical analyses of the consequences of genetic research. Medical sociologists have studied the effects of geneticization on scientific classification, disease nosologies, and etiological narratives by analyzing scientific and clinical articles (Cunningham-Burley & Kerr 1999; Kerr 2000; Hedgecoe 2001, 2002). This research has revealed that the way genetic information reshapes disease categories is varied and sometimes "stealthy" (Hedgecoe 2002). Hedgecoe (2001) raises concern about the emergence of an "enlightened geneticization" in which genetic explanations are privileged over nongenetic explanations, not because of genetic determinism but because genetic causes are seen as more easily specified and researched.

In mass media, content analyses find a pattern of "genetic optimism" in the reporting of genetics research, especially in the United States (Conrad 2001). Newspapers often prominently feature genetic findings but give scant attention to disconfirmations, failed replications, or limitations (Conrad 2001). A case study by Horwitz (2005) finds that media coverage of research on gene-environment interaction selectively emphasizes the gene half of the interaction and largely ignores environmental causes. Attempts to assess over time whether print media evince patterns indicative of an increasing geneticization of public attitudes have yielded discrepant results. Duster (2003) finds an increase in articles invoking a genetic explanation of crime in the late 1970s

[3] What constitutes a reductive statement is not always clear: The Lippman definition quoted above appears to contend that defining "most" traits as "at least in part" genetically influenced reduces individuals to their DNA.

[4] An alternative analytic frame is provided in writing on "molecularization" (de Chadarevian & Kamminga 1998, Shostak 2005, Fullwiley 2007). Molecularization refers to the reorientation of the life sciences to the submicroscopic level (Kay 1993) and the concomitant reorganization of scientific institutions, practices, and forms of capitalization (Rose 2001).

and early 1980s, but Condit et al. (1998) find over a longer time span that, if anything, discourse about heredity may be becoming less deterministic. Condit (1999) also warns against any presumption that the use of supposedly geneticized metaphors for genetics in mass media (e.g., genetic blueprint) implies that layperson conceptions of genetic causation are either deterministic or reductionistic.

Research on geneticization in medical practice has drawn on observation and analysis of clinical settings and interviews with people who are users or potential users of clinical genetic technologies (e.g., individuals who have or may carry a genetic predisposition to a particular condition, pregnant women, etc.), their family members, and clinicians. Analytic foci here include the structure of clinical genetics services, the use of genetic technologies and information by clinicians and lay people, the management of the uncertainty of genetic diagnoses, and communication about test results within families (Bosk 1992, Cox & McKellin 1999, Hallowell 1999, Rapp 1999, Lock et al. 2006, Whitmarsh et al. 2007). These analyses consider also how the possibilities of genetics research reshape relationships between clinicians, activists, researchers, and both the public and private sectors of society (Kerr et al. 1998, Stockdale 1999, Rabeharisoa 2003, Callon & Rabeharisoa 2003, Heath et al. 2004).

Taken together, this research has revealed that neither the empirics nor the implications of geneticization are as simple as early writing suggested. Against the notion that geneticization is an inevitable consequence of genetic research, recent analyses have suggested that path dependency (Shostak et al. 2008), network structures (Hall 2005), and the daily practices of diagnosis and treatment of disease (Cox & Starzomski 2004) may enable or impede geneticization, even for conditions with simple genetic etiologies. For example, hereditary polycystic kidney disease (PKD) is a life-threatening, autosomal dominant trait for which genetic testing is available. However, in a case study of the social construction and clinical management of PKD, Cox & Starzomski (2004) noted a striking absence of attention given to the genetic aspects of the disease by health-care providers, patients, and family members. They attribute the mitigation of geneticization to the irrelevance of genetic information to most practical aspects of PKD diagnosis and treatment. The emphasis on disease management may also minimize focus on the hereditary basis of PKD by bringing patients together with others experiencing different types of kidney disease.

In contrast to understanding of geneticization as a deterministic discourse, research points to the enduring power of local knowledge (Rapp 1999), national contexts (Prainsack & Siegal 2006, Remennick 2006, Parthasarathy 2007), and everyday understandings of risk, kinship, and inheritance (Richards & Ponder 1996) in shaping lay understandings of genetics. In an explicit challenge to the assumption that geneticization is coterminous with genetic determinism and fatalism, Rose and colleagues (Novas & Rose 2000, Rose 2007) argue that genetic information creates new obligations to act on knowledge to protect health and to maximize quality of life. Research also suggests that many users of genetic testing appreciate the nuances of probabilistic risk and predictive uncertainty and are correspondingly circumspect in their interpretations of genetic information, even in regard to prenatal genetic testing, arguably the clinical setting in which genetic testing is the most routinized (Markens et al. 1999, Rapp 1999, Franklin & Roberts 2006, Lock et al. 2006). In the postdiagnosis context, parents of children with genetic conditions (e.g., Klinefelter, Turner, and fragile X syndromes) may simultaneously accept authority of molecular genetic test results and create a space for uncertainty about the condition by emphasizing variation between diagnosed children, the individuality of their diagnosed child and his or her accomplishments, and other ambiguities in prognosis that complicate the significance of genetic information (Whitmarsh et al. 2007). Looking ahead, the expanding focus of the life sciences on complex biological systems (Kitano 2002, Fujimura 2005), gene-environment interactions (Shostak 2003, Freese 2006,

Schwartz & Collins 2007), and epigenetics (Francis et al. 1999, Meaney 2001, Lock 2005) can be expected to continue to challenge how social scientists think about geneticization.

## Public Opinion about Genetics

Public opinion surveys make plain that most Americans regard genes as important determinants of individual and social outcomes. In one recent survey, over 90% of American respondents reported genetic makeup as at least somewhat important for physical illness, and almost two-thirds did so for success in life (Shostak et al. 2009). Belief in the importance of genetics for particular outcomes may be increasing [e.g., obesity (Singer et al. 1998)], but it is not known whether there has been any overall shift toward belief in genetics, as obviously widespread notions of the importance of breeding or inborn character long predate the discovery of DNA (Kevles 1998). Evidence from an experimental investigation of the consequences of clinical genetic information found that participants presented with results for what was called a genetic test for heart disease perceived the disease to be less preventable than those assigned to the unspecified test condition (Senior et al. 2000). However, fears of rising genetic determinism among the general public are not well supported, as people appear to recognize that the importance of genetics can vary for different outcomes and that genetic influence does not rule out the importance of other factors (Parrott et al. 2003).

What outcomes are regarded as more genetic may be influenced by a cultural schema, at least in the United States, in which individual characteristics perceived as closer to the body are seen as most strongly caused by genetics. A recent study of genetic attributions for individual outcomes found that physical health is perceived as more strongly genetically influenced than mental health; mental health is perceived as more strongly genetically influenced than personality; and personality is seen as more strongly genetically influenced than success in life (Shostak et al. 2009). Such a cultural schema may reflect the legacy of Cartesian dualism, which insists that the causes of bodily states, such as physical illness, are to be located in the body (Scheper-Hughes & Lock 1987). The association between genetics and physical health has also been central to popular rationales for contemporary genetic research (Collins & McCusick 2001). In addition, many laypeople have a strong notion of individual will as a causal force independent from either genetics or environment, which could be more important as outcomes are less immediately embodied (Condit et al. 2006).

Although research has considered the possibility of various sorts of social cleavages in beliefs about genetics, race/ethnic differences have received the most attention, motivated partly by the hypothesis that the historical use of biological claims to justify racial inequality will prompt minorities to be more skeptical of genetics. Using vignette data from the General Social Survey, Schnittker et al. (2000) found that blacks are less likely than whites to endorse genetic explanations of mental illness (Schnittker et al. 2000, pp. 1109, 1112). In contrast, however, Shostak et al. (2009) find that individuals who self-identify as black or Latino all rated genetic makeup on average as more important for a set of individual attributes than did individuals who identify themselves as white. They did find that blacks were relatively more averse than whites to endorsing genetic makeup as important to individual differences in intelligence—the outcome for which historical abuse has been most insidious—but that was the only instance in their analysis in which a socially disadvantaged group evinced a pattern suggestive of greater aversion to genetic explanation (Shostak et al. 2009). Regarding genetic testing, blacks and Latinos may be more enthusiastic about being tested, but this result may be mediated by blacks and Latinos being on average less knowledgeable about it (Singer et al. 2004).

Academic discussions of heritability regularly point out that evidence of the importance of genetics for explaining individual differences is not evidence of the importance of genetics

for explaining group differences (e.g., Schaffner 2006). To our knowledge, no published study has considered how the same people respond to questions about individual and group differences in the same trait. Surprisingly, perhaps, Hunt's (2007) results indicate that blacks are not less likely than whites to regard innate ability as important to explaining black-white differences in socioeconomic attainment in General Social Surveys since 1990 (12.0% of whites and 12.2% of blacks).

Only a few studies have considered the relationship between beliefs about genetic causes and specific policy attitudes. Shostak et al. (2009) find that belief in the importance of genetics for individual differences in outcomes are associated with support for policies predicated on genetic causes being important, such as supporting human genetics research and supporting genetic screening before marriage. Regarding beliefs in the genetic basis of group differences, Jayaratne et al. (2006) find that belief in the genetic basis of racial differences is associated with more negative attitudes toward blacks and less support for social policies to help blacks (Keller 2005, Jayaratne et al. 2006). The direction of causality here is unclear, and belief in genetic differences between oneself and an outgroup does not inevitably imply negative attitudes. Notably, genetic attributions for differences in sexual orientation are associated with greater tolerance toward gay men and lesbians, as measured by attitudes toward gay marriage, adoption by gay couples, and whether gay people should be allowed to teach elementary school (Jayaratne et al. 2006; see also Tygart 2000).

The last point deserves highlighting: Instead of public acceptance of genetic explanation having any generic social implication, there seems far greater reason to suppose that existing discourse and public opinion strongly condition whatever consequences findings of genetic influence have (Shostak et al. 2008). As Hacking (2006, p. 90) writes:

> Consider the well publicized searches for a gay gene (typically in men) and an alcoholism gene. Those who hope for an alcoholism gene believe that the discovery will prove beyond all doubt that alcoholism is a disease or, at any rate, an innate disability. Those who hope for a gay gene believe that such a discovery will prove beyond all doubt that homosexuality is not a disease or a disability.

In sum, although broad public belief in the importance of genetic causes is clear, in representative samples of the U.S. population public understanding appears to be more nuanced and resistant to deterministic reasoning than some have feared.

## Biosociality

At least with respect to health, the rise of genetic science has not coincided with any new ascendance of hopelessness or feelings of inefficacy among the public. Indeed, genetic research has secured enormous funding precisely through its success in cultivating hope that understanding genetic causation will lead to the development of improved capacity for intervention (Sunder Rajan 2006, Novas 2007). In articulating the concept of "biosociality," Rabinow (1996) argued that advances in biological knowledge, especially the reclassification of illnesses as genetic and the corresponding emphasis on genetic risks, would yield new forms of individual and collective identity and an increasingly ambitious orientation of individuals toward themselves as material entities. These new forms of identity, Rabinow predicted, will be the basis of new social relations, as people genetically at risk for specific conditions "will have medical specialists, laboratories, narratives, traditions, and a heavy panoply of pastoral keepers to help them experience, share, intervene in and 'understand' their fate" (Rabinow 1996, p. 102). Rabinow predicted that such microlevel political practices and discourses embedding genetic information in social life would make the new genetics "a greater force for reshaping society than was the revolution in physics" (pp. 98–99).

Biosociality has framed inquiry into both the emergence of new identities and the reworking

of existing ones, such as race and gender (Hacking 2006, Atkinson et al. 2007, Gibbon & Novas 2008). Research focused on biosociality has also traced operations and effects in social movement organization and advocacy (Callon & Rabeharisoa 2003), transformations in the relationships between citizens and the state (Petryna 2002, Heath et al. 2004, Epstein 2007), and new modes of capital production and economies, which often rely on innovative relationships between disease advocacy groups and scientists (Heath et al. 2004; Sunder Rajan 2006; Novas 2007, 2008; Silverman 2008). Though not explicitly framed as research on biosociality, sociologists have also examined the consequences of the reclassification of illnesses as genetic for people's perception of the severity and treatability of the illness and their orientation to the affected person (Phelan 2005). Using vignettes to study the effects of genetic attributions for mental illness, Phelan (2005) finds that when people are told that a mental illness has been caused by genetics, they are more likely to perceive the illness as a serious and persistent condition and more likely to believe that siblings and children of the affected person would develop the same problem. Genetic attribution did not have an affect on respondent's endorsement of reproductive restrictiveness or desire for social distance from the ill person, but it did increase desired social distance from the person's sibling (Phelan 2005). Such analyses suggest that genetic attributions might shape not only health-care utilization but also social group formation.

Biosocial research describes several ways that genetic knowledge expands the place of biomedical categories in everyday life (Clarke et al. 2003). First, increasing numbers of phenotypically healthy individuals are transformed into beings who are "genetically at risk," or "patients-in-waiting" (Rose 2007, Sunder Rajan 2006). Diagnosis of genetic risk can involve not just changes in one's understanding of oneself, but also new connections to others based on shared risk and potentially different treatment by employers, insurers, or other institutional entities (Duster 2003, Wailoo 2003, Rose 2007). Second, through a variety of developments, the sense that disease risk compels action has increased, such that "genetic forms of thought have become intertwined with the obligation to live one's life as a project" (Rose 2007, p. 129). Reminiscent of Parsons (1951) on the obligations and entitlements that accompany the social role of "sick," Condit et al. (2006) find that although laypeople do not hold individuals responsible for their genetic endowments, individuals are expected to work to override negative genetic predispositions as much as they are able. Third, increasingly, one response to diagnosis is to contribute to collective efforts to increase and improve the scientific study of one's illness, a rising mixture of medical statuses and politics (Epstein 1996, Petryna 2002, Novas 2007, Silverman 2008). In all these cases, biosocial relations have the potential to reshape the consequences of genetic differences, underscoring that ultimate assessments of genetic causes will depend not just on the causality of genes but on the causality of scientific knowledge claims about genes.

The specter of eugenics is often raised in discussions of the capacity of individuals to act upon genetic information. Few words are more strongly freighted by negative historical connotations. Broad consensus against eugenics stands prospectively in tension with widespread support for the right of women to discontinue pregnancies on the basis of prenatal information indicative of a severe birth defect.[5] The scenario suggests two different slippery slopes that observers such as Duster (2003) argue deserve more careful consideration than they have received. The first is the definition of "defect" (Parens & Asch 2003), including whether mothers should have full rights to determine what genetic tests are performed and what constitutes acceptable grounds for terminating a pregnancy. The second is whether practices that are initially permitted as a matter of individual

[5] Over 70% of respondents in the 2006 General Social Survey said abortion should be legally permitted when there is indication of a "serious" birth defect (analyses by authors).

liberty can be kept voluntary or whether social processes will lead them to become normative or institutionally mandated (e.g., by health insurers). The moral issues here are obviously weighty, and Duster (2003) especially has contributed importantly to highlighting awareness of negative long-term possibilities of technologies that are otherwise widely depicted as benign.

Discussions of eugenics are often part of larger discussions of genetic innovation and race/ethnicity. Both race and geographic ancestry have long been principal categories through which medical research seeks to ascertain individual disease risk and to develop interventions (Reardon 2004). More recently, racial categories have been used in pharmacogenetic and other genetic research, and the FDA approval of the drug BiDil specifically for use among African American cardiovascular patients was denounced by many (e.g., Sankar & Kahn 2005), although these denunciations were themselves criticized (Carlson 2005, p. 468). Part of the question here is whether historically constituted racial/ethnic categorizations overlap enough with genetic differences to provide useful biomedical information for treatment before interventions are developed that account specifically for the genomes of individual patients (Risch et al. 2002). Beyond this are questions of whether such treatments have noxious effects for reifying race/ethnicity regardless of therapeutic value and whether they undermine efforts to ameliorate social causes of racial disparities in health (Sankar et al. 2004, Duster 2006, Abu El-Haj 2007, Bolnick et al. 2007, Fullwiley 2007).

Genetic ancestry testing is also emerging as a novel means by which ideas about the past, notions of kinship, and self-identity are being produced (Nelson 2008; see also Perrin & Lee 2007). Businesses like 23andMe (**http://www.23andme.com**) offer to analyze customer's DNA and provide reports about geographic ancestry and genetic overlap with family members and friends. Fourteen anthropologists, sociologists, and others recently collaborated on an essay in *Science* that raises many concerns about such tests, including their accuracy, interpretation, marketing, possible effect on the well-being and confidence in science of those tested, and possible effect on social research by affecting self-categorization on questionnaires (Bolnick et al. 2007). At the same time, Nelson's (2008) study of geographic ancestry testing in the United States and the UK demonstrates that people are active interpreters of information from ancestry tests, aligning it with other sources of information and meaningful categories of identity in their lives. This is consonant with other research suggesting that the process through which science is incorporated into identity is a continual, creative synthesis between science and "local common sense" (Dumit 2003, p. 44). Hacking (2006, p. 93) goes so far as to speculate, "Only those who want to listen to their genes will do so," though certainly the distribution of the opportunity to listen will be shaped by social and economic factors, alongside emerging medical screening guidelines and practices. As with much else about the social implications of genetics, social science has provided a lucid cautionary voice on the potential harm of broad uncritical acceptance of oversimplifications about genetics, but empirical social science has just begun to examine how new genetic information will actually be incorporated into the attitudes and behavior of the public.

## CONCLUSION

The two halves of our review cover what are today two largely distinct enterprises. The first enterprise is trying to bring behavioral genetics into sociology and in so doing make behavioral genetics more of a social science. The project rejects a separation of inquiry into genetic and social causes but instead maintains that these are so interdependent as to make assessing one confounded and incomplete without the other. The second enterprise is wrestling with genetic science as a transformative social phenomenon. This project recognizes that genetic science cannot be separated from the context of its production but instead depends on that context for

its sustenance and produces claims that provide a basis for subsequent diffusion, interpretation, and action. Having these two enterprises in the same discipline exemplifies the diversity and reflexive tradition of sociology. To wit, in this paper, the same study is referenced as a finding in the first half and as an object of a case study of media coverage in the second (Caspi et al. 2003, Horwitz 2005). At the same time, that the two complement one another is not quite the same as saying they as yet contribute much to one another.

Indeed, these enterprises often involve such different animating interests and epistemological commitments that gains from mutual awareness presently seem more appreciative than generative. We do think some writing on the social implications of genetics might benefit from greater appreciation of the statistical techniques of quantitative behavioral and population genetics, as well as how different conventional quantitative and molecular genetics are. On the other side, studies of gene-environment interaction sometimes seem to conceive of environments without appreciating enough the background reasons for why environments differ and how scientific knowledge and public beliefs may transform environments over time. Additionally, the geneticization critique underscores the importance of sensitivity to possible harmful consequences of genetic science, including how genetic causes may crowd out social explanations and interventions. Especially as the social sciences begin to incorporate genetic information and techniques, it is vital that sociologists examine the processes and outcomes of these new modes of knowledge production as empirical phenomena.

To whatever extent genetic science does expand the human capacity for intervening on the self, then the study of genetic/social feedback effects may be one area that brings the two enterprises together. As already noted, part of how social science may improve behavioral genetics is by giving more appreciation and specificity to such feedback effects. It is not just that genes can influence outcomes by influencing what people experience, but also that direct and indirect effects of genetic differences may often be positively correlated. For example, genes that more directly influence adverse outcomes (e.g., mental illness) may also often influence selection into adverse environments (e.g., stressful life events) because of various ways that mental illness symptomatology and the experience of life setbacks can be mutually reinforcing. We suspect that much remains to be articulated about general social dynamics that can yield mutually reinforcing genes and environments. One social mechanism is simple comparative advantage: the incentives that status hierarchies provide for investing energy in developing traits for which an actor evinces early aptitude. Homophily in social networks provides another obvious example. As already noted, the life course perspective of sociology seems especially well suited to advancing understanding of the complicated configuration of opportunity and choice in nested social contexts that determine the ultimate import of genetic differences (Shanahan et al. 2003).

These feedback effects implicate work in genetic science as a social phenomenon especially when the availability of genetic information causes variation in action. The fundamental cause perspective in epidemiological sociology already highlights how different uses of advancing knowledge preserve enduring health inequalities (Link & Phelan 1995, Link 2008). Some health inequalities may have widened as efficacious intervention has moved from general, population-level measures (like improved sanitation) to measures more contingent on individual access, choice, and management (Freese & Lutfey 2009). Genetic medicine touts the possibility of still more individually tailored interventions (Feero et al. 2008). To the extent that such possibilities are realized, the question remains as to how the benefits of genetic medicine are distributed and why. Simple resource differences will doubtless play an important role, but beliefs about genetics and attitudes toward science and medicine may also be important. If genetic differences influence any characteristics that lead to differences in utilization, these differences may intersect with

technology to become more important as genomic science advances (Freese 2006). Although most writing on fundamental causes of health have focused on differences in benefit from innovations as they arise, the biosociality of patient advocacy groups underscores how information about genetic risk can prompt action to shape access to interventions and to try to hasten the development of scientific knowledge. Consequently, the social distribution of genetic conditions thereby might be implicated not just in who will benefit from available technologies but also in what technologies are developed. Likewise, research on biosociality highlights the possibility that applications of genetic technologies, within and beyond the clinic, can both reshape and reinforce extant categories of identity. The consequences of these transformations for how individuals and groups claim rights and benefits may further shape variations in health and social status. The diversity of ways in which genetics penetrate social life makes clear the importance of continued sociological engagement with all aspects of genetic science using the varied perspectives that our discipline brings.



## SUMMARY POINTS

1. Estimates of effects of social environmental causes on individual-level outcomes may be pervasively confounded in study designs that ignore genetic endowments.
2. Heritability estimates do not index transcendent features of nature and may even be used comparatively to assess characteristics of social structure.
3. Studies of gene-environment interaction and correlation suggest that genetic and social causes may be more thoroughly intertwined than had been appreciated.
4. Writing about the possible geneticization of outcomes encompasses both a moral critique and an empirical phenomenon that can be more neutrally engaged in social science research.
5. Public belief in the importance of genetic causes is plain but also more nuanced and resistant to determinism than some have feared.
6. The concept of biosociality has animated work on how biological advances yield new forms of collective identity and efficacy.

## DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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