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Sociogenomics in the 21st Century: An Introduction to the History and Potential of Genetically-informed Social Science

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Abstract

This article reviews research at the intersection of genetics and sociology and provides an introduction to the current data, methods, and theories used in sociogenomic research. To accomplish this, I review behavioral genetics models, candidate gene analysis, genome-wide complex trait analysis, and the use of polygenic scores (sometimes referred to as polygenic risk scores) in the study of complex human behaviors and traits. The information provided is meant to equip readers with the necessary tools to: (1) understand the methodology employed by each type of analysis, (2) intelligently interpret findings from sociogenomic research, and (3) understand the importance of sociologists in the ever-growing field of sociogenomics. To unify these three tasks, I rely on various examples from recent sociogenomic analyses of educational attainment focusing on social stratification and inequality.

Keywords

Social Stratification; Inequality; Sociogenomics; Genetics; Genome-wide; Polygenic Score; Heritability

Introduction

Scientists have long debated the importance of nature versus nurture, or the degree to which traits and behaviors are determined by inborn characteristics versus the environment, in the development and maintenance of systems of social stratification. And while the statement nature *versus* nurture evokes a sense of all or nothing, the reality is that nature *and* nurture play important roles in creating and maintaining systems of social stratification. But, before entering into a discussion of the history of sociogenomics and the place of sociologists in that field, it's important to understand why sociologists should be interested in the integration of genetic and social measures in the first place. DiEwald and colleagues (2015, p. 374) summarize the argument succinctly, "parents not only pass on resources and experiences to their children, but also their genetic predispositions... [thus] inequalities exist between individuals from birth on, not only in their social origins but also in their genetic endowments." If only social influences are accounted for, any estimates which claim to find

a causal association between the included social influences and the behavioral trait being studied could be biased. The bias generated by omitting genetic influences is often referred to as the endogeneity problem. Only by accounting for genetic influences in traditional social science analyses can scientists identify the “true” causal effect of social and environmental factors free of genetic confounding (Plomin et al., 2016).

There are two general solutions to the endogeneity problem described above: either design an experiment in which social factors are randomly assigned to individuals, or include measures of both important social factors and genetics in your analyses. The first option violates contemporary research ethics rendering it an untenable solution. Similarly, the second option, which requires accurate measurement of genetic variation between individuals, was, until very recently, extremely difficult. Consequently, ignoring the influence of genetics was considered acceptable practice in social science research and the potential bias in the estimated association between the outcomes and mechanism(s) being studied was simply considered part of the uncertainty inherent in modern scientific practice. But, with the increasing prevalence of genome-wide genetic data in many large-scale nationally-representative studies and the development of statistical methods that facilitate the use of genetic data in traditional social science analyses, the second solution to the endogeneity problem is becoming an increasingly viable option. Consequently, social scientists are faced with a growing set of tools and resources capable of integrating sociogenomic data and methods into their research.

The simple fact is that the genomic revolution is here (Conley and Fletcher, 2017; Phelan et al., 2013), and sociologists must engage with the data, methods, and research accompanying the revolution or risk being left behind, or worse, missing the opportunity to affect the precision and direction of this burgeoning field exactly when their expertise is needed most. This paper aims to provide an overview of the history of the field of sociogenomics and a few of the potential benefits of including sociogenomic measures and methods in social stratification research. In section one, I provide a description of previous attempts to measure the influence of genetics on outcomes traditionally studied by social scientists, including behavioral genetics and candidate gene analyses. In section two, I discuss the current trends in sociogenomics that rely on observed measures of individual genetics including genome-wide complex trait analysis and the calculation and use of polygenic scores. In section three, I discuss some of the areas in which sociologists may have a particular advantage in contributing to sociogenomic research. And, lastly, in section four I present some concluding remarks on the importance of incorporating sociogenomics in sociology as well as the continuing need for more sociology in sociogenomics.

Section One: Early Methods in Sociogenomic Research

Two of the early attempts to integrate genetic information into social science research are behavioral genetics analysis and candidate gene analysis. In the following section I briefly review each method and their connection to research on social stratification.

Behavioral Genetics

Behavioral genetics analyses are the oldest and most widely used form of sociogenomic analysis. Between 1958 and 2012 approximately 3,000 articles were published that use behavioral genetics models to estimate the association between genetics and over 17,500 traits (Polderman et al., 2015). While the analyses used to estimate these associations are frequently referred to as behavioral genetics models, they are also known as twin and/or ACE models. Behavioral genetics analyses use information on the average genetic relatedness between siblings, typically twins, to decompose variation in an observed outcome into genetic and environmental influences.

Estimates of the proportion of variance in an outcome attributable to genetic influences are referred to as the heritability of the outcome being studied. Although often assumed to indicate individual-level associations, estimates of heritability represent a population-level measure of the average association between genetics and the outcome being studied within a given society at particular time (Plomin et al., 2013). Due to the fact that heritability is a population-level measure it can be, and often is, affected by differences in social, institutional, and cultural structures (Adkins and Guo, 2008; Adkins and Vaisey, 2009; Diewald et al., 2015).

A recent example of the use of behavioral genetics analyses in social stratification research is the work by Nielsen and Roos (2015), which investigates inequality of opportunity in educational attainment in the contemporary U.S. By comparing estimates of the heritability of educational attainment to the proportion of variance in education attributable to social factors, Nielsen and Roos (2015, p. 22) show that despite numerous policy interventions meant to equalize educational opportunities during the latter half of the 20th century there is “persistent inequality of opportunity for educational attainment in American society at the turn of the twenty-first century.” In this case the use of sociogenomic methods by Nielsen and Roos is applied to depict continuing systems of stratification in relief to genetic effects.

The results of the thousands of behavioral genetics estimates of heritability provide evidence that most human traits are, to some degree, influenced by genetics (Plomin et al., 2016; Polderman et al., 2015; Turkheimer, 2000), suggesting that the pervasiveness of the endogeneity problem discussed in the introduction may be greater even than many social scientists care to admit. The robustness of these findings suggests that scientists who continue to choose to ignore the influence of genetics in their study of inequalities in health, behavior, and other intergenerational outcomes and processes introduce an unknown amount of bias into their findings. What’s worse is that in most cases, the direction and magnitude of the bias is typically unknown. Thus, without incorporating appropriate genetic measures into their analysis, scientists cannot be sure if the reported association between policy indicators, social systems, and/or other important social and environmental factors and the outcome they are studying are over or under estimated.

Candidate Gene Analysis

A second approach used to integrate genetic information into social science research that gained a lot of attention from the late 1990s into the first decade of the 21st century is

candidate gene analysis. Candidate gene analysis revolves around the idea that the genetic influences on an outcome operate via a single, or, at most a few, specific genes. Specifically, candidate gene analysis uses observed measures of genetic variation at one or more specific sites in the genome as a proxy for all genetic influences on an outcome.

To better understand this approach, it's important that we review the structure of genome-wide data and define a few terms before continuing. Figure 1 depicts a simplified version of the double-helix structure of deoxyribonucleic acid (DNA). The blue bands spiraling up the image are the sugar-phosphate backbone of DNA. The "rungs" of the ladder are combinations of the nitrogenous bases Guanine (G), Cytosine (C), Thymine (T), and Adenine (A). Groupings of these base pairs represent the blueprints for the chemical compounds necessary for life, i.e. genes. The sequencing of these base pairs is constant over time, thus the frequency of nitrogenous bases are considered innate or immutable within an individual. Across all human populations, 99.9% of the approximately three billion base pairs in our DNA are identical. The 0.1% of base pairs that do vary are called single-nucleotide polymorphisms (SNPs, pronounced "snips"). Since we all have two sets of chromosomes, the number of copies of a reference allele—the G, C, T, or A a researcher chooses to measure for a specific SNP—across the two sets of chromosomes can be 0, 1, or 2. For a single SNP the number of copies of the reference allele a person has is referred to as an allele frequency. The measurement of allele frequencies at individual SNPs forms the basis of genome-wide data and thus represents the foundation of modern sociogenomic research (Guo and Adkins, 2008). Now that we're familiar with the essential components of genome-wide data let's return to the discussion of candidate gene analysis.

Candidate gene analysis relies on a priori biological theories and hypotheses derived from experimental animal studies and/or discoveries in human genetics linking the trait/behavior being studied to one or at most a few SNPs. The SNP(s) hypothesized to influence the trait are referred to as "candidate genes." Once candidate genes are selected, researchers then estimate the association between the candidate gene and the behavior/trait they are studying. A good example of candidate gene analysis in social stratification research is work by Shanahan and colleagues (2008) in which they investigate the differential impact of the gene *Taq1A* by an individual's social origins (e.g. household income and parental educational attainment). The gene *Taq1A* is known to have a strong negative impact on educational attainment, but Shanahan and colleagues (2008) found that the negative effect of *Taq1A* is fully attenuated by more advantaged social origins. In other words, the negative genetic effects of the *Taq1A* gene can be compensated for if individuals are raised in households in which parents are presumably more likely to be involved in their children's education and/or provide access to higher quality educational resources. This suggests that a significant gene-environment interaction may exist in how genetics influence educational attainment. Stated differently, genetic effects on educational attainment are not immutable, but the capacity to overcome genetic influences may depend on the social circumstances in which individuals are born. And while we cannot change an individual's genetics, policies can be tailored to compensate for negative genetic effects by increasing the quality and availability of resources for disadvantaged youth.

Unfortunately, while the idea that there is “a gene” for some trait/behavior is elegant, the empirical evidence suggests that the relationship between genetics and most human behaviors and traits is much more complicated, typically involving many small associations spread across hundreds if not thousands of SNPs (Chabris et al., 2015). In line with this finding is that fact that, while there are a few successful candidate gene studies, the vast majority of candidate gene analyses fail to replicate in other datasets.

The lack of replicability of candidate gene analyses across samples led to a substantial tempering in the enthusiasm with which these studies were first greeted. In fact, the issue of replicability was so severe that a few journals now have specific editorial policies and warnings about candidate gene analyses. For example, the editorial policy of *Behavior Genetics* states, “[a]s a result [of the rate of reporting false positives] ... it now seems likely that many of the published findings of the last decade [using candidate gene analysis] are wrong or misleading and have not contributed to real advance in knowledge” (Hewitt, 2012, p. 1). The failure of candidate gene analysis is likely due to a combination of relatively small sample sizes and the fact that most genetic effects are spread across many SNPs (Benjamin et al., 2012). The latest empirical evidence suggests that instead of a single candidate gene, or even genes, the genetic influences on most human traits and behaviors stem from a combination of SNPs across the genome (Chabris et al., 2015); when this occurs, the genetic association is said to be polygenic.

Despite its shortcomings, candidate gene analysis should not be dismissed completely. For the rare traits that are due to variation in one or at most a few genes, such as Huntington’s disease, candidate gene analysis provides a powerful tool for exploring biological and social relationships in the development of the trait. But, for most outcomes of interest to stratification scholars, this is not the case. Consequently, researchers must find another way to efficiently and appropriately account for the simultaneous association of many SNPs and the outcome they are interested in. Simplifying the complex relationships that arise when the genetic association with an outcome is polygenic is the primary purpose of the most recent methods and measures in sociogenomic research.

Section Two: Current Trends in Sociogenomic Research

Guided by evidence from behavioral genetics and candidate gene analyses and fueled by technological advancements in the collection of individual genome-wide data, the most recent trends in sociogenomic methods and measures rely on samples with genome-wide data from non-related individuals. Below I briefly discuss two such methods in sociogenomic research: genome-wide complex trait analysis and polygenic scores.

Genome-wide Complex Trait Analysis

Genome-wide complex trait analysis (GCTA) extends the same principle of estimating the proportion of variance in an outcome due to genetics from behavioral genetics analysis to genome-wide data (Yang et al., 2011). Whereas behavioral genetics models rely on known population averages of genetic similarities between related individuals, GCTA uses genome-wide data, i.e. allele frequencies for all SNPs in the dataset, to evaluate the genetic relatedness between pairs of unrelated individuals.

Despite its relatively recent development, researchers have already applied GCTA to a wide range of outcomes. Table 1 presents an updated list of GCTA studies for a variety of outcomes first compiled by Domingue et al. (2016). The results of the GCTAs listed in Table 1 generally follow the same pattern as estimates of heritability from behavioral genetics analyses.

GCTA can also be used to investigate the degree to which the same set of genetic variables influence multiple traits/behaviors, a phenomenon referred to as genetic correlation, or pleiotropy. If ignored, pleiotropy may further exacerbate bias due to the endogeneity problem. For example, Boardman and colleagues (2015) found that a large proportion of the association between education and self-rated health may be due to genetic effects that affect both educational attainment and self-rated health. These findings show that traditional analyses that omit sociogenomic methods/measures may report biased estimates in the association between education and health. If left unaddressed, over estimates of the relationship between education and health could result in public policies that over-estimate the public health benefits for increasing educational attainment. That is not to say that there are zero public health benefits from policies aimed at increasing population levels of educational attainment. But, rather, that the incremental cost-to-benefit analysis will be overstated if policies are based on estimates from analyses omitting the effects of both nature and nurture.

Polygenic Scores (PGS)

One of the most recent advances in sociogenomic measures is the calculation of polygenic scores (PGSs), sometimes referred to as polygenic risk scores or genetic risk scores. PGSs capture the combined genetic influence of SNPs across the entire genome on a specific trait/behavior in a single measure. Importantly, PGSs allow scientists to include a measure of genetic influences on one, or any number of traits/behaviors as just another variable in quantitative analyses already commonly used in social science research. The calculation of PGSs leverages summary statistics from genome-wide association studies (GWASs), which independently estimate hundreds of thousands, and sometimes millions, of regressions examining the association between allele frequencies at individual SNPs and a particular behavior or trait.

Due to the inclusion of all genotyped SNPs, whole genome PGSs capture genetic association from across the entire genome, but in so doing eliminate the possibility to test hypotheses related to specific biological pathways (Belsky and Israel, 2014). Consequently, whole genome PGSs are said to be hypothesis free measures of genetic influence on the outcome for which they are created. Whole genome PGSs are now available for both the Health and Retirement Study (Ware et al., 2018) and the National Longitudinal Study of Adolescent to Adult Health (Braudt and Harris, 2018). While other methods of choosing which SNPs to include in a PGS exist, whole genome PGSs tend to perform as well as, and in many cases better than, other PGSs (Ware et al., 2017).

Lastly, when calculating PGSs it's important to account for differences in genetic variation between different ancestry groups, a phenomenon referred to as population stratification. Due to the genetic bottleneck created by the small number of humans who migrated out of

Africa early in our history as a species, and the tendency for people to couple with individuals from the same, or nearby, geographical regions, genetic variance across the entire genome is highly correlated with geography.

Given the central role of race in many social stratification processes, it is important to emphasize that race and ethnicity are not the same as genetic ancestry. Race and ethnicity are social constructs that vary across and even within place, culture, and history. Key components of race and ethnicity as social constructs are social definitions of otherness based on physical appearance, religion, language, class, and historical power structures (Almaguer, 1994; Domínguez, 1986; Jung, 2006; Mills, 1997; Omi and Winant, 1994; Waters, 1990). Genetic ancestry, on the other hand, is based on an individual's progenitors and the degree of genetic variation they passed on to subsequent generations (Price et al., 2010, 2006). The two concepts have some overlap since, at least within the U.S. racial system, ancestry has historically played a role in defining racial and ethnic groups (Omi and Winant, 1994; Waters, 1990). But, race and ethnicity cannot be reduced to genetic ancestry and in no way is genetic ancestry equivalent to the social constructs of race and/or ethnicity. In an effort to account for population stratification, PGSs are typically calculated and standardized within genetic ancestry groups. A consequence of this standardization process is that comparing the effect of a PGS for one ancestry group to that of a PGS from another ancestry group becomes difficult to interpret.

As with the other methods in this review, evidence from the integration of PGSs in analyses of social stratification outcomes suggests that nature and nurture influence most human behaviors and traits. For example, recent research employs PGSs to study stratification processes in educational outcomes. Domingue and colleagues (2015), show that a PGS for educational attainment is predictive of total years of education in early adulthood, even after taking into account childhood environments and within sibling pair differences. Schmitz and Conley (2016) show that genetic influences on educational attainment moderate the impact of educational benefits from military service, i.e. use of the G.I. bill, for veterans of the Vietnam War. Specifically, Schmitz and Conley find that individuals with a higher relative genetic propensity to complete more education who also served in the Vietnam War were more successful at using the G.I. bill to complete more education than individuals with the same genetic propensity who lacked access to the resources the G.I. bill provided. This suggests that policies aimed at reducing socio-demographic barriers to educational opportunities may increase educational mobility for individuals with the same genetic propensity but who otherwise lack the necessary resources to enter higher education.

In a study investigating the role of parental education in social mobility, Conley and colleagues (2015) find evidence of dual systems of inheritance, one based on genetic inheritance and the other based on the inheritance of social status and/or resources. The nature side of the inheritance is driven by genetic predispositions passed on from parents at conception. The other system of inheritance derives from a social inheritance reflecting differences in parental income, education, and wealth. Liu (2018) identifies a similar duality of inheritance in analyses spanning three generations.

Moving beyond educational outcomes, Belsky et al. (2016) find that, net of observed educational levels, a PGS for educational attainment is predictive of a host of outcomes including occupational prestige, income, assets, and social mobility. Similarly, Papageorge and Thom (2017) find that the social and physical environments that promote educational success, such as greater parental involvement and access to tutors, can compensate for a lower genetic predisposition to graduate from high school. But, those same socio-environmental factors enhance genetic endowments for higher educational outcomes such as college completion. Papageorge and Thom's (2017) findings suggest that while genetic predispositions to succeed in educational settings may offset some social disadvantages in high school, the same cannot be said about college completion. Taken together, the evidence from these studies indicate that ignoring genetic influences on educational stratification would bias findings; which may in turn lead to a less effective allocation of public resources meant to provide an equality of opportunity for more disadvantaged groups.

Previously, the complexity and requisite specialization necessary to employ sociogenomic methods presented a formidable barrier to their use in social science research, but PGSs are relatively easy to calculate and can be included in most quantitative analyses just like other continuous variables. Furthermore, many datasets commonly used by social scientists have calculated, or are in the process of calculating and releasing, PGSs for a variety of behaviors and traits relevant to research in social stratification and inequality. Lastly, as the examples discussed above indicate, PGSs allow researchers to move beyond simply controlling for the influence of genetics to investigate possible gene-environment interactions and how those interactions may vary across different levels of an outcome and/or by various stages of the life course.

Findings from GCTAs and analyses using PGSs add to the growing evidence that most behaviors and traits are significantly influenced by nurture and nature. This doesn't mean that studies that fail to account for genetic influences, or conversely studies that fail to include important social and environmental factors but investigate genetic associations, should be ignored. Such studies, although reporting potentially biased findings, illuminate many of the potential pathways through which nature and nurture operate in important aspects of daily life. Additionally, these studies provide a launching point from which a new generation of social scientists can investigate the robustness of foundational theories in stratification research when sociogenomics are taken into account.

Conversely, advances in sociogenomic methods and measures provide opportunities for social scientists to critically engage with recent findings concerning significant genetic associations in health, education, and other domains by investigating the robustness of those associations in the presence of different social systems (e.g, systemic racism, class hierarchy, and gender norms). In short, while sociogenomic methods and measures can be used to investigate the effect of nurture net of nature, or conversely nature net of nurture, one of the most exciting areas of research in coming decades will likely be the investigation of how nurture modifies the effects of nature (i.e. gene-environment interactions) and how nature may modify the experience of nurture (i.e. gene-environment correlations).

Section Three: The need for an Integration of Sociology and Sociogenomics

Throughout the preceding sections, I review many sources of empirical evidence indicating the existence of non-trivial associations between genetics and most human traits and behaviors, implying that investigations of stratification processes must consider nature *and* nurture, or risk reporting biased findings. In the following, I discuss some areas in which sociogenomics could benefit from sociological insights, including (1) the identification and interpretation of gene-environment interactions versus gene-environment correlations and (2) distinguishing when, or if, variables are subject to social construction.

Gene-environment interactions refer to the general idea that social and/or environmental factors may condition genetic effects on an outcome (Adkins and Vaisey 2009; Conley 2016; Shanahan and Boardman, 2009). Figure 2 presents a conceptual model of gene-environment interactions in which a path diagram depicting the causal relationship between genetic influences and educational attainment is conditioned by social and environmental contexts.

A related concept to gene-environment interactions is gene-environment correlation. Whereas gene-environment interactions refer to the conditioning of the genetic effects on an outcome by social and environmental factors, gene-environment correlations refer to dynamic processes linking genes to context to behavior (Shanahan and Boardman 2009:223). Gene-environment correlations reflect causal processes in which social factors intercede between genetic effects and an outcome. Stated differently, gene-environment correlations occur when genetic propensities for, or against, a behavior co-occur with specific social systems and/or environments, which in turn causes change in the outcome being studied.

Failing to consider gene-environment correlations can bias estimates of gene-environment interactions. For example, if the majority of individuals with a lower genetic propensity for succeeding in educational settings also lack access to quality educational resources (e.g., motivated teachers, computer labs, etc.), it would be difficult to test the hypothesis that access to higher quality educational resources attenuate genetic effects on educational attainment since there is a strong gene-environment correlation. In this hypothetical example, the estimate of the gene-environment interaction would be inflated. Conversely, if nearly every one with a lower genetic propensity for succeeding in educational settings came from more advantaged social origins, and thus had increased access to higher quality educational resources, then an examination of gene-environment interactions in educational attainment would be downwardly biased.

A sociological perspective may prove particularly powerful in distinguishing between gene-environment interactions and gene-environment correlations. Consider the classic example of two groups of people where individuals in the first group have a unique genetic variant and also use chopsticks due to their cultural heritage, while individuals in the second group have neither the genetic variant nor use chopsticks. Without a proper understanding of the social environment it would be easy to confuse this gene-environment correlation as a gene-environment interaction and conclude that the genetic variant unique to individuals in the

first group causes chopstick use. Conversely, consider the classic hypothetical example of two individuals with the same genetic propensity for aggressive behavior who are born into families on opposite ends of the socioeconomic spectrum. It is easy to image that the genetic propensity for aggressive behavior would lead the individual born into more advantage to a job as an entrepreneur or CEO while that same propensity for aggressive behavior could result in a life of crime and a possible prison sentence for the individual born into relative disadvantage. If researchers consider only the negative outcome without considering the context, they may conclude that the genetic predisposition to aggressive behavior was the cause of the second individual's tenure in the criminal justice system when in fact the social environment was likely the primary factor leading to incarceration. As these examples demonstrate, the distinction between gene-environment correlations and gene-environment interactions often requires considerations of broader social and political circumstances, a task for which sociological training is particularly apt.

Sociological theory has the potential to clarify our understanding of what is being measured in sociogenomic analyses. For example, a sociological lens is well suited for distinguishing race from genetic ancestry, gender from biological sex, and placing measures, such as educational attainment and cognitive function, in the context of how each measure was developed. Similarly, most sociological training emphasizes considering the degree to which such measures may, or may not, generalize to other populations, and how each of these affect the interpretation of analyses using such measures. These examples may seem obvious to sociologists, but that is precisely because they have received sociological training and already see the world through a sociological lens, something that even distinguished professors in other disciplines can find difficult due to their own specialization. Simply put, the training and education scientists receive can, and in most cases does, tint the way we view research design, the interpretation of results, and the meaning of variables. Consequently, sociologists cannot expect scientists trained in other fields to immediately see through a sociological lens. Instead, sociologists must actively engage with research at the intersection of genetics and social stratification if they wish to influence the accuracy and direction of a rapidly developing area of research that could benefit greatly from sociological insights.

Conclusions

Robert Alford (1998, pp. 125–126) eloquently wrote that, “attempts to answer a research question should not be based solely on traditions that have become isolated within fragmented ‘subfields.’” Together with the evidence that clearly indicates that most human behaviors and traits are significantly influenced by genetics, the increasing availability of genome-wide data and PGSs suggests that it is well past time to reach across subfields and broaden the scope of research questions that we as sociologists ask. In fact, the continued success of research on social stratification and inequality will likely be contingent on the willingness of social scientists to actively critique, improve, and engage with sociogenomic data and methods in their own research. Furthermore, it is likely that as more sociologists incorporate sociogenomic measures and methods into their research that entirely new areas of research will be uncovered, including the discovery of unique ways in which genetics and the environment interact to produce the diversity of outcomes observed in society. But in

order for any of that to happen, we must first overcome a general aversion to integrating data on the genetic material that underlies our human bodies with the study of how those bodies are differentially treated in the social world.

To be fair to sociologists, their hesitancy to embrace research attempting to integrate genetics and the study of social stratification is not without reason, such as the appropriate rejection of eugenic philosophies in much of the early behavioral genetics research. But the simple fact is that the genomic revolution is here and cannot be ignored. The increasing availability of genomic data means that, independent of sociologists' engagement with the genomic revolution, scientists are now investigating the types of questions proposed by Herrnstein and Murray (1994). And while a few sociologists are currently participating in these debates, if sociology as a discipline wishes to influence the direction, interpretation, and use of genetic data in the study of social stratification and inequality, more sociologists must begin to integrate sociogenomic measures and methods into their own research.

Contrary to claims by a few prominent sociologists (e.g. Fischer, 1996), the application of sociogenomic methods and measures to stratification research can help us better understand the origins of inequality as well as the mechanisms that have led to its growth in much of the world over the past few decades. Questions of how genetics factor into the origins and growth of inequality are no longer limited to philosophical debates, or to the restrictive methodological assumptions of behavioral genetics. The combination of sociogenomic methods and measures with a sociological lens opens up a world of potential questions to empirical investigation. And while this research has, and will likely continue, to show a direct association between genetics and many of the outcomes studied in stratification research—i.e. that nature has a significant effect net of nurture—there are many possible ways in which those associations are likely modified by different social systems (Adkins and Vaisey, 2009; Hahn et al., 2016; Nielsen, 2016; Schulz et al., 2017). The documentation of gene-environment interactions, as well as gene-environment correlations, will only add to the evidence that social facts play a profound role in shaping individual outcomes and trajectories.

It is imperative to the advancement of our understanding of the social world and the proper development, and evaluation, of public policy that sociologists seek out opportunities to incorporate sociogenomic measures and methods in their work. Yet, despite the mounting evidence, ideologically driven skepticism and even outright opposition to genetically informed social science will likely continue. Anticipating such an ideological showdown, the editor of *Cell Reports* recently stated that, “Quantitative genetics is bringing powerful tools to old questions, including some deemed sacred or hopelessly complex. More drama is certain to come. Be sure to get a good seat” (Matheson, 2017, p. 1157). Indeed, there will likely be heated debates over competing interpretations of findings, but the only way sociologists will have a significant voice in those debates is if we actively engage with the genomic revolution and submit ourselves to the embrace of the best contemporary science has to offer.

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Figure 1.
Simplified Structure of Deoxyribonucleic Acid (DNA)
Source: “Talking Glossary of Genetic Terms” (genome.gov).

Figure 2.
Gene-environment Interactions in Educational Attainment

Table 1

Examples of Recent Heritability estimates from Genome-wide Complex Trait Analysis

Outcome	SNP Heritability (SE)	Sample Size	Reference
<i>Socioeconomic Traits/Behaviors</i>			
Education (years)	0.17 (0.07)	6,414	Conley et al., 2014
Education (years)	0.21 (0.05)	6,578	Marioni et al., 2014
Education (years)	0.33 (0.10)	4,233	Boardman et al., 2015
Education (years)	0.15 (0.02)	20,450	Okbay et al., 2016
Educational achievement	0.31 (0.12)	3,152	Krapohl and Plomin, 2016
Reading Achievement Test	0.43 (0.10)	3,680	Robinson et al., 2015
Multiple Deprivation Index	0.18 (0.05)	6,533	Marioni et al., 2014
Family Socioeconomic Status	0.20 (0.11)	3,152	Krapohl and Plomin, 2016
Socioeconomic Status (Age 2)	0.18 (0.12)	3,000	Trzaskowski et al., 2014
Socioeconomic Status (Age 7)	0.19 (0.12)	3,000	Trzaskowski et al., 2014
<i>Health Behaviors/Traits</i>			
Moderate to Vigorous Activity	0.17 (0.09)	4,244	Richmond et al., 2014
Sedentary Time	0.25 (0.09)	4,244	Richmond et al., 2014
Total Physical Activity	0.21 (0.10)	4,244	Richmond et al., 2014
Drug Use	0.22 (0.16)	3,452	Vrieze et al., 2013
Drug Dependence	0.36 (0.13)	2,596	Palmer et al., 2015
Dependence Vulnerability	0.33 (0.13)	2,596	Palmer et al., 2015
Problematic Drug Use	0.25 (0.13)	2,596	Palmer et al., 2015
Alcohol Consumption	0.16 (0.16)	3,452	Vrieze et al., 2013
Alcohol Dependence	0.12 (0.16)	3,452	Vrieze et al., 2013
Nicotine Use/Dependence	0.18 (0.16)	3,452	Vrieze et al., 2013
<i>Psychological Traits/Behaviors</i>			
Subjective Well-Being	0.05–0.10 (0.05–0.10)	11,500	Rietveld et al., 2013
Reporting Stressful Life Events	0.30 (0.15)	2,578	Power et al., 2013
Bipolar Disorder	0.37 (0.04)	2,599	Lee et al., 2011
Bipolar Disorder	0.59 (0.06)	2,000	Speed et al., 2012
ADHD	0.42 (0.13)	1,040	Yang et al., 2013
ADHD Combined Scale (Parent report)	0.22–0.40 (0.14)	5,928	Pappa et al., 2015
ADHD Inattentive Scale (Parent report)	0.24–0.37 (0.14)	5,928	Pappa et al., 2015
Hyperactive-Impulsive (Parent report)	0.33–0.45 (0.14–0.15)	5,928	Pappa et al., 2015
Externalizing Scale (Teacher report)	0.44–0.46 (0.22–0.23)	5,928	Pappa et al., 2015
Attention Problems (Teacher report)	0.49–0.71 (0.22)	5,928	Pappa et al., 2015
Adult Antisocial Behavior	0.55 (0.41)	2,172	Tielbeek et al., 2012
Depression	0.19 (0.10)	4,233	Boardman et al., 2015
Major Depressive Disorder	0.32 (0.09)	4,605	Lubke et al., 2012
Behavioral Disinhibition	0.19 (0.16)	3,452	Vrieze et al., 2013

Outcome	SNP Heritability (SE)	Sample Size	Reference
Neuroticism	0.06 (0.03)	12,000	Vinkhuyzen et al., 2012
Neuroticism	0.15 (0.08)	4,855	Power and Pluess, 2015
Borderline Personality Features	0.23 (0.09)	7, 125	Lubke et al., 2014
Callous-Emotional Behavior	0.07 (0.12)	2,930	Viding et al., 2013
Extraversion	0.12 (0.03)	12,000	Vinkhuyzen et al., 2012
Anxiety Related Behaviors	0.01–0.12 (0.12)	2,810	Viding et al., 2013
Openness	0.21 (0.08)	4,855	Power and Pluess, 2015
Schizophrenia	0.39 (0.12)	1,097	Goes et al., 2015
Loneliness	0.27–0.16 (0.12–0.06)	7,556	Gao et al., 2016
Aggressive Behavior (Age 4)	0.10 (0.06)	5,505	Pappa et al., 2016
Aggressive Behavior (Early Adolescence)	0.12 (0.06)	5,299	Pappa et al., 2016
<i>Physiological Traits/Behaviors</i>			
Self-Rated Health	0.18 (0.10)	4,233	Boardman et al., 2015
Self-Rated Health	0.13 (0.01)	112,151	Harris et al., 2017
BMI	0.31 (0.07)	6,320	Conley et al., 2014
BMI	0.43 (0.10)	4,233	Boardman et al., 2015
Type 1 Diabetes	0.28 (0.04)	2,599	Lee et al., 2011
Type 1 Diabetes	0.73 (0.06)	2,000	Speed et al., 2012
Type 2 Diabetes	0.35 (0.06)	2,000	Speed et al., 2012
Coronary Artery Disease	0.39 (0.06)	2,000	Speed et al., 2012
Cholesterol High Density Lipoprotein	0.45 (0.02)	19,977	Zaitlen et al., 2013
Cholesterol Low Density Lipoprotein	0.20 (0.06)	4,547	Zaitlen et al., 2013
Hypertension	0.42 (0.06)	2,000	Speed et al., 2012
Age at Menarche	0.44 (0.02)	15,150	Zaitlen et al., 2013
Age at Menopause	0.40 (0.05)	5,540	Zaitlen et al., 2013
Total children	0.10 (0.02)	15,000	Zaitlen et al., 2013
Rheumatoid Arthritis	0.57 (0.06)	2,000	Speed et al., 2012
Crohn's Disease	0.61 (0.08)	2,599	Lee et al., 2011
Crohn's Disease	0.54 (0.06)	2,000	Speed et al., 2012
Pediatric Obesity	0.37 (0.15)	3,152	Llewellyn et al., 2013
Parkinson's Disease	0.27 (0.05)	7,096	Keller et al., 2012
Parkinson's Disease	0.22 (0.02)	3,426	Do et al., 2011
<i>Cognitive Traits/Behaviors</i>			
General Cognitive Ability	0.35 (0.12)	3,154	Plomin et al., 2013
General Cognitive Ability	0.29 (0.05)	6,609	Marioni et al., 2014
Nonverbal Cognitive Ability	0.20 (0.11)	3,154	Plomin et al., 2013
Nonverbal Reasoning	0.41 (0.10)	3,657	Robinson et al., 2015
Verbal Cognitive Ability	0.26 (0.11)	3,154	Plomin et al., 2013
Verbal Memory	0.24 (0.10)	3,638	Robinson et al., 2015

Outcome	SNP Heritability (SE)	Sample Size	Reference
Language Ability	0.29 (0.12)	3,154	Plomin et al., 2013
Language Reasoning	0.30 (0.10)	3,651	Robinson et al., 2015
Spatial Reasoning	0.36 (0.10)	3,581	Robinson et al., 2015
Intelligence (Age 7–12)	0.60 (0.26)	2,875	Trzaskowski et al., 2014a
Intelligence	0.51 (0.02)	3,511	Davies et al., 2011
Intelligence from Childhood to Old Age	0.24 (0.20)	1,940	Deary et al., 2012
IQ (Age 12)	0.32 (0.14)	3,000	Trzaskowski et al., 2014b
IQ (Age 7)	0.28 (0.17)	3,000	Trzaskowski et al., 2014b

Source: Updated and modified from Domingue et al. (2016)