Sociological Theory

Toward a Unified Stratification Theory: Structure, Genome, and Status Across Human Societies*

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While social scientists and geneticists have a shared interest in the personal characteristics instrumental to status attainment, little has been done to integrate these disparate perspectives. This is unfortunate, as the perspectives offer complementary insights, which, if properly combined, stand to substantially improve understanding of the stratification process. This article synthesizes research from the social sciences and genetics to develop a multistage theory of how social structure moderates the influence of the genome on status outcomes. Its thesis is that the strength of the genome's influence on status is primarily moderated by two properties of social structure—levels of resource inequality and social mobility. Thus, it is theorized that under conditions of low inequality and high social mobility, the influence of the genome on status will be high relative to conditions of high inequality and low social mobility. The essential logic is (1) as inequality increases, the characteristics and abilities intrinsically useful in status attainment are increasingly influenced by individuals' social backgrounds and decreasingly determined by their genomes; and (2) as social closure and inequality increase, the utility of these characteristics and abilities to status attainment is diminished. In sum, a model of status attainment is developed proposing that while both genome and social background influence the status attainment process, the relative importance of these factors is determined by the surrounding structure of the society.

What determines one's status in society? This seemingly simple question has been debated since the beginnings of civilization with detailed discussions in texts including the Confucian *Analects*, Judeo-Christian *Bible*, Vedic *Laws of Manu*, and classical Greek works such as Aristotle's *Politics* and Plato's *Republic*. Discourse on status and stratification has remained prominent in the modern era, where it has been foundational to the formation of sociology as a discipline (Marx 1963; Durkheim 1984; Weber 1946, 1947). The discussion of these issues within sociology has largely been a continuation of the dialogue throughout history, which Lenski (1966) describes as a dialectic between two primary positions: the thesis that the unequal distribution of resources is just, equitable, and frequently inevitable and the antithesis that the distributive system is basically unfair and unnecessary.

In the contemporary academic world, this division is embodied by the divide between genetically inspired studies of inequality on the one hand, and sociological

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stratification research on the other. These disparate approaches to understanding social outcomes and behavior have been a source of conflict within sociology, as attempts to bring notions of innate dispositions or abilities into our disciplinary framework have often been met with skepticism or even hostility (e.g., Miller and Costello 2001; Udry 2000; Fischer et al. 1996). In many cases, this hostility is quite understandable, since some scholars enamored of genetic or biological approaches to the study of stratification have been quite forthcoming about their reductionist agenda (e.g., Herrnstein and Murray 1994; Jensen 1998). Nevertheless, research in human genetics has found robust and consistent evidence of *some* connection between genotype and social outcomes. Though twin and adoption studies and the specific heritability estimates they produce can—and should—be regarded skeptically, there are nevertheless patterns that can be considered nonnegligible (Freese 2006; Guo 2005). Furthermore, links between specific alleles and a wide variety of socially relevant outcomes, even quite complex phenotypes like continuing on to tertiary education, have been documented (Shanahan, Erickson, Vaisey, and Smolen 2007). There is little reason to believe that this entire line of research will be debunked anytime soon. At the same time, however, decades of sociological research have found equally compelling evidence of social influences on the same outcomes (Jencks 1972; Sewell and Hauser 1975; Bourdieu and Passeron 1977; Lareau 2003). Factors such as family background, parent's education, occupation and income, race, and gender have been shown to play a key role in shaping future trajectories of social position and status. Again, it seems highly unlikely that genetic explanations will arise to negate this large body of social research.

This leaves us in a less-than-desirable intellectual position: we have two competing strains of research on the same phenomenon, each wary of the other, and each unsure about how to incorporate the other into its own logic. Some in each field are willing to say that we need both social and genetic explanations, and some account of how they interact. But as with other thorny theoretical problems like "structure and culture" or "structure and agency," simply saying "both/and" accomplishes very little; in fact, such a "solution" may be worse than none at all because it provides an illusion of intellectual integration that is not, in fact, present (Martin 2003). What are needed are not high sounding assertions but rather a theoretical framework that can account for *how* and *when* genetic and social factors might interact.

As one step toward such a framework, we build on previous work in this area (e.g., Guo and Stearns 2002; Shanahan and Hofer 2005) to present a theoretical model that can more coherently account for how genetic and social factors influence each other in the process of status attainment. We proceed in five steps. First, we theorize which traits are most useful in attaining status. Second, we discuss genetic and sociological accounts of the status attainment process, initially contrasting and then reconciling these perspectives in the context of contemporary American society. Third, we cast the integrated model into comparative context, building on previous work on gene-environment interactions to argue that genetic effects on the formation of ability (and its subsequent utility) will be highly contingent upon social structural factors. We emphasize the role that inequality and social closure/mobility likely play in this conditioning. Fourth, we synthesize the insights of the previous sections to advance a model of status attainment that incorporates both genetic and social factors into a single model. Fifth, we discuss the implications of the model to both sociological research and genetics, outlining a research agenda and noting some important methodological considerations. In sum, we develop a detailed model of the social structure-genetics nexus in the area of stratification, suggesting that not

only does the actualization of genetic potential vary across the strata of a single society (Guo and Stearns 2002), but the magnitude of this variation is contingent on societal characteristics that lie at the core of sociological inquiry.

The Determinants of Status

The study of status attainment, broadly conceived, has been a major substantive thread of continuity across generations and intellectual currents in sociology, with the issue's contemporary conceptualization largely beginning with research by Blau and Duncan (1967) and sociologists at the University of Wisconsin. This research investigated the influence of social background, cognitive ability, and social psychological variables on various indicators of status attainment, producing what has been the principal model of status attainment research for several decades (e.g., Sewell, Haller, and Portes 1969; Hauser, Warren, Huang, and Carter 2000). A primary objective of this research program has been to evaluate the influence of personal ability versus ascribed characteristics on status outcomes. After four decades of status attainment research, it is now well established that both social background and personal ability are significant determinants of status (e.g., Sewell and Hauser 1975; Jencks and Bartlett 1979; Jencks and Phillips 1998; Hauser et al. 2000). However, while advancing the field considerably, conventional status attainment models have been limited by a narrow conceptualization of personal ability as only cognitive ability. This limitation has been addressed in more recent research demonstrating a wider range of personal characteristics to influence status attainment including personality traits (Farkas 2003; Bowles and Gintis 2002) and health (Smith 1999, 2004). Cumulatively, this body of research raises important questions regarding what personal characteristics are generally influential in status attainment and the analytical distinction of these personal characteristics from "ascribed" characteristics such as social background.

As suggested above, identifying the determinants of status attainment is crucial to any attempt at a comprehensive stratification model. To this end, we highlight the longstanding differentiation of status determinants into two categories: social background characteristics and personal characteristics (hereafter referred to as *ability*). This distinction is made on the basis of whether the determinant is intrinsic or extrinsic and whether it has direct or instrumental utility to status attainment. Thus, social background factors include status determinants that are either extrinsic to the individual (e.g., wealth and social capital) or have instrumental or symbolic significance (e.g., race, gender, and class background). Conversely, ability refers to intrinsic, inalienable characteristics that have general, direct utility to status attainment. Thus, for a characteristic to be considered ability it must be a physical, behavioral, or psychological trait of the individual that has direct utility in achieving status across a wide variety of contexts. This raises the question: What exactly are these characteristics? In answering this we rely on recent status attainment literature, which suggests a number of ability traits that are consistent with intuition, including cognitive ability, physical and mental health, and personality traits such as emotional stability and conscientiousness.[1] Below we elaborate the logic for including each of

[1]We recognize that this list of ability traits may not be exhaustive and hope future research will improve this specification. While we have chosen what we consider the most theoretically sensible and empirically supported core of ability traits, this list may be expanded without contradicting the essential logic of our thesis as long as some of the traits specified as ability are partially functions of the genome.

these factors and discuss empirical research showing that each is important in the attainment process.

Cognitive ability has long been a standard variable in both sociological and economic models of status attainment. While many economists have motivated this inclusion by focusing on employer preferences for bright workers, we find equal motivation in the argument that if income, wealth, and the other trappings of status are generally desirable, and obtaining these desirables is a problem with better and worse solutions, then those with superior problem solving, or cognitive, ability will tend to be more successful than those of lesser cognitive ability (see Bowles and Gintis [2002] for discussion). But regardless of the mechanism mediating the association of cognitive ability to status, the association itself has been repeatedly substantiated in stratification research (e.g., Sewell, Haller, and Portes 1969; Sewell and Hauser 1975; Jencks 1972; Jencks and Phillips 1998; Hauser et al. 2000; Warren, Hauser, and Sheridan 2002) such that leading experts have recently stated: "The independent importance of . . . cognitive functioning as [an] earnings determinant is uncontroversial" (Bowles, Gintis, and Osborne 2001a). To cite just one of the multitude of studies demonstrating the effect of cognitive ability as measured by aptitude tests, Bowles, Gintis, and Osborne (2001a) present results from two large, nationally representative studies (the National Longitudinal Study of Young Women and the National Child Development Study) showing highly significant, positive effects for cognitive ability on earnings, controlling for years of education, school quality, and parental socioeconomic status (SES), as well as various dimensions of personality.

The influence of personality traits on status attainment has long been the topic of active research in psychology and, to a lesser extent, economics and sociology (e.g., Farkas 2003; Bowles, Gintis, and Osborne 2001b; Osborne Groves 2005). The rationale driving this research is intuitive—individuals who are conscientious and emotionally stable may reasonably be expected to be more effective and productive than those who are not, or as put by the leading experts in the area: "it is hard to conceive of a job where it is beneficial to be careless, irresponsible, lazy, impulsive and low in achievement striving (low conscientiousness) . . . Similarly, being anxious, hostile, personally insecure and depressed (low emotional stability) is unlikely to lead to high performance . . ." (Barrick, Mount, and Judge 2001). These hypotheses have received tremendous support as shown by a recent second-order meta-analysis of virtually all studies ever published on the association of primary personality characteristics to job performance. Examining data from approximately 450 studies with a total sample size of over 75,000, Barrick, Mount, and Judge (2001) show definitive evidence that conscientiousness and the absence of neuroticism promote effective job performance as measured by various criteria, including salary level and promotions. Consistent findings have also been presented by other prominent researchers, including James Heckman (Heckman and Rubinstein 2001), Greg Duncan (Dunifon and Duncan 1998), and Christopher Jencks (Jencks and Bartlett 1979). Thus, while there are indications that the effect of some personality traits (e.g., aggression) vary by class and gender (Bowles, Gintis, and Osborne 2001a, 2001b), there is very strong evidence that conscientiousness and neuroticism have consistent, robust effects on status attainment.

The influence of physical and mental health on status has also been the subject of extensive empirical investigation. Indeed, the significant positive correlation of health and SES is one of the most consistent empirical findings in the social sciences over the last 50 years (see Haas [2006] for review). However, the causal direction of this effect has been the subject of considerable debate. While much of the research to date has used cross-sectional, observational data incapable of supporting strong causal

inference, there are several studies employing methodologically rigorous designs indicating support for both social selection (health → status) and causation (status → health). Regarding the evidence of social selection, Smith (1999), using longitudinal, nationally representative data from both the Health and Retirement Survey and the Asset and Health Dynamics of the Oldest Old survey, demonstrates that accounting for prior health status, the exogenous shock of experiencing a new, severe health problem results in an average wealth reduction of about $17,000 over the following year, while also noting that since these health problems may persist into the future, the eventual impact on wealth is likely to be even larger. Similar results have been found in other studies employing comparable methods (e.g., Haas 2006; Smith 2004; Chirikos and Nestel 1985). While not precluding the presence of social causation effects, these results provide substantial corroboration of the intuition that illness is apt to reduce status through reducing work capacity while increasing expenditures.

Recognizing the significance of ability to status attainment raises the important question: What are the origins of these ability traits? Answering this question carries several serious implications, including indicating the relative influence of social and genetic factors in status attainment. Indeed, these implications have not been lost on stratification researchers of any disciplinary or ideological bent. Consequently, the question of the origins of various ability traits have been hotly contested for many years, and there are coherent but seemingly contradictory explanations, each replete with volumes of supporting evidence, for both social and genetic ontogeny.

SYNTHESIZING CURRENT ACCOUNTS OF ABILITY AND STATUS

Few would dispute the notion that there are some social and some genetic factors that facilitate success in the status attainment process. No one would deny, for instance, that having wealthy parents is an influential factor in producing economic success. Conversely, few would deny that the presence or absence of severe, heritable diseases would also influence one's ultimate status. Yet beyond these obviously social and obviously genetic factors, the origins of the ability characteristics that are widely held to be the proximate causes of status attainment are debated. In this section, we briefly review sociological and genetic research on ability characteristics. Our goal is not to provide a comprehensive review, since these can be found elsewhere (Bowles, Gintis, and Osborne Groves 2005; Morgan, Grusky, and Fields 2006; Plomin et al. 2003). Rather, our objective is to ascertain the overall logic of each approach to understanding the process of status attainment with an eye toward their subsequent integration.

The Sociological Account

On the social ontogeny side of the debate, the individual factors that matter most for success are generally viewed as the products of social processes. In a step beyond the descriptive accounts of associations in the status attainment literature, various theories of "capitals" have arisen to explain the mechanisms linking social background and respondent outcomes. For instance, cognitive ability (a form of human capital) is held to come from access to and participation in education and training (Jencks and Phillips 1998; Ceci and Williams 1997; Winship and Korenman 1997) and cultural capital comes from the investments made by privileged parents (Bourdieu and Passeron 1977; Lareau 2003). Thus, the advantages well-off children enjoy are not simply through the mechanisms of patronage and direct influence; they actually become "smarter" and "savvier" than their less-advantaged peers because

they have benefited from extensive investment on the part of their parents. These advantages extend beyond obvious traits such as knowledge and connections into physical health and mortality (Pearlin 1989; Link and Phelan 1995), mental health (Turner and Lloyd 1999; Turner, Wheaton, and Lloyd 1995), and various personality dimensions such as conscientiousness (Kohn and Schooler 1983; Dumais 2002; Roberts and Bogg 2004; Robins, Caspi, and Moffitt 2002), all factors that can be "capitalized" in the sense that they serve as resources that promote status attainment. Thus, by this account, status inequalities are reproduced via the transmission of a wide variety of status-relevant advantages from parents to children via direct socialization and the class-based contexts associated with parental characteristics. These advantages then become the proximate mechanisms in the attainment of individual status.

The Genetic Account

The origins of personal characteristics influential in status attainment have also been the focus of extensive research by geneticists. Genetic research into these topics has employed two primary approaches, biometric models that analyze covariance on the traits of interest among twins, adoptees and other family members, and molecular ones that examine the association of DNA variation to these traits. Overall, results from these genetic approaches have converged in lending evidence for significant genetic influence on status-influential personal characteristics. For instance, the etiologies of innumerable Mendelian disorders[2] have been mapped and reliable susceptibility genes for complex pathologies have also been found.[3] Molecular genetic research into complex psychological traits including various psychopathologies, cognitive ability, and personality traits have also identified susceptibility genetic loci (e.g., Lopez et al. 2005), but progress in this area has been slow due to the latent nature and complex origins of these phenotypes. Consequently, most established genetic findings on psychological characteristics rely on more longstanding, biometric methods that estimate the contribution of additive genetic factors to the outcome trait variance (i.e., the *heritability* of the trait). For instance, an influential meta-analysis published in the journal *Nature* of 212 studies of cognitive ability estimate heritability ranging from 47 to 68 percent depending on model specification (Devlin, Daniels, and Roeder 1997).

Ample research into the heritability of personality dimensions has also been conducted, and the results of this research have consistently indicated that conscientiousness and neuroticism are heritable in 35–50 percent range. In one well-cited study, Jang, McCrae, Angleitner, Riemann, and Livesley (1998) analyzed revised NEO Personality Inventory data collected in two independent twin samples, one in Germany and the other in Canada, with a total sample of approximately 2,000 respondents and found that heritability was consistent across the two samples and averaged approximately 35 percent and 42 percent for contentiousness and neuroticism traits, respectively. Overall, the results of conventional biometric genetic (often referred to as *behavior genetic*) studies on the origins of ability characteristics are quite easy to summarize. As stated by behavior geneticist Turkheimer (2000), virtually all traits,

[2]Such as X-linked muscular dystrophies (Koenig et al. 1987) and cystic fibrosis (Riordan et al. 1989). A complete compendium of Mendelian disorders has been compiled and made accessible at the NIH Online Mendelian Inheritance in Man (OMIM) database at: www.ncbi.nlm.nih.gov/omim/.

[3]Including the apoE locus, which has been shown to influence Alzheimer's disease and various cardiovascular impairments in over 100 studies (Malley and Rall 2000).

including ability, are substantially heritable (around 50 percent of outcome variance accounted for by genetic factors) and the effect of being raised in the same family is smaller than the genetic effect and often close to zero. In other words, the orthodox view among behavior geneticists is that genes are quite influential on ability traits and that many of the variables of interest to sociologists, such as family background and social origins, have virtually no effect on the development of these characteristics.

A Static Integration

As the above review has made clear, there is disagreement between sociological and orthodox behavior genetic perspectives regarding the source of observed differences in ability that must be reconciled in order to integrate findings of these literatures into a unified framework. However, the fundamental incompatibility here is actually fairly limited—while most sociologists focus on the social causation of ability, many within the discipline recognize that genetics may also play a role (see Freese, Li, and Wade 2003). Similarly, within molecular genetics there is a burgeoning realization that for complex disorders like depression or cognitive ability, genetic influence is likely to act through gene-environmental interactive paths and failure to model this interaction significantly weakens the ability to detect genetic effects (Moffitt, Caspi, and Rutter 2005). Indeed, the only essential incompatibilities between these literatures are (1) the blanket dismissal of all biometric heritability estimates among some sociologists (e.g., Conley 2006) and (2) the assumption within orthodox behavior genetics that characteristics shared within a household, such as parental income and educational attainment, are generally irrelevant to the development of ability (Turkheimer 2000). In order to reconcile the apparent incompatibility of genetic and sociological perspectives, we consider the common criticisms of conventional biometric genetic methods and also new, more sophisticated directions in genetics that attempt to incorporate social environment. By doing so we intend to demonstrate that although heritability is a limited tool, it is not a worthless one, while also showing that new movements in genetics are dismantling the assumption that shared environment does not matter.

Biometric methods, which analyze twin, adoptee, and other family data to estimate trait heritability, have long been the methodology of choice in behavior genetics. But while these methods are generally considered legitimate within behavior genetics, they have been frequently criticized outside the discipline. The most challenging and analytically sound criticism has questioned one of the central assumptions of the method (e.g., Goldberger 1978, 1979; Conley 2006). Specifically, in order to identify the model and estimate a value for heritability, one must assign a value for *gene-environment correlation*—which describes the degree to which the environments of identical twins are more similar than those of fraternal ones.[4] The standard approach to handling this in orthodox behavior genetics is to assign a value of zero to this parameter, which assumes the environments of identical twins are equally alike as those of fraternal twins. It has been argued that this most likely introduces upward bias into heritability estimates, resulting in an overstatement of the role of genetics (e.g., Conley 2006). Though difficult to test empirically, this critique seems a valid one as it is generally accepted from daily experience that identical twins are typically treated more similarly than are fraternal twins. However, even granting the legitimacy

[4]The same reasoning holds for other familial dyad comparisons.

of this critique, there are still compelling reasons for taking heritability seriously as an indicator of genetic influence.

The primary line of evidence offering limited support to heritability is found in recent research in which a wide confidence interval is given for heritability estimates based on a reasonable range of values for gene-environment correlation (e.g., Bowles and Gintis 2002). Thus, instead of assuming that gene-environment correlation equals zero, these researchers estimate heritability using a range of values of gene-environment correlation (e.g., 0–.8 in Bowles and Gintis 2002). This research has shown that for many traits related to status attainment heritability is large and significant even assuming very high levels of gene-environment correlation (e.g., Bowles and Gintis 2002; Björklund, Jantti, and Solon 2005). A second line of reasoning supporting the validity of heritability comes from studies showing that when identical twins are mislabeled as fraternal, their covariance on behavioral traits is the same as correctly coded identical twins (Guo 2006), indicating that either identical twins' environments are no more similar than fraternal ones or that if they are more similar that this does not bias heritability estimates. Based on these findings, we contend that though heritability estimates are probably inflated in most cases and thus should not be interpreted as precise estimates of genetic influence, they are nonetheless sufficient to demonstrate that genes are influential (Freese 2006). Thus, based on the very large volume of biometric research indicating substantial heritabilities for cognitive ability, various mental illnesses, and personality traits (see Plomin et al. [1997, 2003] for review), we conclude that genes are influential in these traits.

The second major obstacle in integrating sociological and genetic findings in the area of ability formation is the assumption in orthodox behavior genetics that shared environments do not matter for virtually any traits. Here, shared environment refers to anything that is constant within a household and thus, includes many standard social science variables such as parental income or education. But while this has long been an enshrined tenant of behavior genetics it has recently been challenged by both biometric and molecular findings within genetics. Iconoclastic studies using biometric methods can be traced back to work by Fischbein (1980) and Rowe, Jacobson, and van den Oord (1999) in which samples were stratified by occupational class and parental education, respectively, and biometric models fit for cognitive ability among each social group. In both cases, the influence of shared environment was close to zero for high SES groups, but was much greater for low SES individuals. Building on this work using more sophisticated biometric methods, several recent investigations have substantiated the finding that shared environment is important for cognitive ability among low SES individuals. For instance, using Add Health, the premier panel data for contemporary U.S. adolescents, Guo and Stearns (2002) found that the effect of shared environment on cognitive ability was larger than the effect of heritability for children with parents with less than high-school education, while the opposite was true for children with better educated parents. These findings have also been substantiated in a number of other recent, methodologically sophisticated studies (Turkheimer et al. 2003; Kremen et al. 2005; Harden, Turkheimer, and Loehlin 2007). Thus, recent genetics research has shown that social origins do matter and that this can be detected using biometric methods. These findings are also echoed in recent molecular genetics research modeling the interaction of specific genes and environments, where structural factors such as neighborhood-level (Manuck et al. 2005) and parental SES (Eley et al. 2004) have been shown to interact with specific genotypes in influencing cognitive outcomes.

In discussing the principal disagreements between behavior genetics and sociology, we have outlined the first step in our synthetic model. Specifically, by demonstrating (1) that heritability estimation, while being a very limited tool, is sufficient to demonstrate genetic influence (see Freese [2006] for a more detailed argument) and (2) that a convergence of new biometric and molecular genetic studies have debunked the idea that social backgrounds are irrelevant to ability formation, we have presented evidence that both social and genetic factors are important in ability formation. This brings us to the very important question of *how* these factors interact. Answering this question is currently the topic of vigorous inquiry and according to some prominent figures in sociology will be a major task for sociologists into the foreseeable future (Conley 2006). Although there are still many aspects of this process that are poorly understood, some progress has been made. For instance, regarding cognitive ability, the research to date has indicated greater heritability for individuals from higher SES backgrounds, implying that individuals from less advantaged backgrounds are less likely to achieve their genetic potential (Guo and Stearns 2002; Turkheimer et al. 2003). This finding has also been replicated in a sample of middle and high SES individuals (Harden et al. 2007), showing that genetic influences are stronger among those from a high SES background. Thus, the available evidence suggests that genetic influence monotonically increases as SES increases for cognitive ability.

However, the evidence on mental health suggests a different mechanism. Work in molecular genetics has shown that in cases where there is evidence of gene-social environment interaction, genetic susceptibility is generally triggered by adverse environmental cues. For instance, in their seminal work demonstrating molecular evidence of gene-environment interaction, Caspi et al. (2003) have shown that when individuals with genetic variants predisposing less efficacious neurotransmission[5] experience stressful life events, they are more likely to react with depression than those with more efficacious genetic variants. Other studies have found similar results (e.g., Caspi 2002; Gillespie et al. 2005; Guo, Wilhelmsen, and Hamilton 2007) and on the whole the results suggest that adverse social environments tend to trigger genetic vulnerabilities to mental illness and favorable environments tend to buffer against these vulnerabilities. These results imply that mental illness heritability should be higher in adverse environments as everyone, including those with genetic susceptibility, tends to be buffered by favorable environments.

Cumulatively, the discussion above suggests a complex scenario indicating fairly consistent additive, direct effects for both genes and social environments and also a considerable degree of gene-environment interaction for key ability traits (Guo and Stearns 2002; Turkheimer et al. 2003; Harden et al. 2007). Furthermore, there appears to be multiple gene-environment interactive mechanisms at work (Shanahan and Hofer 2005), and these mechanisms appear to differ across ability traits. Thus, by integrating contemporary research on the direct and interactive effects of social and genetic factors, we arrive at a theoretical model with substantial utility to empirical research. For example, this model suggests that complex pathologies, both mental and physical, are apt to be more heritable in adverse environments, with favorable environments buffering against the manifestation of these traits; while normally distributed, quantitative traits such as cognitive ability and height are apt to be more heritable in favorable environments, with adverse environments limiting the actualization of genetic potential for such traits. However, though such integration

[5]Specifically, the short allele of 5-HTTLPR, which is associated with lower serotonin promoter transcriptional efficiency.

is valuable for guiding future empirical research, it is still static in the sense that all of the studies cited above are from essentially the same historical, structural, and cultural context. No research to date has employed a dynamic perspective examining how genetic and social influences on ability formation systematically differ across societies in response to variation in structural context.

STRUCTURAL INFLUENCES ON THE FORMATION AND CONSEQUENCES OF ABILITY

To model how structural context moderates the influences of genetic and social factors on status attainment, it is helpful to distill the discussion above, identifying and elaborating the three paths through which genetic and social factors affect status outcomes—namely, direct genetic effects, direct social effects, and interactive effects between these two factors. Direct, additive genetic effects encompass genetic factors that are not very contingent on the environment; simple examples of these are things like eye color or Mendelian diseases such as cystic fibrosis (Riordan et al. 1989). However, complex ability phenotypes also generally have some degree of additive genetic effects. For instance, the apoE polymorphism plays a key role in the metabolism of cholesterol and triglyceride and, consequently, those possessing the susceptibility apoE allele are at heightened risk of coronary heart disease, regardless of environmental risk (Song, Stampfer, and Liu 2004). Most geneticists agree that even if much genetic expression is environmentally contingent, there is still likely to be some degree of direct, main effects for complex phenotypes (Risch 2000; Plomin, Owen, and McGuffin 1994). The second major path consists of direct social effects. This is a topic well studied by sociologists and refers to factors such as schooling—things that enhance ability and/or status, regardless of genetic factors (Jencks and Phillips 1996; Winship and Korenman 1997). Finally, gene-social environment interactions have been discussed above—these are genetic predispositions that are contingent on social environmental conditions and examples include the interaction effects of genes involved in neurotransmission with various forms of environmental adversity in promoting psychopathology (e.g., Caspi et al. 2002, 2003).

Having developed a working model of the three broad factors that influence status attainment, the question remains: How are the effects of these factors structurally moderated? We see structural moderation of the direct and interactive influences of genetic and social factors in two key places: first, in the development of phenotypic ability (the distal component), and second, in the translation of ability into status (the proximate component). That is, the *convertibility* of genome and social background into ability and the *convertibility* of ability into status are themselves variables that are subject to the influence of particular societal contexts. But what sorts of social structures might matter? Here we emphasize two social factors that empirical work has shown to be influential in moderating the status attainment process: inequality and social closure/mobility. Thus, we present several lines of converging evidence that inequality moderates the relative influences of social background and the genome on ability formation, while also showing that both inequality and mobility influence the extent to which ability is translated into status.

Inequality and the Formation of Ability

The research discussed above indicates that the genome and social background have direct and interactive influence on the formation of ability. Thus, it is suggested that while the genome predisposes individuals to various developmental paths and vulnerabilities, the expression of these genetic potentialities is generally contingent

on the social circumstances of the individual. From this logic, it follows that when the social circumstances of individuals are widely divergent (i.e., inequality[6] is great), the relative importance of the genome on ability formation will be diminished and that of social background will be increased. While we find support for this claim in the several deductive, theoretical, and empirical sources discussed below, the most fundamental evidence of inequality's moderation comes from the following basic tenet of quantitative genetic theory: "There is no one unique heritability... Heritability can differ among environments because... as environmental variance (V_E) increases, heritability decreases, because a smaller proportion of phenotypic variance is additive genetic" (Conner and Hartl 2004:113–14).[7] Thus, if there is no variation between environments, then no phenotype variance will be due to the environment, it will all be due to genes and chance. From this it follows that as environments become more diverse, a smaller proportion of phenotypic variance is explained by additive, genetic factors.[8] Thus, as variation in social backgrounds decreases, the contribution of social background to ability variation decreases, while the genetic contribution remains—resulting in ability becoming more a function of genetics and less a function of social background.

This logic is intuitively illustrated in the example of skin color—in a slightly idealized scenario skin color can be seen as determined by two major factors: genetics and exposure to the sun. If everyone has the same exposure to the sun (no inequality), then all variation in skin color is genetic, but if there is large variation in sun exposure (high inequality) then skin color is more a function of the environment. Next, to more fully illustrate our logic, let us introduce a third source of variation in skin color—GxE. Thus, skin color can be seen as determined by three factors: (1) genetic heritage, or your skin color at birth (additive genetic effects); (2) exposure to the sun (additive environmental effects); and (3) genetic differences in rates of tanning (GxE effects). So in this scenario, some people are born darker than others due to genetic differences, some people become darker due to exposure to sun, and some people become darker faster because they are genetically disposed to be "quick tanners." So we have the three basic sources of phenotype variance that are discussed above—additive genetic, additive environmental, and GxE.[9]

To demonstrate the effect of inequality, consider three scenarios: (1) exposure to the sun varies widely (high inequality, medium mean); (2) exposure to the sun varies extremely little and everyone has virtually no exposure to the sun (low inequality, low mean); and (3) exposure to the sun varies extremely little, everyone has virtually the same, high level of exposure to the sun (low inequality, high mean). In scenario 1, the additive environmental effects are going to be large because there is a lot of variation in sun exposure and all those under heavy sun become somewhat darker, while those in no sun get no darker regardless of genetics. GxE will be present with quick tanners tanning relatively fast in sunny environments, but both slow tanners and quick tanners tanning little in overcast environments; additive genetic effects will also be present but somewhat overshadowed by the other two types of effects. Thus,

[6]By inequality we refer to the variance of the distribution of resources in the society.

[7]These expression—V_E and V_P—refer to the equation defining heritability: $h^2 = V_G/V_P$ with P (phenotypic) = G (genetic) + E (environmental) + ε (residual).

[8]Of course, depending on the plasticity of the phenotype there may be thresholds or other nonlinearities in this relationship, but the overall trend remains: as environmental variation increases, the role of the genetic effect decreases.

[9]The only difference from the ability trait situation is we are not looking at social environment, but instead we are considering exposure to the sun as our environmental factor; however, after establishing our logic here we will address this discrepancy in the next example.

in this high inequality scenario genetic effects on the whole will be relatively small, as much of the variation in skin color will come from differences in sun exposure.

In scenario 2, however, the additive environmental effects will be very small because everyone has the same exposure to the sun; GxE effects will also be very small because no one has much exposure to sun, making quick tanning genes irrelevant; and additive genetic factors will be present. Thus, in this low inequality, low mean scenario, virtually all of the skin color variance will be due to genetic factors and stochasticity. In scenario 3, again, additive environmental effects will be very small because everyone has the same exposure to the sun; GxE effects will, in a sense, be large because everyone is highly exposed to the sun and thus, quick tanners will darken faster than slow tanners (however, this GxE effect will be subsumed into the additive genetic effects because there is no environmental variation[10]); finally, additive genetic effects will be present. Thus, in the low inequality, high mean scenario, virtually all skin color variance is due to genetic factors and stochasticity. Obviously, skin color is not an ability trait and sun exposure is not resource inequality, but having established the general logic with this noncontroversial example we now demonstrate that this logic holds for ability traits by considering the example of cognitive ability.

Earlier in our examination of work by Guo and Stearns (2002) and others, we found that regarding cognitive ability in contemporary America, genes seem more influential than social background among higher SES groups, while social background seems to be more important to lower SES groups. This is a close analogy to the skin color example above—some people are born with the propensity to become "smarter" than others due to genetic differences, some people become smarter due to exposure to favorable social environments, and some people become smarter faster when in favorable environments because they are genetically disposed to be "quick learners." So again, we can apply our three scenarios to illustrate the effects of inequality on ability traits: (1) high inequality in social background, medium mean; (2) very low social background inequality and low mean; and (3) very low social background inequality and high mean.

Here again, we see that in scenario 1 the aggregate effects of genetic factors of cognitive ability are relatively low compared to the other two scenarios. This is because in scenario 1 the additive environmental effects are going to be relatively large because everyone who is in a high-quality social environment will reap some measure of cognitive benefits, while those in low-quality social environments will benefit less, regardless of genetics; GxE will be present with those with quick learning genes making quicker cognitive gains in high-quality social environments, but both slow learners and quick learners gaining relatively slower in low-quality social environments; additive genetic effects will also be present, but somewhat overshadowed by the other two types of effects. Thus, in this high inequality scenario, genetic effects on the whole will be relatively small, as much of the variation in cognitive ability will come from differences in social background.

In scenario 2, however, the additive environmental effects will be very small because everyone has the same social background; GxE effects will be very small also because no one has a high-quality social background, thus, the quick learning genes will be irrelevant; and additive genetic factors will be present. Thus, in this very low inequality, low mean scenario virtually all of the cognitive ability variance will be

[10]This can be well conceptualized as an interaction variable in which all cases have a value of 1 on the first original variable (environment)—thus, the values of this interaction variable will be equivalent to the second original variable (genetics).

due to genetic factors and stochasticity. In scenario 3, again, additive environmental effects are very small because everyone has the same social background; GxE effects will, in a sense, be large because everyone is in a high-quality social environment and thus, quick learners will gain faster than slow learners; however, this GxE effect will be subsumed into the additive genetic effects because there is no environmental variation; finally, true additive genetic effects will be present. Thus, in the very low inequality, high mean scenario virtually all cognitive ability variance is due to genetic factors and stochasticity.

Perhaps the simplest way to conceptualize this logic is to consider heritability a societal-level variable predicted by inequality—as inequality increases the heritability of ability traits decreases. More precisely, we propose that inequality operates by moderating the proportion of phenotypic variance comprised by social background factors—in high inequality societies a greater proportion of the phenotypic variance will be due to social background, even if the amount of genetic variance remains the same. While we have thus far supported this proposition largely with quantitative genetic theory and deductive logic, it is also supported by empirical research from both quantitative genetics and historical demography.

The effect of inequality on heritability is well documented in quantitative genetics literature (e.g., Falconer and Mackay 1996; Conner and Hartl 2004), but has received little attention by behavior geneticists employing similar methods. Quantitative geneticists' interest in the effect of environmental variation on heritability is due to the fact that while much quantitative genetic inquiry aims to describe evolutionary processes in the natural environment, the actual empirical research generally takes place in laboratories where environmental variation is minimized. Thus, there is a methodological issue of how well laboratory estimates of heritability generalize to natural environments characterized by far greater environmental heterogeneity. In order to address this issue, there has been considerable research examining differences in heritabilities between laboratory and field conditions, showing that the reduced environmental variation of the lab leads to greater heritability (e.g., Falconer 1981; Coyne and Beecham 1987; Riska, Prout, and Turelli 1989; Prout and Barker 1989). To give just a couple of examples from this substantial literature, Orengo and Prevosti (1999) showed wing size heritability of *Drosophila subobscura* reared in a laboratory was about six times larger than those in nature. Similarly, Prout and Barker (1989) found that for another species of *Drosophila*, thorax length heritability was about six times larger for those reared in the lab versus nature. In this research environmental variation is generally conceptualized as differences in access to food, water, and also factors such as crowding and climatic conditions. In human populations, a single construct—resource inequality—captures much of this environmental variation, serving as a proxy for differential access to food, potable water, medical care, leisure, and emotional security, as well as exposure to violence, stress, and environmental toxins.

A second empirical line of evidence comes from historical demographic research on height, which most historical demographers contend is the best proxy for health for which substantial historical data exist (Floud, Wachter, and Gregory 1990). Using historical military and philanthropic data from the United Kingdom, Floud et al. (1990) found that class differences in height lessened considerably from 1750 to 1980 due to better nutrition, sanitation, and other environmental improvements for the working class. Thus, as social environments became more equal, the amount of variance in height accounted for by social background factors declined, and the proportion of height variance accounted for by genes increased; in other words, height became more a function of the genome and less of social background.

In sum, we find a confluence of evidence from quantitative genetics theory and research and historical demography supporting the proposition that societal-level resource inequality moderates the relative influence of social and genetic factors on ability formation.

Social Closure, Inequality, and the Ability-Status Link

Regarding the proximate link between ability and status, we argue that both high social closure and high inequality limit the strength of the association between ability and status. The basic logic here is illustrated in the following example: an intelligent, ambitious, hard-working, and energetic individual will be more likely to attain high status in a society characterized by openness and low inequality such as contemporary Denmark, than as an untouchable in the Indian caste system or a serf in medieval Russia. It is important to note here that our argument is relative; we are not arguing that social background is irrelevant to the life chances of Danish (or any other advanced industrial society's) citizens, but rather that the influence of ability is stronger here relative to more closed societies. The basic premises here are straightforward and empirically validated: (1) social closure on the basis of social background prevents individuals from moving to a place in the stratification system commensurate with their level of ability and (2) inequality in key resources such as wealth, political decision making, and social capital creates inherited status inertia based on social background, regardless of ability.

Though the basic idea of social closure has been discussed throughout history, the formal delineation of the concept has its roots in the work of Weber (1968) and has been further theorized by Parkin (1974, 1979). Parkin (1974), following Weber (1968), defined social closure as: "the process by which social collectivities seek to maximize rewards by restricting access to resources and opportunities to a limited circle of eligibles. This entails the singling out of certain social or physical attributes as the justificatory basis of exclusion." In discussing the various ascriptive mechanisms attenuating the influence of ability in status attainment, we begin with more blatant, legal distinctions (e.g., gender and racial distinctions, slavery, aristocracy, and caste) before examining more covert mechanisms (e.g., covert racism, sexism, and classism).

The role of legal inequalities in attenuating the ability→status relationship is sufficiently obvious to warrant only a very brief discussion. Simply stated, when rights and privileges vary according to ascribed criteria, status will be systematically denied to some (as in the case of slavery) and virtually ensured for others (as in the case of aristocracy), regardless of ability. Under conditions of ascribed legal inequality, status assortment on the basis of ability cannot take place because the tiers of the stratification system are highly impermeable. Thus, the role of ability (and, consequently, of the genome) in status attainment is diminished in such closed societies.

Extreme examples of the effects of social closure on the composition of social strata can be found in the case of consanguineous marriages within European, Mayan, Egyptian, and Hawaiian aristocracies (Palmer 1997; Van Den Berghe and Mesher 1980; Bixler 1982; Christensen 1998), where inbreeding lessened the ability of heirs, yet structural rigidity held these individuals in privileged positions far above those warranted by their abilities. This phenomenon is well illustrated by the inbreeding among the Spanish and Austrian Habsburg lineages (Palmer 1997; Hindley 2000), which led to genetic defects including mental retardation, epilepsy, hydrocephaly, and the infamous "Habsburg Lip"—a jutting underbite, so pronounced in Emperor Ferdinand I that he was purported not to be able to eat or speak properly (Palmer

1997). In a telling example of social closure lessening the role of ability in status assortment, Ferdinand—buttressed by structural privileges—sat at the throne of the Austrian Empire for 13 years despite suffering from multiple debilitating defects, including what appears to be a form of mild mental retardation (Hindley 2000).

While legal distinctions based on ascribed characteristics (e.g., slavery, aristocracy, and caste) are the most obvious examples of social closure interrupting status assortment on the basis of ability, their presence has declined sharply in modern societies characterized by movements toward universal legal equality. However, though closure has loosened and the stratification system has become more permeable, contemporary Western societies are far from free of social closure. Rather, less obvious mechanisms of covert discrimination based on ascribed characteristics such as race, gender, and social origins/class predominate in these societies. Some of the strongest evidence of the continuing presence of gender and race discrimination comes from field experiments in which matched pairs of confederates apply in the same markets (e.g., for jobs or housing). Such experiments show consistent evidence of persistent race and gender discrimination within the United States (e.g., race: Bendick Jackson, and Reinoso 1994; Cross, Kenney, Mell, and Zimmermann 1990; gender: Neumark, Bank, and Van Nort 1996; Nunes and Seligman 2000) and Europe (e.g., race: Smeesters and Nayer 1998; Goldberg, Mourinho, and Kulke 1996; de Prada, Actis, Pereda, and Perez Molina 1996; gender: Weichselbaumer 2000). Thus, in advanced industrial societies legal social closure is extremely rare, but covert mechanisms, while declining in prevalence,[11] continue to influence the attainment process, attenuating the influence of ability, and thus reducing genomic influence on status.

The final consideration concerns the additional moderating effects of inequality net of its influence on ability. The logic here is intuitive: as inequality increases, it becomes more difficult for those at the bottom of the stratification system to ascend and easier for those at the top to maintain their status. Increasing inequality promotes this status inertia by providing differential access to key resources (e.g., educational opportunities, social, cultural, political, and financial capital) based on social background, not ability. This effect has been empirically indicated by comparative mobility studies showing the relationship of family background to status to be weaker in highly egalitarian societies such as the Scandinavian nations (Björklund, Eriksson, Jäntti, Raaum, and Österbacka 2002; Sieben and de Graaf 2001; Treiman and Yip 1989). Building on such empirical findings, leading mobility experts have theorized a systematic negative effect of inequality on social mobility (Erickson and Goldthorpe 1992, 2002). So while a direct test of inequality's moderation of the ability→status relationship has not yet been conducted,[12] comparative mobility research offers strong support for this hypothesized influence.[13]

[11]Though the evidence is clear that such discrimination persists, equally convincing is the evidence that its incidence has steadily declined over the past 40 years (e.g., Heckman 1998; Jarrell and Stanley 2004).

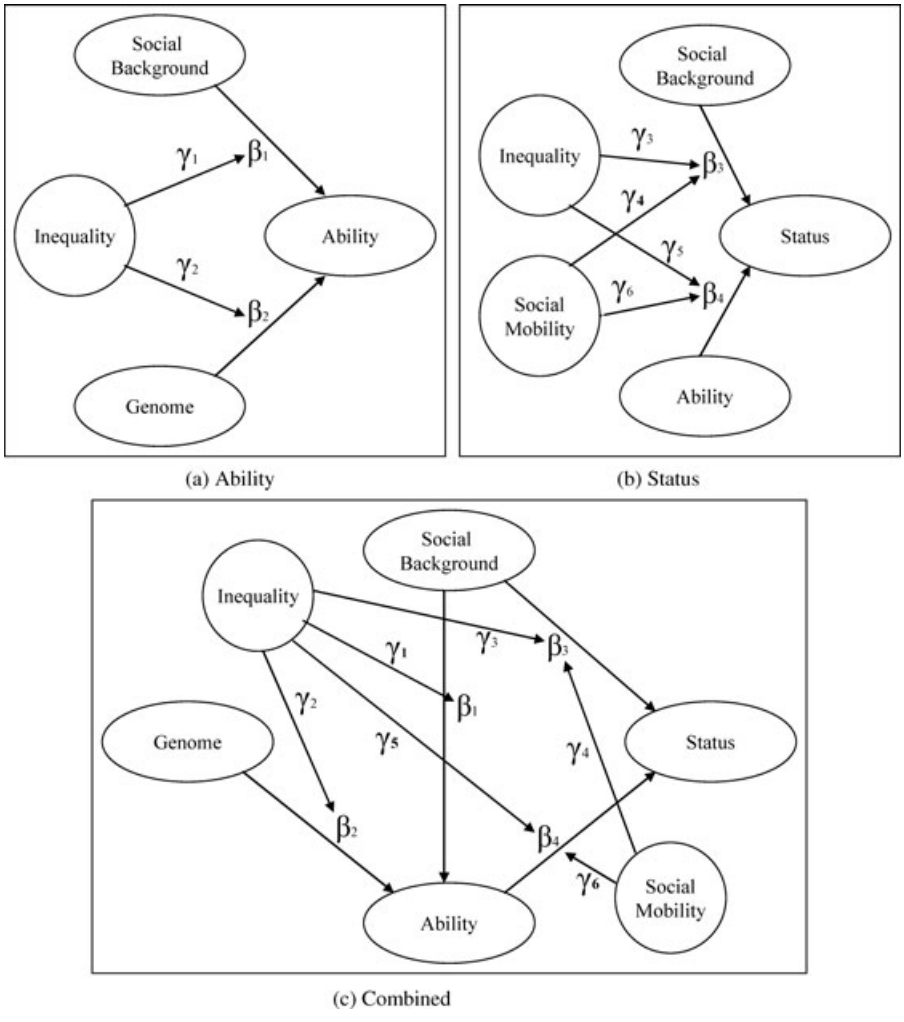
[12]The ideal test of inequality's moderation of the ability→status relationship would be a multilevel model with the effects of ability and social background on status modeled as random coefficients predicted by societal-level inequality. Unfortunately, this study has not been conducted, probably due to the lack of cross-national data with data collection and measurement standardized (Breen and Jonsson 2005).

[13]There are a number of other points one could argue here about the probable relationships between social institutions and gene-environment dynamics. We have drawn on the existing literature to outline what we see as the clearest and most probable associations between social structures and genetic expression. The larger point here, however, is that given their knowledge about the institutions that produce both mobility and inequality—and the relations between them—sociologists are probably the best positioned of all to generate hypotheses about the complex relationship between inequality, social mobility, and the actualization of genetic potential.

A COMBINED MODEL OF STATUS ATTAINMENT

Specifying the Model

In the foregoing sections, we have done three things: first, we have theorized what traits are most useful in attaining status; second, we have integrated genetic and sociological accounts of the status attainment process, reconciling these perspectives in the context of contemporary American society; third, we have presented evidence that inequality and mobility are social factors that condition both the formation and utility of ability. With these points in place, we now advance our combined model of status attainment, which integrates the research we have discussed into the classic status attainment model. This model is shown graphically in Figure 1 and as equations in Table 1.



Note: Circular and elliptical variables represent societal- and individual-level characteristics, respectively.

Figure 1. General status attainment model expressed as path diagrams.

Table 1. General Status Attainment Model Expressed as a System of Equations

Figure	Title	Equation	Level
1.a.	Ability	$A = \beta_1 B + \beta_2 G + \varepsilon_1$	1
1.a.	Ability	$\beta_1 = \gamma_1 I + \upsilon_1$	2
1.a.	Ability	$\beta_2 = \gamma_2 I + \upsilon_2$	2
1.b.	Status	$St = \beta_3 B + \beta_4 A + \varepsilon_2$	1
1.b.	Status	$\beta_3 = \gamma_3 I + \gamma_4 M + \upsilon_3$	2
1.b.	Status	$\beta_4 = \gamma_5 I + \gamma_6 M + \upsilon_4$	2
1.c.	Combined	$St = (\gamma_3 I + \gamma_4 M + \upsilon_3)B + ([\gamma_5 I + \gamma_6 M + \upsilon_4] \times [(\gamma_1 I + \upsilon_1)B + (\gamma_2 I + \upsilon_2)G + \varepsilon_1]) + \varepsilon_2$	1 & 2

Variable definitions: Level 1 (individual): B = social background; G = genome; A = ability; St = status; Level 2 (societal): I = inequality; M = social mobility.

This model formalizes each of the relationships we have proposed, fusing conventional status attainment and behavior genetic models, and theorizing primary structural moderators of these individual-level relationships. Specifically, Equation 4 represents the classic, individual-level status attainment model, modeling status as a function of social background, ability, and chance. Similarly, Equation 1 essentially summarizes the basic behavior genetic model, suggesting that ability phenotypes are a function of genome, social background, and other miscellaneous factors. Equations 5 and 6 describe structural moderation of the effects of social background and ability on status, which, though implied by former research, to our knowledge has not previously been formally theorized. Finally, Equations 2 and 3 represent perhaps our most novel proposition—that the relative weights of genome and social background are influenced by societal levels of resource inequality. Cumulatively, this model enhances understanding of the status attainment process in several ways. First, through including an account of the formation of ability, the model allows an assessment of the role of genetics in status attainment, while also facilitating the appraisal of the indirect effects of social background via ability. Second, the model generates a testable theory positing specific structural factors as primary moderators of not only the relative influences of the proximate determinants of status attainment, but also, more fundamentally, of the actualization of genetic potential.

IMPLICATIONS OF THE MODEL FOR EMPIRICAL RESEARCH

We do not propose this model purely as a theoretical exercise; on the contrary, it has straightforward and important implications for the study of stratification and stratification-related phenotypes by both geneticists and sociologists. Here we outline three of these implications, first examining the benefits of integrating current work on social and genetic ontogeny in a single societal context, then discussing the implications of a comparative view of structural moderation of the genetic-social environment nexus, before finally discussing methodological implications.

The first contribution of the model is predictive. Though genetic predispositions and social location are both effective predictors of status attainment, a model that uses them either singly or additively achieves nothing approaching perfect prediction. This is in part because social location and genetic predispositions are inherently

interactive—the influence of each is dependent on the value of the other. (Moreover, both influences depend on macro-level contextual variation, which is rarely employed in status attainment research.) The contingent interplay of genetic and social structural effects has been demonstrated in recent work by Shanahan et al. (2007). This work, which uses genetic and social data from the National Longitudinal Study of Adolescent Health, shows that the link between a dopamine receptor polymorphism (DRD2) and continuation on to higher education is highly conditional upon one's social location. These studies show that particular configurations of social capital completely compensate for genetic differences; specifically, among individuals who are in situations of high parental involvement and high-school quality, or among those who have a mentoring relationship with a teacher, there are no differences in school continuation rate by DRD2 status.[14] Results like these confirm the utility of looking at gene-environment interactions. In the absence of interactive models, geneticists are simply estimating *average* genetic effects across socially different groups and sociologists are estimating *average* social background effects across genetically variable populations. Allowing these effects to vary systematically will increase predictive power and allow for a more nuanced understanding of the effects of both social background and genetic predispositions.

The second contribution is explanatory. Researchers have come to acknowledge the role that ability phenotypes play in status attainment, but they still implicitly disagree on the origins of ability. While, as noted above, there is a small but growing body of research that aims to resolve this debate by empirically estimating the direct and interactive contributions of genetics and social environment to ability traits in contemporary society, no research to date has explored the possibility that these contributions may systematically vary across societies. Here we have presented four sources of evidence that indicate societal-level resource inequality influences the relative importance of genetics versus social background. This evidence all points to high inequality increasing the relative influence of social background, and thus, decreasing the role of genetics in ability formation. Thus, the model suggests that in very unequal societies, individuals from disadvantaged social backgrounds suffer not only increased material disadvantage, but also increased disadvantage in actualizing their genetic potential. This realization is crucial since it injects perhaps the most important sociological insight—social structure matters—into the heart of genetics. This has implications for the interpretation of both genetic and behavioral research, in that both types of findings should be considered contingent on macro social structure.

The third implication is methodological. One of the things this model makes clear is that our understanding of the dynamics of status attainment will be greatly advanced through cross-national comparative research that is genetically informed. Collecting data with similar instruments across a wide variety of social contexts and countries would allow us to move toward more systematic knowledge on the central question of how social structure moderates the expression of genetic potential. The model also has implications for data analyses. The fundamentally interactive nature of genetics and social structure makes additive statistical modeling a poor approach to elucidating this process. One can, as we have done here, specify models that model coefficients as a function of the other covariates in order to capture

[14]More research is needed, however, since—in this particular case, for example—we do not know if the diminution of DRD2's predictive power is due to changes in the genome→ability phenotype link or the ability phenotype→status phenotype link.

this complexity.[15] But other techniques—like combinatorial analyses—can also shed light on the complex relation between genotype, social structure, and phenotype. Using such combinatorial techniques, Shanahan and his colleagues (2007) have shown that environment-environment interactions are also operative in conditioning genetic effects. Researchers who analyze social and genetic data—whatever statistical techniques they employ—should be aware of the probability of these complex, interactive relationships both within a given societal context and comparatively.

CONCLUSION

Sociologists and geneticists are each heavily invested in understanding the formation and status-relevant consequences of ability phenotypes like cognitive ability, personality, and mental and physical health. Unfortunately, each discipline has largely been working independently of the other and we now have a variety of insights into these processes that are not easily combined. Though both disciplines now have some sense that they need each other, institutionalized wariness and, perhaps more importantly, the absence of a common theoretical framework, have prevented integration. In this article, we have reviewed both literatures and have advanced a combined model of status attainment that should allow sociologists and geneticists to better understand and communicate the complex relationships we now know exist between genotype, phenotype, and the social environment. For geneticists, the advantage of this model is that it relies on decades of sociological research that suggest two key analytic sites for exploring genetic moderation—inequality and mobility—and provides a wealth of insight for exploring the mechanisms by which these social forces affect genetic action. For sociologists, the advantage of this model is that it provides an intellectual framework for understanding the thoroughly social nature of the genotype→phenotype link; put differently, the model shows that the "translation" of genetics into characteristics relevant to a variety of sociological outcomes is conditional on precisely the kinds of social structures in which sociologists are interested. Society is not something that plays around at the edges of genetic expression—it is vitally implicated in the actualization and subsequent consequences of genetic potentialities. It is our hope that this model, and the empirical research that it may inform, will help us move past "nature," "nurture," and the even more unhelpful "both," toward an understanding of precisely *how* genetics and the social environment work together to produce the stratified social world.

REFERENCES

Barrick, M. R., M. K. Mount, and T. A. Judge. 2001. "Personality and Performance at the Beginning of the New Millennium: What Do We Know and Where Do We Go Next?" *International Journal of Selection and Assessment* 9:9.

Bendick, M., C. W. Jackson, and V. A. Reinoso. 1994. "Measuring Employment Discrimination Through Controlled Experiments." *Review of Black Political Economy* 23(1):25–48.

Bixler, R. H. 1982. "Sibling Incest in the Royal Families of Egypt, Peru, and Hawaii." *Journal of Sex Research* 18(3):264–81.

Björklund A., T. Eriksson, M. Jäntti, O. Raaum, and E. Österbacka. 2002. "Brother Correlations in Earnings in Denmark, Finland, Norway and Sweden Compared to the United States." *Journal of Population Economics* 15:757–72.

[15]For instance, the model presented in Figure 1, could be statistically specified as a multilevel mixed model (Raudenbush and Bryk 2002) or a structural equation model (Bollen 1989). See Guo and Adkins (2008) for a primer of approaches for including genetic information in social science research.

Björklund, A., M. Jantti, and G. Solon. 2005. "Influences of Nature and Nurture on Earnings Variation: A Report on a Study of Various Sibling Types in Sweden." Pp. 145–64 in *Unequal Chances: Family Background and Economic Success*, edited by S. Bowles, H. Gintis, and M. Osborne Groves.. New York: Russell Sage Foundation and Princeton University Press.

Blau, P. and O. Duncan. 1967. *The American Occupational Structure*. New York: Wiley and Sons.

Bollen, K. A. 1989. *Structural Equations with Latent Variables*. New York: John Wiley & Sons.

Bourdieu, P. and J.-C. Passeron. 1977. *Reproduction in Education, Society, and Culture*. London: Sage Publications.

Bowles, S. and H. Gintis. 2002. "The Inheritance of Inequality." *Journal of Economic Perspectives* 16(3):3–30.

Bowles S., H. Gintis, and M. Osborne. 2001a. "The Determinants of Earnings: A Behavioral Approach." *Journal of Economic Literature* 39:1137–76.

———. 2001b. "Incentive-Enhancing Preferences: Personality, Behavior, and Earnings." *American Economic Review* 91:155–58.

Bowles S., H. Gintis, and M. G. Osborne Groves (Eds.). 2005. *Unequal Chances: Family Background and Economic Success*. New York: Russell Sage Foundation and Princeton University Press.

Breen, R. and J. O. Jonsson. 2005. "Inequality of Opportunity in Comparative Perspective: Recent Research on Educational Attainment and Social Mobility." *Annual Review of Sociology* 31:223–43.

Caspi, A. 2002. "Role of Genotype in the Cycle of Violence in Maltreated Children." *Science* 297:851–54.

Caspi, A. et al. 2003. "Influence of Life Stress on Depression: Moderation by a Polymorphism in the 5-HTT Gene." *Science* 301(5631):386–89.

Ceci, S. J. and W. M. Williams, 1997. "Schooling, Intelligence, and Income." *American Psychologist* 52:1051–58.

Chirikos, T. N. and G. Nestel. 1985. "Further Evidence on the Economic Effects of Poor Health." *Review of Economics and Statistics* 67(1):61–69.

Christensen, A. F. 1998. "Ethnohistorical Evidence for Inbreeding Among the Pre-Hispanic Mixtec Royal Caste." *Human Biology* 70(3):563–77.

Conley, D. 2006. "On Twin Studies." *Contexts* 5(1):7–8.

Conner, J. K. and D. L. Hartl. 2004. *A Primer of Ecological Genetics* Sunderland, MA: Sinauer Associates.

Coyne, J. A. and E. Beecham. 1987. "Heritability of Two Morphological Characters Within and Among Natural Populations of Drosophila Melanogaster." *Genetics* 117:727–37.

Cross, H., J. Kenney, J. Mell, and W. Zimmermann. 1990. *Employer Hiring Practices: Differential Treatment of Hispanic and Anglo Job Seekers*. Washington, DC: Urban Institute Press.

Devlin, B., M. Daniels, and K. Roeder. 1997. "The Heritability of IQ." *Nature* 388:468–71.

Dumais, S. A. 2002. "Cultural Capital, Gender, and School Success: The Role of Habitus." *Sociology of Education* 75(1):44–68.

Dunifon, R. and G. J. Duncan. 1998. "Long-Run Effects of Motivation on Labor Market Success." *Social Psychology Quarterly* 61:33–48.

Durkheim, E. 1984. *The Division of Labor in Society*. New York: Free Press.

Eley, T. C., K. Sugden, A. Corsico, A. Gregory, P. Sham, P. McGuffin, R. Plomin, and I. Craig. 2004. "Gene–Environment Interaction Analysis of Serotonin System Markers with Adolescent Depression." *Molecular Psychiatry* 9:908–15.

Erikson, R and J. H. Goldthorpe. 1992. *The Constant Flux: A Study of Class Mobility in Industrial Societies*. Oxford: Clarendon Press.

———. 2002. "Intergenerational Inequality: A Sociological Perspective." *Journal of Economic Perspectives* 16(3):31–44.

Falconer, D. S. 1981. *Introduction to Quantitative Genetics*, 2nd ed. New York: Longmans.

Falconer, D. S. and T. F. C. Mackay. 1996. *Introduction to Quantitative Genetics,* 4th ed. Burnt Mill, Harlow, Essex, UK: Longman.

Farkas, G. 2003. "Cognitive Skills and Noncognitive Traits and Behaviors in Stratification Processes." *Annual Review of Sociology* 29:541–62.

Fischbein, S. 1980. "IQ and Social Class." *Intelligence* 4:51–63.

Fischer, C. S., M. Hout, M. S. Jankowski, S. Luca, A. Swidler, and K. Voss. 1996. *Inequality by Design: Cracking the Bell Curve Myth*. Princeton, NJ: Princeton University Press.

Floud, R., K. Wachter, and A. Gregory. 1990. *Height, Health and History: Nutritional Status in the United Kingdom.* Cambridge: Cambridge University Press.

Freese, J. 2006. "The Analysis of Variance and the Social Complexities of Genetic Causation." *International Journal of Epidemiology* 35:534–36.

Freese, J., J. Li, and L. Wade. 2003. "The Potential Revelances of Biology to Social Inquiry." *Annual Review of Sociology* 29:233–56.

Gillespie, N. A. 2005. "The Relationship Between Stressful Life Events, the Serotonin Transporter (5-HTTLPR) Genotype and Major Depression." *Psychological Medicine* 35:101–11.
Goldberg, A., D. Mourinho, and U. Kulke. 1996. "Labour Market Discrimination Against Foreign Workers in Germany." *International Migration Papers 7*. Geneva: International Labour Office.
Goldberger, A. S. 1978. "Genetic Determination of Income—Comment." *American Economic Review* 68(5):960–69.
———. 1979. "Heritability." *Economica* 46:327–47.
Guo, G.. 2005. "Twin Studies: How Much Can They Tell Us About Nature and Nurture?" *Contexts* 4(3):78–83.
———. 2006. "On Twin Studies: Response." *Contexts* 5(1):8.
Guo, G. and D. Adkins. 2008. "How is a Statistical Link Established Between a Human Outcome and a Genetic Variant?" *Sociological Methods and Research* 37(2):201–26.
Guo, G. and E. Stearns. 2002. "The Social Influences on the Realization of Genetic Potential for Intellectual Development." *Social Forces* 80(3):881–910.
Guo, G., K. Wilhelmsen, and N. Hamilton. 2007. "Gene-Lifecourse Interaction for Alcohol Consumption in Adolescence and Young Adulthood: Five Monoamine Genes." *American Journal of Medical Genetics Part B, Neuropsychiatric Genetics* 144:417–23.
Haas, S. A. 2006. "Health Selection and the Process of Social Stratification: The Effect of Childhood Health on Socioeconomic Attainment." *Journal of Health and Social Behavior* 47:339–54.
Harden, K. P., E. Turkheimer, and J. C. Loehlin. 2007. "Genotype by Environment Interaction in Adolescents' Cognitive Aptitude." *Behavior Genetics* 37:273–83.
Hauser, R. M., J. R. Warren, M. Huang, and W. Carter. 2000. "Occupational Status, Education, and Social Mobility in the Meritocracy." Pp. 179–229 in *Meritocracy and Economic Inequality*, edited by K. Arrow, S. Bowles, and S. Durlauf. Princeton: Princeton University Press.
Heckman, J. 1998. "Detecting Discrimination." *Journal of Economic Perspectives* 12:101–16.
Heckman, J. and Y. Rubinstein, 2001. "The Benefits of Skill: The Importance of Noncognitive Skills: Lessons from the GED Testing Program." *American Economic Review* 91(2):145–54.
Herrnstein, R. and C. Murray. 1994. *The Bell Curve: Intelligence and Class Structure in American Life*. New York: Free Press.
Hindley, G. 2000. *The Royal Families of Europe*. New York: Carroll & Graf.
Jang, K. L., R. R. McCrae, A. Angleitner, R. Riemann, and W. J. Livesley. 1998. "Heritability of Facet-Level Traits in a Cross-Cultural Twin Sample: Support for a Hierarchical Model of Personality." *Journal of Personality and Social Psychology* 74:1556–65.
Jarrell, S. B. and T. D. Stanley. 2004. "Declining Bias and Gender Wage Discrimination? A Meta-Regression Analysis." *Journal of Human Resources* 39(3):828–38.
Jencks, C. 1972. *Inequality: A Reassessment of the Effect of Family and Schooling in America*. New York: Basic Books.
Jencks, C. and S. Bartlett. 1979. *Who Gets Ahead? The Determinants of Economic Success in America*. New York: Basic Books.
Jencks, C. and M. Phillips. 1998. *The Black-White Test Score Gap*. Washington, DC: Brookings Institution Press.
Jensen, A. R. 1998. *The G Factor: The Science of Mental Ability*. Westport, CT: Preager.
Koenig, M., E. P. Hoffman, C. J. Bertelson, A. P. Monaco, C. Feener, and L. M. Kunkel. 1987. "Complete Cloning of the Duchenne Muscular Dystrophy (DMD) cDNA and Preliminary Genomic Organization of the DMD Gene in Normal and Affected Individuals." *Cell* 50:509–17.
Kohn, M. L. and C. Schooler. 1983. *Work and Personality: An Inquiry into the Impact of Social Stratification*. Norwood, NJ: Ablex Publishing.
Kremen W. S., K. C. Jacobson, H. Xian, S. A. Eisen, B. Waterman, R. Toomey, et al. 2005. "Heritability of Word Recognition in Middle-Aged Men Varies as a Function of Parental Education." *Behavioral Genetics* 35(4):417–33.
Lareau, A. 2003. *Unequal Childhoods: Class, Race, and Family Life*. Berkeley, CA: University of California Press.
Lenski, G. [1966] 1984. *Power and Privilege*. Chapel Hill, NC: University of North Carolina Press.
Link, B. G. and J. Phelan. 1995. "Social Conditions as Fundamental Causes of Disease." *Journal of Health and Social Behavior* Spec No:80–94.
Lopez L. S., E. A. Croes, F. A. Sayed-Tabatabaei, S. Claes, C. Van Broeckhoven, and C. M. van Duijn. 2005. "The Dopamine D4 Receptor Gene 48-Base-Pair-Repeat Polymorphism and Mood Disorders: A Meta-Analysis." *Bio Psychiatry* 57(9):999–1003.
Mahley, R. W. and S. C. Rall Jr. 2000. "Apolipoprotein E: Far More than a Lipid Transport Protein." *Annual Review of Genomics and Human Genetics* 1:507–37.

Manuck, S. B., M. E. Bleil, K. L. Petersen, J. D. Flory, J. J. Mann, R. E. Ferrell, and M. F. Muldoon. 2005. "The Socio-Economic Status of Communities Predicts Variation in Brain Serotonergic Responsivity." *Psychological Medicine* 35:519–28.

Martin, J. L. 2003. "What is Field Theory?" *American Journal of Sociology* 109(1):1–49.

Marx, K. 1963. *Karl Marx: Early Writings*, edited by T. B. Bottomore. McGraw-Hill.

Miller, E. M. and C. Y. Costello. 2001. "The Limits of Biological Determinism." *American Sociological Review* 66:592–98.

Moffitt, T. E., A. Caspi, and M. Rutter. 2005. "Strategy for Investigating Interactions Between Measured Genes and Measured Environments." *Archives of General Psychiatry* 62:473–81.

Morgan, S. L., D. B. Grusky, and G. S. Fields. 2006. *Mobility and Inequality: Frontiers of Research in Sociology and Economics*. Stanford, CA: Stanford University Press.

Neumark, D., R. Bank, and K. Van Nort. 1996. "Sex Discrimination in Restaurant Hiring: An Audit Study." *Quarterly Journal of Economics* 111:915–41.

Nunes, A. and B. Seligman. 2000. *A Study of the Treatment of Female and Male Applicants by San Francisco Bay Area Auto Service Shops, The Testing Project of the Impact Fund*. San Francisco: Impact Fund.

Orengo, D. J. and A. Prevosti. 1999. "Wing-Size Heritability in a Natural Population of Drosophila Subobscura." *Heredity* 82 (Pt 1):100–06.

Osborne Groves, M. 2005. "Personality and the Intergenerational Transmission of Economic Status." Pp. 208–31 in *Unequal Chances: Family Background and Economic Success*, edited by S. Bowles, H. Gintis, and M. Osborne Groves.. New York: Russell Sage Foundation and Princeton University Press.

Palmer, A. 1997. *Twilight of the Habsburgs: The Life and Times of Emperor Francis Joseph.* New York: Grove/Atlantic.

Parkin, F. 1974. *The Social Analysis of Class Structure*. London: Tavistock.

———. 1979. *Marxism and Class Theory: A Bourgeois Critique*. Taylor & Francis Books.

Pearlin, L. I. 1989. "The Sociological Study of Stress." *Journal of Health and Social Behavior* 30:241–56.

Plomin, R., J. C. Defries, I. W. Craig, and P. McGuffin. 2003. *Behavioral Genetics in the Postgenomic Era.* Washington, DC: American Psychological Association.

Plomin, R., J. C. DeFries, G. McClearn, and M. Rutter. 1997. *Behavioral Genetics*. New York: Freeman.

Plomin, R., M. J. Owen, and P. McGuffin. 1994. "The Genetic Basis of Complex Human Behaviors." *Science* 264:1733–39.

Prada, M. de, W. Actis, C. Pereda, and R. Perez Molina. 1996. "Labour Market Discrimination Against Migrant Workers in Spain." *International Migration Papers 9*. Geneva: International Labour Office.

Prout, T. and J. S. F. Barker. 1989. "Ecological Aspects of the Heritability of Body Size in Drosophila-Buzzatii." *Genetics* 123(4):803–13.

Raudenbush S. W. and A. S. Bryk. 2002. *Hierarchical Linear Models: Applications and Data Analysis Methods*. Thousand Oaks, CA: Sage.

Riordan, J. R., J. M. Rommens, B.-sheva Kerem, N. Alon, R. Rozmahel, Z. Grzelczak, J. Zielenski, S. Lok, N Plavsic, J.-L. Chou, M. L. Drumm, M. Iannuzzi, F. S. Collins, and L.-C. Tsui. 1989. "Identification of the Cystic Fibrosis Gene: Cloning and Characterization of Complementary DNA." *Science* 245:1066.

Risch, N. J. 2000. "Searching for Genetic Determinants in the New Millennium." *Nature* 405:847–56.

Riska, B., T. Prout, and M. Turelli. 1989. "Laboratory Estimates of Heritabilities and Genetic Correlations in Nature." *Genetics* 123:865–71.

Roberts, B. W. and T. Bogg. 2004. "A Longitudinal Study of the Relationships Between Conscientiousness and the Social-Environmental Factors and Substance-Use Behaviors that Influence Health." *Journal of Personality* 72:325–53.

Robins, R. W., A. Caspi, and T. E. Moffitt. 2002. "It's Not Just Who You're With, It's Who You Are: Personality and Relationship Experiences Across Multiple Relationships." *Journal of Personality* 70:925–64.

Rowe, D., K. Jacobson, and E. Van Den Oord. 1999. "Genetic and Environmental Influences on Vocabulary IQ: Parental Education Level as Moderator." *Child Development* 70:1151–62.

Sewell, W. H., A. O. Haller, and A. Portes. 1969. "The Educational and Early Occupational Attainment Process." *American Sociological Review* 34(1):82–92.

Sewell, W. and R. Hauser. 1975. *Education, Occupation and Earnings*. New York: Academic Press.

Shanahan, M. J., L. D. Erickson, S. Vaisey, and A. Smolen. 2007. "Helping Relationships and Genetic Propensities: A Combinatoric Study of DRD2, Mentoring, and Educational Continuation." *Twin Research and Human Genetics* 10(2):285–98.

Shanahan, M. J. and Scott M. Hofer. 2005. "Social Context in Gene-Environment Interactions: Retrospect and Prospect." *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences* 60: 65–76.

Sieben, I. and P. M. de Graaf. 2001. "Testing the Modernization Hypothesis and the Socialist Ideology Hypothesis: A Comparative Sibling Analysis of Educational Attainment and Occupational Status." *British Journal of Sociology* 52(3):441–67.

Smeesters, B. and A. Nayer. 1998. "La discrimination à l'accès à l'emploi en raison d'origine étrangère: le cas de la Belgique." *International Migration Papers* 23. Geneva: International Labour Office.

Smith, J. P. 1999. "Healthy Bodies and Thick Wallets: The Dual Relation Between Health and Economic Status." *Journal of Economic Perspectives* 13:145–66.

———. 2004. "Unraveling the SES: Health Connection." *Population and Development Review* 30:108–32.

Song, Y., M. J. Stampfer, and S. Liu. 2004. "Meta-Analysis: Apolipoprotein E Genotypes and Risk for Coronary Heart Disease." *Annals of Internal Medicine* 141:137–47.

Treiman, D. and K. B. Yip. 1989. "Educational and Occupational Attainment in 21 Countries." Pp. 374–94 in *Cross-National Research in Sociology*, edited by M. L. Kohn. Newbury Park: Sage.

Turkheimer, E. 2000. "Three Laws of Behavior Genetics and What They Mean." *Current Directions in Psychological Science* 9:160–64.

Turkheimer, E., A. Haley, M. Waldron, B. D'Onofrio, and I. I. Gottesman, 2003. "Socieconomic Status Modifies Heritability of IQ in Young Children." *Psychological Science* 14:623–28

Turner, R. J. and D. A. Lloyd. 1999. "The Stress Process and the Social Distribution of Depression." *Journal of Health and Social Behavior* 40:374–404.

Turner, R. J., B. Wheaton, and D. A. Lloyd. 1995. "The Epidemiology of Social Stress." *American Sociological Review* 60:104–25.

Udry, J. R. 2000. "Biological Limits of Gender Construction." *American Sociological Review* 65:443–57.

Van Den Berghe, P. and G. Mesher. 1980. "Royal Incest and Inclusive Fitness." *American Ethologist* 7(2):300–17.

Warren, J. R., R. M. Hauser, and J. T. Sheridan. 2002. "Occupational Stratification Across the Life Course: Evidence from the Wisconsin Longitudinal Study." *American Sociological Review* 67:432–55.

Weber, M. 1946. *From Max Weber: Essays in Sociology*, edited and translated by H. H. Gerth and C. Wright Mills. Oxford University Press.

———. 1947. *The Theory of Social and Economic Organization*, translated by A. M. Henderson and T. Parsons. New York: Free Press.

———. 1968. *Economy and Society Vol. I*, edited by G. Roth and C. Wittich. Taylor & Francis Books.

Weichselbaumer, D. 2000. "Is it Sex or Personality? The Impact of Sex-Stereotypes on Discrimination in Applicant Selection." Working Paper No. 0011, May, University of Linz, Department of Economics.

Winship, C. and S. Korenman. 1997. "Does Staying in School Make You Smarter? The Effect of Education on IQ in the Bell Curve." Pp. 215–34 in *Intelligence, Genes and Success: Scientists Respond to "*The Bell Curve," edited by B. Devlin, S. Fienberg, D. Resnick, and K. Roeder. New York: Springer-Verlag.