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BRAVE NEW WORLD OF BIOSOCIAL SCIENCE*

DOUGLAS S. MASSEY[1]

[1]Office of Population Research, Princeton University

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The last attempt to marry biological and social science occurred in the late nineteenth and early twentieth centuries, and at that time, the union did not go well. Neither biology nor social science was very well developed, leaving scientists in both disciplines ill positioned to make use of the two perspectives. The field of genetics, in particular, was in its infancy. The managed breeding of simple organisms such as sweet peas and fruit flies confirmed that something called “genes” existed, that genetic traits could be inherited, and that gene expression depended on combinations of dominant and recessive genes; but no one knew what genes were made of or how genetic information was transmitted in the course of reproduction.

Social science, for its part, had only recently been invented, and powerful statistical techniques, complex data sets, and sophisticated analytic models lay years in the future. As a result, there was much theorizing and little hard data analysis, yielding slow progress adjudicating between competing concepts and theories. This reality left ample room for fallible human scientists to project their own prejudices into the theoretical schemes they constructed, leading to a proliferation of competing schools of thought—structuralist, functionalist, Marxist, Freudian, and Darwinian—all with very different political implications.

The marriage of social and biological science was pursued mainly by social scientists working in the Darwinian tradition, such as Herbert Spencer, Francis Galton, and Karl Pearson. They drew from a simple model of Mendelian inheritance—the only model available at the time—in which genes were passed from parents to offspring and duly expressed as a phenotype. Once expressed, the genes were subject to natural selection through competition to produce a “survival of the fittest.” Because Darwinian social scientists were themselves White and upper class, it was a short inferential leap to conclude that people like them had risen to the top of society because of their superior genetic endowments.

This hypothesis was seemingly confirmed by the nascent field of intelligence testing, which showed that the measures of mental ability varied sharply by race, class, and ethnicity, with

*Direct correspondence to Douglas S. Massey, Office of Population Research, Princeton University, Wallace Hall, Princeton, NJ 08544 (dmassey@princeton.edu).

upper class Whites of northern European extraction coming out on top. Differences in measured intelligence also were found to follow family lines, whereas class differences persisted over time and across generations. It was thus another short inferential leap to conclude that human society could be improved by encouraging the reproduction of people with high measured intelligence and discouraging the reproduction of those with low assessed abilities. This view perforce meant promoting the reproduction of upper class northern Europeans and suppressing the reproduction of southern Europeans, non-European minorities, and the poor.

These views coalesced into the eugenics movement, which advocated the implementation of public policies and private actions to achieve precisely these patterns of differential reproduction. Eugenics ultimately went on to provide the ideological justification for such twentieth-century horrors as national origins immigration quotas, compulsory sterilization, euthanasia, segregation, and ultimately the “final solution” of the Third Reich.

It was the moral repugnance of the Holocaust that led to a decisive rejection of eugenics by mainstream scientists in the postwar period, bringing about a bitter and seemingly intractable divorce between biological and social science. In the late twentieth and early twenty-first centuries, however, advances in both fields dramatically transformed the intellectual and cultural landscape to create new and fruitful possibilities for a possible remarriage.

Within the biological sciences, the discovery of DNA in 1953 led to rapid advances in understanding of the molecular biology of inheritance and gene expression, advances that increasingly underscored the critical importance of environment circumstances in both heritability and expression. Within the social sciences, investigators created a growing stock of rich longitudinal data that increasingly included biomarkers as well as traditional social and behavioral measures. In recent years, rich individual-level data sets have been linked to detailed information about the socioecological environments that people inhabit, which in concert with the development of sophisticated multilevel statistical methods, have made it possible to model complex interactions between individual characteristics and environmental circumstances.

Both lines of scientific advance have come together in the emerging field of *epigenetics*, which in contrast to eugenics’ emphasis on simple gene inheritance and automatic expression independent of environment circumstances, instead focuses on *interactions* between genes and the environment that influence both inheritance and expression (Allis et al., 2007). Because humans are a social species whose adaptive capacities are rooted in a socially constructed order, the relevant environment in explaining human heredity and gene expression is not the physical world but the social world. Because humans are always socially embedded, social and biological scientists must work together if they are to describe and accurately understand the biosocial channels by which human outcomes are determined.

In technical terms, epigenetics refers to a “stably heritable phenotype resulting from changes in a chromosome without alterations in the DNA sequence” (Berger et al., 2008: 781). The critical insight here is that genes are not simply inherited and automatically expressed, but

they are turned on and off through interactions with the environment, yielding a variety of complex sociobiological processes that we are only beginning to understand.

A key process underlying many epigenetic outcomes is DNA methylation, which involves the attachment of a methyl molecule (a carbon atom bonded with three hydrogen atoms) to a cytosine or adenine nucleotide along a strand of DNA within a person's chromosomes. Methylation occurs throughout the course of human development and is responsible for creating, maintaining, and repairing a person's genetic code. Methylation substantially influences which inherited genes are expressed, and the process is surprisingly sensitive to environmental influences. It is now clear, for example, that variation in DNA methylation stemming from different environmental conditions can alter the expression of genes to produce different phenotypes from the same genetic material and that these differences can be passed from parents to offspring.

If genetic expression and inheritance are determined by highly variable, individually specific patterns of interaction between genes and the environment, then attempts to determine a particular human trait's heritability make little sense, especially if the trait itself likely results from the joint expression of multiple genes that themselves are activated or suppressed through environmental interactions at different phases of the life cycle. Trying to estimate a trait's genetic heritability makes sense if one is trying to quantify gene expression as part of an effort to develop disease-resistant corn seeds in a controlled laboratory environment, but it makes little sense in trying to understand the variability of expression for complex behavioral traits and social outcomes among humans.

Thus, I side decisively with Burt and Simons (2014, 2015) in their current debate with Barnes, Wright, and colleagues (2014, 2015). Indeed, in my recent book, *Spheres of Influence* (Massey and Brodmann, 2014), I argued for a more intensive study of the social ecology of human development—examining differential exposure to advantages and disadvantages within family, school, neighborhood, and peer environments—precisely because of epigenetics. A good example of the direction we need to go is the recent study by Mitchell et al. (2014), which demonstrated a gene–environment interaction in postpartum depressions. Using two variants of the serotonin transporter gene interacted with socioeconomic status, they showed that some women displayed greater reactivity to stress, leading to a greater likelihood of postpartum depression in poor environments but a lower likelihood in rich environments.

Of course, epigenetics is not the only reason for social and biological scientists to join forces, for there are other important biosocial channels by which an individual's position in the social structure interacts with human biology to produce disparate outcomes with respect to health and well-being. In addition to epigenetics, recent work has examined social-structural influences on telomere length, allostatic load, and cognitive impairment.

In terms of social structure, it is now well established that racial residential segregation interacts with high rates of Black poverty to produce a unique geographic concentration of disadvantage for African Americans (Quillian, 2012). As a result of this interaction, Black citizens are routinely exposed to much higher levels of disorder and violence than Whites.

Indeed, the distributions of Black and White neighborhoods by income and crime barely overlap (Peterson and Krivo, 2010; Sampson, 2012).

Recent studies have linked African Americans' unique exposure to disadvantage to shortened telomeres, which are repetitive nucleotide sequences located at the ends of human chromosomes. Telomeres protect genetic material from deterioration and errant recombination, and their length shortens in the course of aging and through exposure to chronic stress. Shorter telomeres are harbingers of later poor health (Epel et al., 2004).

Mitchell et al. (2014) recently demonstrated that African American boys living in disadvantaged environments display significantly shorter telomeres by age 9 than statistically similar boys who grew up in advantaged environment. This finding also had an epigenetic component, as the association between environment and telomere length was moderated by genetic variation in serotonin and dopamine pathways such that boys with high sensitivity ended up with the shortest telomeres when exposed to disadvantaged environments but the longest telomere lengths when exposed to advantaged environments.

Exposure to advantage or disadvantage as a result of one's position in the social structure also has been linked to variation in allostatic load. Allostasis refers to bodily changes experienced by people in response to changes in the environment. Potential threats trigger an allostatic reaction (the fight-or-flight response) through the hypothalamic–pituitary–adrenal axis (McEwen and Lasley, 2002). Repeated triggering of the allostatic response because of chronic exposure to stressful environment circumstances yields a condition known as allostatic load, which over time can have powerful negative effects on physical and mental health.

Research by Schulz et al. (2012) revealed that concentrated neighborhood poverty is strongly associated with allostatic load independently of individual and household characteristics and that the relationship is mediated by self-reported environmental stress. Exposure to concentrated violence and disadvantage also has been shown to undermine children's cognitive development. Sampson, Sharkey, and Raudenbush (2008) found that living in a severely disadvantaged neighborhood reduces the later verbal ability of Black children by an average of four points, an effect equivalent to missing 1 year or more of schooling. Similarly, Sharkey (2010) showed that the exposure of Black children to a homicide in their block group within the past week reduced performance on vocabulary and reading tests by half of a standard deviation or more.

These examples concretely reveal the importance of biosocial mechanisms in the production of social stratification in the United States and underscores the importance of such mechanisms in understanding the production and reproduction of poverty in contemporary society. Not only do genes and environment interact to affect the heritability and expression of genes, often in ways that undermine individual life chances, but the conditions in the social environment interact with other biological processes such as telomere regulation and allostasis to shape human destinies in potentially powerful ways. Biological scientists might understand the molecular and physiological processes underlying these phenomena, but they

do not necessarily understand the social structures and processes that give rise to the environmental context in which these biological processes play out.

It is thus essential that social scientists take part in the ongoing investigation of the growing array of biosocial processes that play out in stratified social structures. To accomplish this goal, social scientists need to establish a firmer grounding in the basics of contemporary biological thinking and, especially, to move beyond outdated Mendelian concepts of inheritance and gene expression. Our incipient understanding of sociobiological dynamics increasingly suggests that many maladies that the poor and excluded exhibit are not caused by choices or behaviors so much as by the biological consequences of their long-term exposure to stressful circumstances associated with their disadvantaged position in a stratified social structure.

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