The Emergence of Socio-Genomics
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What is This?
Ever since Darwin penned *The Descent of Man* in 1871, there has been a contentious relationship between biologists and many of us who seek to explain human difference—i.e., social scientists. There was, of course, Herbert Spencer, who applied natural selection as a metaphor to human society. And there was Darwin himself, who became embroiled in a debate about whether blacks and whites constituted separate species.¹ The controversy surrounding the relationship between evolution, genes, and society has ebbed and flowed over the 150 years since *Descent*. Most recently, the revelation that the Beijing Genomics Institute is sequencing the genomes of 2,000 individuals with IQs over 150 in search of intelligence genes has led to renewed concerns in the West of an emerging program of eugenics through embryo testing and selective abortion (Normile 2002).

Part of the reason for such trepidation when it comes to the examination of genetics as it relates to human behavior is that it is commonly assumed that the answers obtained by examining genetics are deterministic ones: to the extent that genes explain any social outcome, this “naturalizes” any inequality in that outcome. For example, many queer activists have cheered the search for the “gay gene” in hopes that if the innate, genetic bases of homosexuality were found, it would increase tolerance since LGBT individuals will be shown not to be making a lifestyle (i.e., moral) choice about their orientation (Hamer et al. 1993).

Conversely, in the best-selling book, *The Bell Curve*, Richard Herrnstein and Charles Murray argued that thanks to meritocracy, today class stratification is based on innate (i.e., genetic) endowment (Herrnstein and Murray 1994). Meanwhile, by selectively breeding with others of similar genetic stock, parents reinforce their offspring’s advantages or disadvantages. In their view, social policy to promote equal opportunity is counterproductive since each individual has reached the level of social status best suited

¹ Ethnocentrists of the nineteenth century had sought to use Darwin’s concepts to argue for fundamental biological differences between the groups we call races. When Darwin countered these claims, he curiously found himself allied with his one-time foes: the religious conservatives of the day who also argued for the unity of humankind.
to his/her native abilities. This is the nightmare conclusion of progressive social scientists and the reason why most avoid genetic data like it was the plague.

If all of our human outcomes and traits are now increasing explained by discoveries in genetics, and some of our basic social constructs becoming complicated by incorporating findings from genetics and the biological sciences, will the social sciences lose their relevance to understanding human social behavior? Put another way, if heritability measures are large and accurate or if genetics is pointing the way toward new and better kinds of “racial” and social classification, why do we still need sociologists, economists, and political scientists?

As it turns out, however, the more serious empirical investigations of genes and society that these new data afford often yield counterintuitive results. For example, it turns out that while genes matter for both IQ and social class, they are just as much an engine of social mobility as they are of social reproduction thanks to the mixing up that takes place through chromosomal recombination when sperm and eggs are formed.

Meanwhile, a deeper look at race shows that genetic analysis does not reify our racial categories but instead destroys them: thanks to the population bottleneck coming out of Africa, we Caucasian authors of this article are most likely more genetically similar to Eskimos than are two Ugandans 200 miles apart (Tishkoff et al. 2009). The vastly greater genetic diversity within African descended populations does not just mean that race as we knew it is a mirage. It also has real life and death consequences today: it is much harder for an African American in need of an organ transplant to obtain one (Daw 2013). This longer waiting list for blacks is not primarily due to racism on the part of anonymous donors or transplant surgeons. Nor is it due to the “dissolution” of the black family—i.e., lack of relatives willing to donate. It is simply a result of the greater difficulty in finding a good match when the genetics of a group vary so much, even among siblings.

Something as simple as the effect of sin taxes takes on a more complicated spin when viewed through the prism of genetics. Do cigarette taxes lower smoking rates?

Well, yes and no. As it turns out, some of us have a variant of the nicotine receptor that makes us very sensitive to price. Smoking is a luxury that we can give up when its price rises. For those of us with a different version of that same protein, we might keep our two-pack a day habit even if the price rose to $100 a pop. Below we highlight some interesting work in this area that speaks to these and related issues. It is by no means meant to be a “best of” nor a comprehensive review of the literature but rather a non-representative buffet of interesting (to us) research:

The rise of professional social sciences was in many ways a response to Darwinistic interpretations of human behavior. Efforts by scholars like Galton and others to establish a science of eugenics engendered social scientific responses attempting to show the importance of the environment in shaping outcomes ranging from delinquency to class attainment to family relations. However, the science of genetics never gave up on trying to show the genetic bases of many human tendencies. During the 1970s, a spate of studies emerged suggesting that between a third and a half of socioeconomic outcomes could be explained by “genetic” differences. Even though it was some of their own who authored many of these studies, social scientists wasted no time in questioning both the core assumptions in the models that gave rise to these estimates of “heritability” as well as the utility of such estimates themselves. The fire was further fanned by the publication of The Bell Curve during the 1990s, which argued that meritocracy had led to the perverse outcome that inequalities were largely based in innate ability—and thus resistant to being remedied by public policy. The Bell Curve was, in short order, attacked for its poor methodology and ideological spin. However, the truth is that all of these studies—which utilized twins, adoptees, separated family members, and so on—were really just trying to estimate the size of a black box called genotype without being able to peer inside it. Meanwhile, until recently, no social scientists even bothered to defend the concept of “heritability” as a useful datum in the designing of policy. But recently, a new group of sociologists, political scientists, and economists have
joined forces with statistical geneticists to make serious arguments about the utility of genetic information toward understanding social dynamics and about the right ways to get that information.

Fast forward to today and that black box has been cracked by cheap DNA genotyping platforms that allow a researcher to actually measure around a half-million or more of the base-pair differences between individuals for a few hundred dollars (and impute up to three million more markers of human difference). The genomics revolution ran into some stumbling blocks early out of the gate, however. These included inadequate sample sizes for studies that would search for statistically meaningful effects with hundreds of thousands of discrete “hypotheses” (i.e., for each marker) and the fact that the markers typically measured by the big chip companies (Affymetrix, Illumina, and so on) were common variants and ignored rarer and potentially more powerful sites of difference among humans. The result of these and other challenges was that these measured markers did not seem to explain the level of variation in outcomes—ranging from schizophrenia to height to IQ—that was meant to be due to “genetics” according to the prior generation of twin studies. Slowly but surely, statistical geneticists have made significant progress in solving this “missing heritability” mystery using a range of newly developed tools. For example, did the earlier twin studies overestimate the role of genes to begin with? That is, the key assumption of 40 years of twin studies (and their would-be Achilles heel) is the fact that the estimation of genetic influences rest on the notion that the reason identical twins demonstrate more similar outcomes than do their same-sex fraternal twin counterparts is that they share more DNA in common. However, plaguing this research is the fact that these twins are probably experiencing more similar environments as well, both because of how others treat them (confusing them, for example) and because they may be closer to each other as well and thus mutually influence each other’s choices and experiences than fraternal twins do. This “equal environments assumption” has been the weak link in genetic studies—until now. Now that we have actual DNA markers of twin sets, a natural experiment emerged: some twins were misinformed about whether they were monozygotic or dizygotic. These “misclassified” twins allowed us to see if those twins who lived their entire lives as identical but were genetically fraternal (and vice versa) led us to the same results as those earlier studies. That is, if there was indeed any conflating of environmental effects with genetic ones, these misclassified twin sets should tease that out (Conley, Rauscher, and Dawes 2013). As it turned out, the equal environment assumption of the 1970s held, and the case of missing heritability remained unsolved. For now, at least.

Meanwhile, statistical geneticists have developed a number of innovative techniques to solve this missing heritability problem. For example, while on average siblings share 50 percent of their genome, there is significant variability in that figure thanks to the randomness of recombination and segregation of the grandparental alleles when parents form sperm and eggs. So one pair of siblings may share 45 percent of their genome while another (even in the same family) may share 60 percent. By correlating these differences in relatedness among siblings to differences in their outcomes, we are able to generate an unbiased estimate of the genetic contribution to a given trait—i.e., heritability (Visscher, Medland, and Ferreira 2006). And lo and behold, those 1970s estimates again hold up. Using this sibling and other approaches, earlier heritabilities were confirmed. And while any individual DNA base (or entire gene for that matter) may not tell us much in terms of how we turn out, the sum total of all nucleotide differences did provide a measure of genetic stock—if you will—that was predictive of important social outcomes, directly measurable and, to a certain extent, randomly assigned at birth (Rietveld et al. 2013).

At the same time, more and more national surveys were asking respondents to spit into a cup, adding genotype data to the rich tapestry of social variables that economists, sociologists, and political scientists had worked with for decades. It seemed that genetics has once and for all gained a foothold in social science. And why not? Why should we be afraid of additional data that may help scientists better understand patterns of human behavior, enhance
individuals’ self-understanding, and design optimal public policy? Especially when the answers we get from peering into the black box are not always—or even often—the kind that reify existing inequalities, assumptions, and policies. As it turns out, adding genetic data to social science upends the apple cart on many of our assumptions. For example, were Herrnstein and Murray right in The Bell Curve when they argued that meritocracy has perversely resulted in more intransigent inequalities today because we are now sorted by genetic ability? As it turns out, the data show that thanks to the magic of sexual reproduction—where the deck of genetic cards is reshuffled each generation—genetics does as much (if not more) to upset existing inequalities (i.e., create social mobility) than it does to reinforce social reproduction.

So far, we have focused our attention on what new discoveries in genetics can tell us about very thorny issues and ideas in modern society such as racial identity or IQ. In general we have neglected a focus on the environment—specifically how environmental factors upend (again) received wisdom about genetic determination and the naturalization of human behaviors and societal structures. However, we now bring the environment back into the conversation and discuss the many complications that emerge. Indeed, genetics and environmental factors seem to interact to determine what we see in human behavior—it is not “nature vs. nurture” as much as “nature and nurture.”

This idea of nature and nurture dynamically interacting and interplaying with one another has also led to various ideas that feed back into our understanding and theorizing in genetics and evolution. An early suggestion was the stress-diathesis hypothesis, where the idea was that some individuals were born with “risky” genetic variants and others were born with “safe” genetic variants—but both individuals would have similar outcomes if placed in a neutral environment. However, if individuals with “risky” genetic variants were placed in “risky” environments, we would see disproportionately different outcomes—a gene-environment interaction. Indeed, there is quite a bit of (still controversial) evidence that children who are abused and have “risky” gene variants related to the serotonin or dopamine systems in our brains experience incredible reductions in life outcomes, while abused children with alternate gene variants are worse off, but not multiplicatively so (Caspi, Sugden, and Moffitt 2003; Risch, Herrell, and Lehner 2009; Conley and Rauscher 2013; Karg and Burmeister 2011; Conley, Rauscher, and Siegal 2013).

The next question for the stress-diathesis hypothesis, though, comes from an evolutionary perspective—why would humans have “risky” genetic variants at all, if there were no benefits? Why would evolutionary pressures fail to wipe out these variants? Two theories have been proposed. The first set of theories is that these “risky” variants were, in the not-too-distant past, beneficial. Perhaps the genetic variants that we seem to see producing “over-reactions” to current stressful environments would have been excellent variants to have in the dangerous Serengeti, and humans are too recently displaced from these environments for the variants to have disappeared through evolution. Or perhaps these “risky” variants are risky for some outcomes but actually protective for other, often unmeasured, outcomes.

A second theory considers the gene at the level of the species rather than individual and supposes that there are variants which are “orchids” and other variants which are “dandelions.” The dandelions do fine in most environmental circumstances, within a reasonable range. However, the orchids thrive in some environments but wilt in
others. Again, this is gene/environment interaction, and at the level of a species, humans would like to have both types of genes in our pool in case of larger changes in the environment (e.g., climate change) that might wipe out all the dandelions and most of the orchids, but allow some orchids to thrive (Conley, Rauscher, and Siegal 2013).

These theories and the evidence for gene/environmental interactions also potentially have more pressing implications: while we are waiting for evolution to change genotype distributions, we would also like to figure out how the purposeful shaping of our environment that is under human control may affect us. This leads us into thinking about the built environment, schooling, health policies, energy use, and the whole array of government action.

Specifically, we might ask what allows some policies to be effective and others to fail? Many policies are guided by theory and data. We tax products we do not want people to use because economic theory posits a Law of Demand—when prices go up, people consume less of the product. And this theory is often verified by data—when states increase taxes on cigarettes (or soda or whatever), people shift their choices away from these products. We give people housing vouchers to move away from impoverished neighborhoods following sociological and economic theories about the presence of poverty traps and the importance of neighborhoods. However, our policies have a very mixed success rate. Some are successful for some people or during some periods of time but not others. New evidence merging genetics and public policy has started to uncover why we see such different impacts of the same policy for different people and how future policies might be targeted—taking the concept of “personalized medicine” to allow “personalized policy.”

A case in point is the example of tobacco taxes in the United States. The Institute of Medicine and Centers for Disease Control and Prevention, among others, have ranked this policy as one of the top ten most impactful on increasing public health in the past century. This is because the United States has witnessed enormous changes in smoking—which have been cut in half since the first taxes were introduced following the Surgeon General’s report in the mid-1960s. And raising taxes is cheap for the government, so the cost/benefit ratio is incredibly favorable. But . . . the last decade has seen a reversal. In the face of the largest tax increases in our history, tobacco use has remained virtually unchanged. Has the Law of Demand been repealed? New evidence that combines genetics and social policy evaluation points to an explanation: the pool of smokers in the 1960 was genetically different from the pool of smokers now. People carry around different variants of a group of nicotinic receptor genes with them—and as the name suggests, these genes decide how much dopamine (a pleasure chemical) is released when you smoke; how much you “like” smoking. These genetic variants also interact with our environments. It looks like many of the smokers in the 1960s did not need a big push (tax increase or social pressure) to quit smoking (or never start). But over time, these small pushes have mostly pushed people to stop smoking if their genes did not put up a big fight. And what we have left in the smoking pool are disproportionately folks whose genes are fighting back, partly because nicotine is so pleasurable. This shows up in evaluations of tobacco tax policy, where only adults with low genetic risk of smoking will still respond to taxation and adults at higher genetic risk seem to be unshaken (Fletcher 2012).

What does this mean for policy? Should we keep increasing taxes—forcing the remaining smokers to pay a greater and greater share of their incomes in part because they were unlucky in the parental (genetic) lottery of life? Or should we subsidize their smoking because they love it so much (due in part to their genetics)? Smoking is not the only area where “personalized” policy may come into play. Evidence has shown that some educational interventions have greater or lesser effects depending on targeted students’ genotypes. Ditto for crime prevention policy. And even the Earned Income Tax Credit spurs low-wage workers to labor more or less depending on their genes. While personalized medicine may be just over the horizon, the potential for custom-tailored social policy is here today. Whether we want to go down that path is another question altogether.
Genetic analysis not only can inform micro-level analysis of human behavior, it can also add to macro-historical analysis of social phenomena. For example, a fundamental question in macroeconomics is why some countries have thrived and others have stagnated over the past several hundred years. There has been a revival of interest in this topic in the past few decades and many novel and stark hypotheses have been proposed. Jared Diamond, in a series of books and articles, has suggested that environmental factors that contribute to differences in disease burden, soil efficiency, and similar “endowments” across countries are a primary source of difference in what we currently see between rich and poor countries. For example, tropical diseases reduce life expectancy in places like Zambia so that trained workers can expect to have only 10 years of economic productivity—the same figure for the United States is over 35 years (Diamond 1997). Agricultural grain yields in U.S. soil are about 10 times greater per acre than for their African counterpart. And African River Blindness means that Africa is the only continent where populations concentrated away from the rivers and coasts, where the most fertile soil is and where growth-stimulating trade is facilitated.

More recently, economists Daron Acemoglu and Simon Johnson and others are dismissive that the environment is a key factor explaining economic success and instead consider the development and expansion of institutions (legal systems, democracy, property rights, corruption, and so on) as the key set of factors explaining why some countries are rich and others are poor (Easterly and Levine 2003; Easterly et al. 1993; Acemoglu 2002). Some countries arrive at having “extractive” institutions in which small groups of individuals exploit the rest of the population (think diamond mines in Sierra Leone or Congo) while other countries arrive at “inclusive” institutions where many people are included in governance (Acemoglu and Robinson 2012). Key to this argument are examples where geography is held constant but institutions differ. For example, North Korea and South Korea share a peninsula and a micro environment. The land is of similar quality, the topology of the environment not too different, even the populations are nearly identical. However, a history of inclusive governance in South Korea, and one of non-inclusive (extractive) governance in North Korea, Acemoglu and Johnson argue, shines a bright light on the importance of institutions and not geography as the key to development. Think also about the economic performance of East vs. West Germany or economic growth trajectories in towns on the U.S. versus Mexico side of the Rio Grande—similar environments, similar people, similar disease burden, but different institutions and different growth and development.

As these new ideas are being sorted out, economists interested in the intersection of economic development, institutions, and geography have begun to explore another aspect of populations that might fit into these grand theories: Population Genetics. In particular, a new breed of macroeconomists has posited that genetic diversity within countries is a key to development. Quamrul Ashraf and Oded Galor published a paper providing evidence that a “goldenlocks” level of genetic diversity within countries might lead to higher incomes and better growth trajectories (Ashraf and Galor 2013). The authors discuss the observation that there are many countries with low diversity (e.g., Native Americans) as well as populations with high diversity (e.g., many sub-Saharan African countries) that have experienced low economic growth, while many countries with intermediate (just right) diversity (European and Asian populations) have been conducive for development in the pre-colonial as well as modern eras. Basically areas/countries face a tradeoff with genetic diversity between cooperation and innovation. The idea is that areas with genetic diversity that is “too high” have higher likelihoods of disarray and mistrust, which would reduce cooperation and disrupt socioeconomic order and lead to low productivity. However, areas with genetic diversity that is “too low” might have less innovation, fewer new ideas and therefore reduced technological capacity. The authors argue that higher diversity “enhances society’s capability to integrate advanced and more efficient production methods, expanding the economy’s production possibility.
frontier and conferring the benefits of improved productivity”—an idea motivated by Darwin’s theory of evolution by natural selection. These conflicting costs and benefits of genetic diversity lead to the proposition that a “middle level” of diversity will lead to the highest growth and development patterns. The authors find that, for countries that have low genetic diversity, small increases in diversity (one percentage point) could increase their population density (a measure of economic development) by 58 percent; likewise countries with current high genetic diversity who reduced this diversity by one percentage point might see an increase in population density of 25 percent.

In addition to considering the “optimal” level of genetic diversity in populations to maximize economic development, other economists have considered the role of population genetics as it interacts with environmental resources to affect growth patterns across countries. Justin Cook has shown that populations with the (genetic) ability to digest milk after weaning that appeared early in human history conferred large advantages in population density around 1500 CE (Cook 2013a). As other studies have shown that economic development differences in history have been remarkably persistent, the implication is that (relatively) small changes in the genome, at the right time and in the right place (during the Neolithic Revolution in areas able to raise cattle) can lead to large, persistent, and accumulating differences in economic development across countries.

Macroeconomists have also begun exploring how population genetics might affect economic development through the health of populations which then would affect productivity and incomes. For example, Justin Cook has also shown that populations with immune systems that are “genetically diverse” have had a health advantage in the pre-modern period (Cook 2013b). The idea is that pathogens evolve to target specific immune function weaknesses, and populations with limited genetic diversity (and hence limited diversity in immune response) are at particular risk of infectious pathogens spreading and reducing the health of wide swaths of the population. However, at the population level, genetic diversity can be beneficial in inoculating against such widespread health insults by constraining epidemic spreads of illness. Because many pathogens specialize in their attack strategy, populations with multiple defenses (through genetic diversity) can potentially reduce the long-run effects of infectious diseases on population health. Indeed, Cook finds that increases in immune genetic diversity (through the human leukocyte antigen [HLA] system) leads to increases in population (country-level) life expectancies. He then further documents this causal argument by showing that the invention and widespread use of modern vaccinations and other medical technologies has led to a decline in the genetic advantage. That is, modern science and medicine is substituting for “natural” (genetic) defenses against illnesses—at the population level—and in doing so is promoting convergence in life expectancy, and eventually growth and income, across rich and poor countries. This is another example of gene-environment interactions, at the population level. In earlier times (in diseased environments with lack of medications), genetic variation acted as a buffer against disease—leading to country-level differences in life expectancy based in part on genetic differences. But now that the environment has changed, with new medications/vaccinations, the previous genetic advantages have been largely eliminated, so that genes interact with the larger environment in producing outcomes. Likewise, in our earlier example of the “milk genes”—these genes only confer advantages when in environments which have the ability to foster agriculture. With no cows, goats, or other domesticable mammals, the gene confers no population advantage.

Conclusion

Until recently, the study of human genetic variation has consisted mainly of behavior genetics studies, where twin and adoption designs were used to identify heritable, or genetic, variation in various traits (e.g., Björklund, Lindahl, and Plug 2006; Plomin, Owen, and McGuffin 1994; Plomin 2009; Plug 2004; Sacerdote 2007). These studies are controversial and the assumptions underlying them have been questioned.
Whether social scientists are interested in embracing, not fleeing, this deluge of data, they would do well to integrate genotypic data into their analyses—and ignore it at their peril.

References


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In addition to citizen/consumer genotyping databanks, major funding bodies, including those in the social and behavioral sciences, have now begun to incorporate genetic and biological markers into major social surveys. For example, in addition to the pioneering study—the National Longitudinal Survey of Adolescent Health (Add Health)—more recently the Wisconsin Longitudinal Study (WLS) and the Health and Retirement Survey (HRS) have released datasets with comprehensively genotyped subjects. Similar efforts are also underway in Europe, for example with the Biobank Project in the United Kingdom (Ollier, Sprosen, and Peakman 2005; Platt et al. 2010) and large-scale genotyping of subjects at several European twin registries (Rønning and Paltiel 2006). These datasets provide new opportunities for asking scientific questions that could not be explored until very recently.

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