

What Can the Slim Initiative in Genomic Medicine for the Americas (SIGMA) Contribute to Preventing, Treating, or Decreasing the Impact of Diabetes among Mexicans and Latin Americans?

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Many years ago, in the late 1980s, as a postdoc in genetics at the University of California at Davis, I was interviewed by National Public Radio on the subject of the Human Genome Project, then beseeching Congress and the American public for a few billion dollars. Sure, it would keep molecular biologists employed into the foreseeable future, but was it science? Of course not, I told NPR, with the assuredness that comes with having recently earned a doctorate and of working in a laboratory with radioactive isotopes, toxic chemicals, and expensive machines with flashing multicolored lights. Science involves testing hypotheses; we all know that. . . .

A few years later, I was recruited to help review a few hundred grant proposals by the scientific society Sigma Xi, which gives small sums to graduate students starting their thesis research. I was instructed to divide the proposals into two piles: those that tested hypotheses, and those that did not. The ones in the first pile would get about five hundred dollars each, and the ones in the second pile would not. So, if you did not test a hypothesis, you could be denied five hundred dollars, but you could get three billion.

—Jonathan Marks, *Why I Am Not a Scientist:
Anthropology and Modern Knowledge*

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Introduction: The Science in SIGMA

First, I thank Terence Keel for inviting me to participate in this conversation. If I understand the debate correctly, one key issue is how social scientists—especially those who are skeptical of technical fixes to social problems—can make sense of highly technical fields, such as genomics, so that their work may complement rather than interfere with the contribution of these fields to the health and well-being of traditionally excluded groups such as racialized populations or women.

In particular, we might ask whether the Slim Initiative in Genomic Medicine for the Americas (SIGMA), sponsored by a partnership between the Broad Institute and the Carlos Slim foundation, could ever contribute to its purported goal of addressing high levels of diabetes among Mexicans and Latin Americans, given its inherent conflict of interest (SIGMA Type 2 Diabetes Consortium 2014). By conflict of interest I mean the tension between the kind of world that makes possible the fortune of Carlos Slim—who stood fourth in the *Forbes* ranking of world billionaires for 2016 (Kroll 2016)—and the kind of world that would promote the living conditions needed to lessen the disproportionate impact of diabetes, especially type 2, among certain populations. These disparities have been well documented, now over several decades, in the Americas and throughout the world (World Health Organization 2016; Zimmet 1996).

Another angle might be to question SIGMA's assumption that "Mexicans and Latin Americans," on the one hand, and "Europeans and Africans," on the other, are biologically distinct populations (SIGMA Type 2 Diabetes Consortium 2014). Can a study that purports to have identified gene variants predisposing the first of these populations, but not the second, to type 2 diabetes (hereafter "diabetes") yield medically useful information—i.e., applicable to prevention, diagnosis, or treatment? (I invite readers to ponder whether they would locate the author, born to Jewish parents of Polish and Syrian descent and raised in Latin America, in the same "genetic/ancestry group" as her parents' caregiver, of 100 percent Native Latin American ancestry, as SIGMA researchers would.)

Here, however, I set aside these potentially fruitful issues, as well as the question of whether Neanderthal genes contribute to the risk of diabetes, as SIGMA researchers suggest, since I have no qualifications in these matters. Rather, I concentrate on the science underlying SIGMA in order to ask whether the initiative can achieve its stated goals. Does identifying diabetes gene variants that are present with a frequency greater in some populations than in others illuminate the pathophysiology of diabetes, thus contributing to preventing, diagnosing, treating, or decreasing the disparate impacts of this disease? My decade-long experience in medical practice and an additional decade and a half studying the so-called genetics of diabetes as a social scientist have led me to conclude that the short answer is "no." Let me explain why.

Gene Variants for Spanish Language?

For the sake of my argument, which I elaborate to the limit of its implications, I begin by accepting one of SIGMA's key assumptions. The study rests on the idea that there exists a gene variant, SLC16A11, that is relatively common among Mexicans and Latin Americans yet rare among Europeans or Africans. Moreover, knowing the presence or absence of this variant in any random individual in the world would increase our ability to predict group membership—i.e., whether the individual is Mexican or Latin American rather than European or African. Again, for the sake of the argument I assume that these categories are straightforward and easy to operationalize.

I subsequently assume that the presence of this variant indicates an identifiable “risk” or “genetic influence” (however one understands the concept)—a risk not of experiencing diabetes, as proposed by SIGMA, but rather of speaking Spanish. Put another way, based solely on the fact that SLC16A11 is more prevalent among certain populations than others and is associated with Spanish language skills, I assert that the said gene variant explains these skills, not in the *technical meaning* of “explain” as the term is used by statisticians (i.e., accounting for observed variation among groups but not implying necessary causality) but in its *most common meaning* as understood by ordinary mortals, including clinicians and other health professionals, as “cause.”

In fact, I could complicate my argument further by following the Framingham Heart Study, one of the first prospective diabetes studies, which used a genetic risk score (GRS) computed from eighteen loci associated with diabetes to predict who would develop the disease over time (Meigs et al. 2008). Framingham researchers found that adding this GRS to a predictive model based purely on clinical markers (age, fasting plasma glucose level, HDL-cholesterol levels, body mass index, etc.) increased the C-statistic of the model, representing its ability to distinguish subjects with and without the outcome of interest, from 0.9031 to 0.9050. In the words of another researcher, the GRS added “only marginal information” to well-established clinical markers of diabetes (Hivert, Vassy, and Meigs 2014, 2), barely greater than “flipping a coin” (Lyssenko and Laakso 2013, S122) and “not yet useful for clinical predictions” (Billings and Florez 2010, 11, emphasis added).

Be that as it may, I propose that the identification of the gene variant SLC16A11 among Mexicans and Latin Americans, either in isolation or combined with eighteen (or forty, or sixty-two) others, indicates that the ability to speak Spanish is subject to genetic influences.¹

At this point readers might be puzzled and conclude that I've lost my marbles. Although the assertion that genes influence diabetes appears to make sense, the assertion that genes influence the ability to speak Spanish clearly does not. But why?

The answer lies in a major conceptual confusion that all too frequently pervades studies of genetic influences on X, or Y, or Z, where X, Y, and Z are so-called “complex” or “common” phenotypes, whether in humans or animals. To put it simply, by “common” or “complex” phenotypes or traits, including diseases, the scientific literature means traits presumed to be caused by multiple, nonadditive inputs—which can be environmental, developmental, and genetic—rather than by one major gene variant determining the trait, such as with blood type.

But readers may still wonder: Aren't conceptual confusions the business of philosophers, whereas the business of scientists is to collect data, deal with empirical evidence, and run experiments? What do they have to do with science, let alone with science of a practical sort, intended to improve human health? I believe that when making claims about health and disease, or any other issue pertaining to human welfare, the implications of confused conceptual models for evaluating the worth (or worthlessness) of a given scientific enterprise are huge, measurable in significant human suffering as well as in millions of dollars divested away from the real causes of major public health challenges. How best to deal with the well-documented, and undisputed, disproportionate impact of diabetes on certain populations is merely one implication of the conceptual confusion I develop below.

Of Conceptual Muddles and Scientific Nonstarters

So where do we begin? Let me use the example of one popularizer of genomic research and medicine, Mary Carmichael. Carmichael (2010) argues that it cannot possibly be correct to assert, as I and others have done (Chaufan 2007; Chaufan and Joseph 2013; Joseph 2000a; Latham and Wilson 2010), that gene variants have never been shown to explain—not *statistically* but *empirically*—observed differences in the distribution of complex phenotypes such as diabetes (or cancer, or schizophrenia, among many others), notwithstanding weekly claims to the contrary from lay and expert sources. As she writes in a 2010 blog, affectionately mocking the claims of food expert Michael Pollan, whom she otherwise admires:

A number of people outside the genetics community also must have seen [Pollan's] essay [arguing that genes play virtually no role in the majority of diseases]. Maybe some of them read it and thought, “huh. My kid looks just like me, but these people are saying genotype has little to no effect on phenotype. Do they really think my kid has my distinctive Roman nose because he grew up in my house?” Maybe some of them wisely thought, “hmm, I was taught that nature and nurture both mattered, this can't be right.”

Quite differently from what Carmichael (2010) suggests about “environmental determinism”—that our unique genotypes do not matter to anything—my

argument assumes that whatever influence gene variants may have on diabetes, it cannot be *empirically*, as opposed to *statistically*, separated from environmental inputs and developmental noise (random molecular motion within the local cellular environment) (Griffiths et al. 2005). This makes the enterprise of identifying gene variants influencing *complex* phenotypes, such as diabetes—as opposed to gene variants influencing *rare* phenotypes (under 1 percent of diseases) such as phenylketonuria—both a conceptual muddle and a scientific nonstarter.

Faced with this claim, the genetics popularizer is puzzled. As illustrated in the quote above, Carmichael reasons that it cannot be the case that if a child's nose resembles her parents' this is because she grew up in the same house as her parents, or because of some obscure technical concept like developmental random noise. The only possible answer *has* to be the genes. And on this matter she is obviously right.² She would be wrong, however, to conclude that it follows that if a child speaks Spanish as her parents do, *it must also be because she carries the same gene variant* or combination of variants that her Mexican or Latin American parents, who also speak Spanish, carry—such as SLC16A11.

This is because it is well established that a child will speak Spanish if she grows up in a Spanish-speaking household or is otherwise exposed to Spanish from an early age. Language ability is a crystal-clear case in which gene variants play no role whatsoever in a child's ability to speak a language—not a human language *in general*, but *a particular language*—however much she may share particular gene variants with her parents or other members of her “race” (whatever that may mean), and however widespread the observation that language abilities run in families. Environmental factors alone make the difference in a child's ability to speak Spanish: they explain, perhaps statistically but definitely empirically, why a child speaks that language; meanwhile, whatever complicated statistical acrobatics may indicate the presence (or absence) of gene variants, these variants are causally irrelevant.

Granted, those of us who reject genetic explanations for a child speaking Spanish (as opposed to, or at least in addition to, some other language) do not mean that genes *in general* do not affect language abilities *in any way*. After all, when it comes to a pet dog rather than a child, growing up in the same environment (or same amount of developmental random noise) would not do the job of influencing a puppy to speak Spanish. So we may be willing to accept that *in some sense*, speaking Spanish would be due to genetic influences, because speaking a human language requires a human genome rather than that of a dog. When it comes to explaining *differences in language abilities between children and puppies*, *gene differences between species are indeed difference makers*. This is the case even if the specific difference-making genes between species—causing *humans, but not puppies*, to speak *any* human language—are as yet unidentified (assuming they exist).

But of course this is not what Carmichael means when she alludes to gene differences causing phenotypic differences. Nor, I suspect, is it what SIGMA researchers mean when they assert that Mexicans and Latin Americans who carry the gene variant SLC16A11 are “genetically predisposed” to type 2 diabetes *for that reason* (in addition to others). They allege a plausible, yet speculative and untested, alteration of the lipid metabolism *that would make a relevant difference, all else equal*—while suggesting that those who do not carry this gene are less susceptible to the disease. SIGMA researchers move on to explain the differences *within* groups of Mexicans and Latin Americans via their degree of “genetic admixture” with Native Americans, about 50 percent of whom appear to carry the “diabetes gene variant.” This variant is in turn allegedly known to explain *their* high rates of diabetes, according to studies based on similarly shaky assumptions (Cheng et al. 2012; Tandon 2015).³

Put another way, SIGMA researchers do not mean that a human genome is necessary for developing diabetes (which, by the way, puppies can develop too). Rather, they assert that *some* members of the *Homo sapiens* species carry *particular* gene variants, such as SLC16A11, that *cause*—empirically and not merely statistically—their diabetes. That is, these variants *are the difference makers* of their diabetic state, to some *measurable and quantifiable degree*: carriers of these gene variants are 29 percent more likely to have diabetes than the noncarrying group, according to SIGMA (2014, 97). They also imply that those who are not carrying the variant would remain free from diabetes, *even under exactly the same conditions relevant to the developmental history of diabetes*. Further, they assert this discovery without having conducted a single experiment, merely identifying gene variants more common among those *already diagnosed with diabetes*.⁴

Genetic Influences or Just-So Stories?

If we knew nothing about the developmental history of language—which, everyone agrees, shows that gene variants are unnecessary to explain *differences in the ability to speak one language rather than another one*—we might attribute the fact that specific languages, like Spanish, generally aggregate in cultural/social/racial groups, or families, to as-yet-undiscovered genes. In fact, we could attempt to measure the *heritability* of speaking a specific language, and even compute some number, a really impressive number, even if this number would not be meaningful, i.e., not remotely measure what genomic researchers would love it to measure: the magnitude or extent to which genes *cause* language abilities (Chaufan 2009; Lush 1949; Shönemann 1997; Wahlsten 2003). In fact, it would be as meaningful as claiming that because height is influenced by genetics—as asserted by geneticists who claim that the heritability of height, which they measure at 80 percent, provides evidence for this assertion (Polderman et

al. 2015)—twenty-four inches of my five-foot stature must be caused by genes, whereas the rest would be caused by environmental influences and developmental random noise, and that moreover, the specific genes contributing to those two feet can be identified by genetic studies. None of this makes any sense at all—it is literally nonsense (Chaufan and Joseph 2013).

This misleading kind of thinking has a long history: it is the thinking pervading causal debates around pellagra in the US South at the turn of the twentieth century. The condition was declared to be (partly) “hereditary” (Davenport 1916)—today we would say “genetic”—because it aggregated among certain social groups and ran in families, although this explanation turned out to be false.⁵ At any rate, even if, for the sake of the argument, we granted the existence of a genetic predisposition to pellagra, the fact is that pellagra was wiped out with the addition of niacin to basic staples. Consequently, as has been noted, we will never know whether the descendants of former sufferers bore (as they would) their parents’ supposed genetic predisposition (Joseph 2000b). We do know, however, that the phenotype “free from pellagra” is invariant—i.e., displays no relevant variations—when the population is adequately nourished, and that whatever gene variants individuals may carry make no difference to their pellagra (or pellagra-free) status. And if there were a new outbreak of the disease, one would need a lot of spare change to undertake a search for pellagra genes rather than ensuring that everybody received the right amount and type of food—that is, unless one’s job, reputation, or income depended on it.

Clearly, if we know very little about the developmental history of some disease (or if, for whatever reason, we refuse to acknowledge how much we know already—but I digress) the fact that it may aggregate in certain groups, or run in families, may lead to the belief that specific gene variants cause some individuals to suffer from the disease while *others under exactly the same conditions relevant to the development of the said disease* do not. But the inference would be unwarranted.

Biology or Ideology?

What can we conclude, at least tentatively, about the possible implications of SIGMA for the betterment of the health of Latin American populations in the Americas, one ostensible goal of the initiative? Or for the use of racial categories as proxies for genetically relevant health and disease information more generally? These questions are critical, as there is a real risk that, when faced with increasingly mind-boggling computations of “mathematical elegance and biological vacuity” (Lewontin, Rose, and Kamin 1982, 13), non-experts, including social scientists, may be tempted to walk away from assertions about the importance of gene variants to disease predispositions, shrugging their shoulders. They might conclude, as Carmichael (2010) did, that if genetic reasons cause a child’s nose to

resemble that of the parents, then similar reasons, including specific gene variants, must also influence—i.e., cause—other matters of life, health, and death.

More troublingly, as the mass media uncritically reproduce the latest so-called genetic discovery—crediting gene variants with everything from developing cancer to getting good grades at school, voting conservative rather than progressive, or believing in God—the uninitiated may be seduced by expert claims that closing the gap in genomic research between whites and racialized populations will lead to more equitable health policies (National Institutes of Health 2017). The uninitiated may also conclude that anyone who denies this must be stubbornly unwilling to allow the fruits of science to be broadly distributed in society, and perhaps that the deniers are unreasonably concerned about the historical roots of genomics in the eugenics movement (Kevles 1995).

Elsewhere, I have laid out in detail the developmental history of diabetes and the myriad ways in which a history of exposures (in addition to chance), beginning quite literally at conception, empirically explains observable variations in diabetes among social groups. This remains the case even if we cannot pin down every single factor with precision—and indeed, given the current knowledge *already* available, such an effort would not serve any health-promoting purpose (Chaufan 2007). In fact, there are excellent reasons to believe that no special genes are necessary to account for the staggering rates of diabetes affecting indigenous peoples worldwide: these rates are fully explained by colonialist policies of land dispossession that led to the intergenerational transmission of diabetes via biological—not genetic—processes.

Briefly, these processes include periods of starvation among women, who consequently give birth to insulin-resistant babies with low birth weights. These babies are later exposed to reservation lifestyles during critical periods of development. As adults, now insulin resistant or with poorly controlled diabetes themselves, they continue reproducing diabetes in turn as they give birth to insulin-resistant babies, *made* insulin resistant in the womb (Benyshek, Martin, and Johnston 2001) through no fault of either mothers or babies. This hypothesis of an intergenerational transmission of diabetes through nutritional manipulation and independent from genetic variations has been confirmed with animal models (Benyshek, Johnston, and Martin 2004; Benyshek et al. 2008), although such models are unable to account for the political element of this transmission in human populations.

I have also argued that the search for gene variants for diabetes is at best redundant and at worst conceptually confused, based on statistical acrobatics rather than on a proper understanding of living systems. Worse still, this search diverts attention and societal resources away from the root causes of diabetes, of diabetes complications, and of diabetes inequalities. Lastly, I have proposed, and I reiterate, that there is no need to disguise what remains unknown about the biology of diabetes, or of any other complex phenotype, with scientific-looking,

speculative narratives that substitute “thrifty genes” (Frayling and Hattersley 2001, 89) for actual, documented processes of accumulation by dispossession (Harvey 2004).⁶ Such obfuscation cannot but lead to the continuing reproduction and explosion of diabetes among populations as ethnically diverse as those of China, India, or the Micronesian island Nauru (Chaufan 2006; Chaufan 2007; Chaufan 2008). In fact, even as researchers claim that up to sixty-five gene variants relating to diabetes susceptibility have been identified, these findings have not added an iota to the *already existing tools to prevent, predict, diagnose, or treat diabetes*—even by these researchers’ standards, as detailed earlier.

What Health Equity Truly Requires

Study after study, of diabetes as well as many other ills affecting human populations, confirms the factors that significantly improve—i.e., make a difference to—health and disease rates, *regardless of genetic background*: proper housing, living wages, clean environments, good schools, nutritious food, equitable medical care, love, justice, absence of war, and so forth. They also confirm that when any of these things are missing, health deteriorates: our human genes, whatever their variants, simply do not thrive (Chaufan, Davis, and Constantino 2011; Raphael 2011; Phipps et al. 2006). Humans are remarkably equal in this respect, so there is nothing to be gained from scrutinizing further the “genetic architecture of common disorders” (Fuchsberger et al. 2016, 41) or from chasing their “missing heritability” (Manolio et al. 2009, 461).

I therefore conclude that scholars who reject the use of race in genomics are on the right track (Fullwiley 2008; Marks 2009; Keel 2016). Not only do they doubt that the so-called biological or, rather, genetic concept of race can ever bring more good than harm (I doubt this, too); they also see that the emperor is naked *even beyond the question of race*. The whole enterprise of identifying genetic variants that supposedly influence complex diseases and phenotypes, even with the goal (well-meaning or not) of better preventing, diagnosing, or treating diabetes, is hopelessly flawed.

Still, after years of seeing genes for diabetes come and go, I must end this commentary on a less than hopeful note: this inconvenient truth is unlikely to make the evening news any time soon.

NOTES

1. See Figure 2 in Hivert, Vassy, and Meigs 2014 for diabetes studies computing genetic risk scores.

2. Clearly, virtually all the differences between species are determined by differences in their genomes. Even within species, some variations can be caused entirely by genetic differences as they remain invariant under any sequence of environments. For example, this is the case of skin color when a baby is adopted away and raised by parents of different skin colors (Griffiths et al. 2005). Shape of nose is phenotypically comparable to skin color. I know of

nobody who has ever demonstrated otherwise, even if shape-of-nose gene variants have never been found—or even sought, as far as I know.

3. I refer to assumptions that correlations indicate causation: not of variances but of traits themselves.

4. For the purpose of this essay, I set aside the question of what exactly counts as diabetes, a diagnosis that has changed many times over the years since I graduated medical school in the 1980s.

5. Very Important People supported the genetic hypothesis of pellagra. One such person was Charles Davenport, prominent US biologist, Harvard graduate, director of Cold Spring Harbor Laboratory, and founder of the Eugenics Record Office.

6. The thrifty gene hypothesis was first proposed by James Neel (1962) to explain the disproportionate impact of type 2 diabetes among certain populations—usually indigenous peoples. Neels proposed that the genetic background that he and others assumed predisposes these populations to diabetes in modern times was the same as the genetic background—“thrifty gene”—that in the past had given these populations an evolutionary edge vis-à-vis the food scarcity that they must have experienced. Today there is no longer scarcity but abundance of food supplies, which works in tandem with sedentary lifestyles.

It is important to note that Neels and other supporters of the theory of a genetic predisposition to type 2 diabetes do not deny that environmental factors play a role: they very often acknowledge social conditions as well as other biological, nongenetic factors (e.g., developmental ones). Yet they still support a search for diabetes genes that will allegedly allow for better prevention, diagnosis, and treatment of the condition.

REFERENCES

- Benyshek, Daniel C., Carol S. Johnston, and John F. Martin. 2004. “Post-natal Diet Determines Insulin Resistance in Fetally Malnourished, Low Birthweight Rats (F1) but Diet Does Not Modify the Insulin Resistance of Their Offspring (F2).” *Life Sciences* 74 (May): 3033–3041.
- Benyshek, Daniel C., Carol S. Johnston, John F. Martin, and William D. Ross. 2008. “Insulin Sensitivity Is Normalized in the Third Generation (F3) Offspring of Developmentally Programmed Insulin Resistant (F2) Rats Fed an Energy-Restricted Diet.” *Nutrition and Metabolism* 5 (1): 26.
- Benyshek, Daniel C., John F. Martin, and Carol S. Johnston. 2001. “A Reconsideration of the Origins of the Type 2 Diabetes Epidemic among Native Americans and the Implications for Intervention Policy.” *Medical Anthropology* 20 (1): 25–64.
- Billings, Liana K., and Jose C. Florez. 2010. “The Genetics of Type 2 Diabetes: What Have We Learned from GWAS?” *Annals of the New York Academy of Sciences* 1212 (November): 59–77.
- Carmichael, Mary. 2010. “DNA, Denial and the Rise of ‘Environmental Determinism.’” Personal website, December 22. <http://marycarmichael.wordpress.com/2010/12/22/dna-denial-and-the-rise-of-environmental-determinism/>.
- Chaufan, Claudia. 2006. “Sugar Blues: Issues and Controversies Concerning the Type 2 Diabetes Epidemic.” PhD diss., University of California, Santa Cruz.
- . 2007. “How Much Can a Large Population Study on Genes, Environments, Their Interactions and Common Diseases Contribute to the Health of the American People?” *Social Science and Medicine* 65 (8): 1730–1741. <https://doi.org/10.1016/j.socscimed.2007.05.049>.
- . 2008. “What Does Justice Have to Do With It? A Bioethical and Sociological Perspective on the Diabetes Epidemic.” In *Bioethical Issues, Sociological Perspectives: Advances in Medical Sociology*, edited by Barbara Katz Rothman, Elizabeth M. Armstrong, and Rebecca Tiger, 269–300. New York: Elsevier.
- . 2009. “Unpacking the Heritability of Diabetes: The Problem of Attempting to Quantify the Relative Contributions of Nature and Nurture.” *DataCritica: International Journal of Critical Statistics* 2 (2): 23–38.
- Chaufan, Claudia, Meagan Davis, and Sophia Constantino. 2011. “The Twin Epidemics of Pov-

- erty and Diabetes: Understanding Diabetes Disparities in a Low-Income Latino and Immigrant Neighborhood." *Journal of Community Health* 36:1032–1043.
- Chaufan, Claudia, and Jay Joseph. 2013. "The 'Missing Heritability' of Common Disorders: Should Health Researchers Care?" *International Journal of Health Services* 43 (2): 281–303.
- Cheng, C.-Y., et al. 2012. "African Ancestry and Its Correlation to Type 2 Diabetes in African Americans: A Genetic Admixture Analysis in Three U.S. Population Cohorts." *PLOS ONE* 7 (3): e32840.
- Davenport, Charles B. 1916. *The Hereditary Factor in Pellagra*. Cold Spring Harbor, NY: Eugenics Record Office.
- Frayling, T. M., and A. T. Hattersley. 2001. "The Role of Genetic Susceptibility in the Association of Low Birth Weight with Type 2 Diabetes." *British Medical Bulletin* 60 (1): 89–101.
- Fuchsberger, Christian, et al. 2016. "The Genetic Architecture of Type 2 Diabetes." *Nature* 536 (7614): 41–47.
- Fullwiley, Donna. 2008. "The Biological Construction of Race: 'Admixture' Technology and the New Genetic Medicine." *Social Studies of Science* 38 (5): 695–735.
- Griffiths, Anthony J. F., Susan R. Wessler, Sean B. Carroll, and John Doebley. 2005. *An Introduction to Modern Genetic Analysis*. 8th ed. New York: W.H. Freeman and Company.
- Harvey, David. (2004). "The New Imperialism: Accumulation by Dispossession." *Socialist Register* 40:63–87.
- Hivert, M.-F., J. L. Vassy, and J. B. Meigs. 2014. "Susceptibility to Type 2 Diabetes Mellitus—from Genes to Prevention." *Nature Reviews, Endocrinology* 10 (4): 19–205.
- Joseph, Jay. 2000a. "Not in Their Genes: A Critical View of the Genetics of Attention-Deficit Hyperactivity Disorder." *Developmental Review* 20 (4): 539–567.
- . 2000b. "Potential Confounds in Psychiatric Genetic Research: The Case of Pellagra." *New Ideas in Psychology* 18 (1): 83–91.
- Keel, Terence. 2016. "Race Unmasked: Biology and Race in the Twentieth Century." *Journal of the History of Medicine and Allied Sciences* 71 (1): 107–109.
- Kevles, Daniel. 1995. *In the Name of Eugenics: Genetics and the Use of Human Heredity*. Cambridge, MA: Harvard University Press.
- Kroll, Luisa. 2016. "Forbes 2016 World's Billionaires: Meet the Richest People on the Planet." *Forbes*, March 1. <https://www.forbes.com/sites/luisakroll/2016/03/01/forbes-2016-worlds-billionaires-meet-the-richest-people-on-the-planet/#67c2ee4477dc>.
- Latham, Jonathan, and Allison Wilson. 2010. "The Great DNA Data Deficit: Are Genes for Disease a Mirage?" *Independent Science News*, December 8. <https://www.independent-science.org/health/the-great-dna-data-deficit/>.
- Lewontin, Richard C., Steven Rose, and Leon K. Kamin. 1982. "Bourgeois Ideology and the Origins of Biological Determinism." *Race and Class* 24 (1): 1–16.
- Lush, Jay L. 1949. "Heritability of Quantitative Characteristics in Farm Animals." *Hereditas* (Supplement): 356–375.
- Lyssenko, Valeriya, and Markku Laakso. 2013. "Genetic Screening for the Risk of Type 2 Diabetes: Worthless or Valuable?" *Diabetes Care* 36 (Supplement 2): S120–S126.
- Manolio, Teri A., et al. 2009. "Finding the Missing Heritability of Complex Diseases." *Nature* 461 (7265): 747–753.
- Marks, Jonathan. 2009. *Why I Am Not a Scientist: Anthropology and Modern Knowledge*. Berkeley: University of California Press.
- Meigs, J. B., et al. 2008. "Genotype Score in Addition to Common Risk Factors for Prediction of Type 2 Diabetes." *The New England Journal of Medicine* 359 (21): 2208–2219.
- National Institutes of Health. 2017. "Study Identifies African-Specific Genomic Variant Associated with Obesity." News release, March 13. <https://www.nih.gov/news-events/news-releases/study-identifies-african-specific-genomic-variant-associated-obesity>.
- Neel, James. 1962. "Diabetes Mellitus: A 'Thrifty' Genotype Rendered Detrimental by Progress?" *American Journal of Human Genetics* 14 (December): 353–362.
- Phipps, S. A., et al. 2006. "Poverty and the Extent of Child Obesity in Canada, Norway and the United States." *Obesity Reviews* 7 (1): 5–12.

- Polderman, Tinca J. C., et al. 2015. "Meta-Analysis of the Heritability of Human Traits Based on Fifty Years of Twin Studies." *Nature Genetics* 47 (May): 702–709.
- Raphael, Dennis. 2011. "Poverty in Childhood and Adverse Health Outcomes in Adulthood." *Maturitas* 69 (1): 22–26.
- Shönemann, Peter H. 1997. "On Models and Muddles of Heritability." *Genetica* 99 (2–3): 97–108.
- SIGMA Type 2 Diabetes Consortium. 2014. "Sequence Variants in SLC16A11 Are a Common Risk Factor for Type 2 Diabetes in Mexico." *Nature* 506 (7486): 97–101.
- Tandon, A., et al. 2015. "African Ancestry Analysis and Admixture Genetic Mapping for Proliferative Diabetic Retinopathy in African Americans." *Investigative Ophthalmology and Visual Science* 56 (6): 3999–4005.
- Wahlsten, Douglas. 2003. "Airbrushing Heritability." *Genes, Brain and Behavior* 2 (6): 327–329.
- World Health Organization. 2016. "Diabetes Fact Sheet." Accessed October 17, 2016. <http://www.who.int/mediacentre/factsheets/fs312/en/>.
- Zimmet, Paul. 1996. "The Global NIDDM Epidemic: Treating the Disease and Ignoring the Symptom." *Diabetologia* 39 (11): 1247–1248.