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1

Two Acids

In the 1960s, a Harvard psychologist named Robert Rosenthal and an elementary school principal named Lenore Jacobson teamed up to conduct an unusual (and arguably unethical) research study.[1] They hoped to complicate theories about why certain students succeed academically, which at the time tended to center on the psychological characteristics of a child, like personality and intellect. Rosenthal and Jacobson, however, wondered about the importance of how adults *perceive* a child, irrespective of the child's actual characteristics. This question proved a classic case of "the chicken or the egg"; the cause and effect seemed impossible to disentangle. Do students succeed because their teachers believe in them, or do teachers form their beliefs about students based on characteristics that lead to success? The duo devised an experiment to settle the matter: Using Jacobson's elementary school in South San Francisco as a laboratory, the pair rounded up 300 first- and second graders and administered the "Harvard Test of Inflected Acquisition." When the test results came back, Rosenthal and Jacobson gave each teacher a list of the students in their classroom who were "bloomers"—or children whose results indicated that they would likely excel academically in the coming years.

Unbeknownst to the teachers, the Harvard Test of Inflected Acquisition didn't actually exist! Instead, Rosenthal and Jacobson had administered a common IQ test. Either way, the test didn't

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matter: Rosenthal and Jacobson had randomly selected the so-called bloomers, meaning that they were no different than any of the other students. A year later, Rosenthal and Jacobson discovered that the bloomers had learned at faster rates than their peers. Teachers' beliefs about which students had inherent potential for academic success had become a self-fulfilling prophecy. While Rosenthal and Jacobson's experiment is the subject of contentious scientific debate (their sample size was small, and they have been accused of cherry-picking the statistical results), recent studies using more rigorous methods confirm that teachers' expectations really do impact their students, even if the effects may be more modest than Rosenthal and Jacobson originally argued.[2]

Rosenthal and Jacobson's experiment pushed researchers to break away from an outdated paradigm that focused only on a child's mind, shifting the perspective to also consider the child as a social object molded by external influences. The study demonstrated that ideas and perceptions, even unfounded ones, have the power to shape a person's life trajectory. Like Rosenthal and Jacobson's experiment, this book illustrates the ways in which ideas and perceptions, however untrue, shape people's understandings of themselves and others. Our goal is to expand the way in which people think about genes, broadly conceived. In particular, *What We Inherit* outlines two intertwined inheritance processes: DNA itself, and the myths about genes that also span generations.

You may think of genes in terms of DNA or deoxyribonucleic acid: a molecule that sits in the center of a cell and acts as a kind of biological instruction book. DNA plays a key role in the evolution and adaptation of a wide range of life-forms, from fungi to palm trees to whales—and, of course, human beings. Furthermore, genes function as an iconic social object with a powerful grip on the human collective imagination.[3] The literal acid within cells gets passed down biologically from parent to child; the conceptual acid, or stories and myths about genes and how they affect human life, gets passed down culturally from a eugenic past.

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For better or for worse, the train has left the station; novel genomic technologies and discoveries have already begun to accumulate and disseminate throughout a range of life domains—from academic research to the direct-to-consumer genetic testing industry to the fertility clinic. The growing importance of DNA demands new frameworks for understanding human genetic differences and for considering the regulation of genomic tools. *What We Inherit* argues that a full account of the power and influence of genes must consider the dual inheritance processes of DNA and genetic myths. To ensure that the benefits of the unfolding genomic era are maximized and its risks minimized, researchers and policymakers need to account for historical and social context when deciding what research to conduct and how to discuss it. Careful attention must be paid to social inequalities, past and present, when considering how new genomic technologies may be used in healthcare, schools, industry, and society writ large. This book stems from a shared motivation to combine expertise in two acids that society and its members inherit—Daphne, the myth, and Sam, the molecule.

Better understanding the acids that human beings inherit requires rewinding to the birth of the modern field of human genomics. Its genesis at the turn of the twenty-first century resulted from a high-profile and dramatic clash, the likes of which the often-mundane world of scientific research rarely sees. The field's contentious birth would foreshadow the many controversies surrounding DNA to come in the following decades.

The Human Genome Project launched in 1990 with an ambitious and unprecedented scientific goal: to map out the entire DNA sequence of the human species, from beginning to end. Led by Francis Collins, the director of the National Human Genome Research Institute (the primary government-funded genomics institute in the United States), the Human Genome Project

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intended to make its discoveries widely and freely available to researchers around the world. To the frustration of Collins and his team, Craig Venter, a researcher-turned-entrepreneur who founded a company called Celera Genomics, had a different plan; he hoped to privately sequence the genome and sell the ensuing scientific discoveries.

Each group raced to finish their sequencing first, and the competition quickly turned nasty. Venter publicly criticized the Human Genome Project, calling it a waste of public resources. One of the Human Genome Project's leading scientists shot back, calling Celera's commercialization of the genome a "con-job."[4] Eventually, the White House stepped in and initiated peace talks between the two groups of researchers. It took time (and pizza), but ultimately Venter and Collins were able to resolve their dispute. In 2000, when researchers finished a first draft of the human genome, Collins and Venter both flanked President Bill Clinton as he announced: "Today we are learning the language in which God created life . . . With this profound new knowledge, humankind is on the verge of gaining immense new power to heal."[5]

The Human Genome Project wrapped up in 2003, two years ahead of schedule. Some say the rapid technological changes following the completion of the project amount to a "DNA revolution." Over the last two decades, the cost of DNA sequencing has dramatically decreased. It took thirteen years and cost nearly $3 *billion* to sequence the very first human genome; today, it costs only a few hundred dollars to sequence a genome in less than 24 hours.

What exactly is DNA—the molecule that the Human Genome Project thrust into view? Think of a person's DNA as a figurative "book" of biological instructions written using an alphabet of four letters: adenine (*A*), cytosine (*C*), guanine (*G*), and thymine (*T*). Each letter, or nucleobase, is paired with a complementary letter to form base pairs. *A* pairs with *T*, and *C* pairs with *G*. Each human has roughly three billion base pairs comprising their DNA sequence, which are organized into forty-six "chapters" (or *chromosomes*). The ordering of *A*s, *C*s, *T*s, and *G*s is, for the most part, the

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same for all humans. That is, every human being has a very similar book of DNA; nonetheless, each person's DNA sequence is one of a kind, almost like a fingerprint. (The exception to this rule, of course, is identical twins.) Each person inherits a unique DNA sequence from their parents, and that sequence remains unchanged throughout a person's entire lifetime. When one person has a certain letter (or set of letters) at a particular location in the genome (say a *T*), whereas another person has a different letter at that same spot (say a *G*), those two people have different *DNA variants*.

In the decades since the completion of the Human Genome Project, scientific advances in collecting and analyzing genomic data have produced a torrent of discoveries linking DNA to a wide range of human traits. For thousands of years, the ability to understand the effects of DNA hinged on observing it indirectly via familial relatedness (for example, by comparing identical and fraternal twins, or siblings and cousins). Now, it is possible to observe each person's unique ordering of *A*s, *C*s, *T*s, and *G*s at the molecular level. Can an individual's DNA data be used to make predictions about their life outcomes—for instance, which diseases they will come to develop or how their personality will change as they age?

If you took high school biology in the United States, you may remember learning about an Austrian monk named Gregor Mendel who conducted experiments with peas. Mendel cross-pollinated different kinds of pea plants (crossing tall plants with short plants and yellow plants with green plants, for example) to try to understand how traits get passed down between generations. He is credited with discovering many of the basic principles of genetic inheritance—namely, that organisms pass portions of their DNA to their offspring (although the term "DNA" hadn't yet been coined in Mendel's day). Mendel argued that, for every trait, offspring inherited one DNA variant from each parent. Some of these variants, he concluded via experimentation, are dominant, while others are recessive.

Until the DNA revolution, most researchers believed that just a single or a few DNA variants impacted a given trait; traits

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influenced by a single region of the genome known as *monogenic*. Huntington's disease, for example, is a monogenic trait caused by a DNA variant of the HTT gene on chromosome 4. Similarly, sickle cell anemia is a monogenic trait caused by a DNA variant of the β-globin gene on chromosome 11. High school classes in the United States rely on Mendel's experiments and monogenic traits to introduce students to genetics (remember Punnett squares?). In an abstract classroom environment, DNA may first seem to operate in a clear-cut and straightforward manner; however, the technological advancements brought on by the completion of the Human Genome Project paint a markedly different picture.

A key discovery of the genomic era is that most human characteristics are *not* monogenic; instead, they are *polygenic*, or complex. You can throw "dominant" and "recessive" out the window; polygenic traits, like height, are influenced by countless DNA variants dispersed widely across the genome. There is no "height gene"—a lone variant responsible for genetic influences on how tall a person grows to be. Instead, thousands (or even *millions*) of DNA variants are correlated with a person's height.[6] For a polygenic trait, any given DNA variant contributes just a tiny fraction of the total genetic influence.

As a method of summarizing these myriad DNA variants, researchers have developed a new genomic tool known as a *polygenic score*. (Note that polygenic scores are also referred to as polygenic indexes,[7] polygenic risk scores, and genetic risk scores.) Polygenic scores use a person's DNA to make predictions for a wide range of outcomes—for example, how tall they will likely grow, their chances of developing skin cancer, and what level of education they will reach. While there are very few traits that are "genetic" in a monogenic sense, the vast majority of traits are "genetic" in a polygenic sense. The realization that most traits are associated with many DNA variants rather than one or just a few has transformed the focus of human genomics from specific inherited diseases to almost all types of individual difference.

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As the costs of collecting and analyzing DNA continue to drop and genomic databases grow in size, the predictiveness of polygenic scores also continues to improve. Importantly, the predictions offered by polygenic scores are probabilistic, not deterministic. To visualize the probabilistic relationship between a polygenic score and an outcome (like educational attainment), researchers use scatterplots; figure 1 displays three. Each small grey dot in figure 1 represents a single person, and all three plots contain data on the same 874 American adults. These 874 Americans were all born around 1980 and are participants in the National Longitudinal Study of Adolescent to Adult Health, an ongoing biosocial survey used by researchers studying health and human behavior.[8] The variable on the vertical axis is each person's educational attainment—the number of years of formal schooling each individual completed. For instance, those who dropped out of high school without graduating have fewer years of schooling than those who got their high school diploma. Those who entered the workforce after high school graduation, in turn, have fewer years of schooling than those who continued on to university. The variables on the horizontal axes are three different polygenic scores. Each person's polygenic score is generated by statistically combining their unique string of *A*s, *C*s, *G*s, and *T*s to produce a single number, ranging from about -3 to 3, that will correlate with their eventual educational attainment.

The punchline of figure 1 is that the predictive accuracy of polygenic scores has increased significantly over the past two decades. The DNA sequences of the 874 Americans represented in figure 1 are identical across the three panels (and so is their educational attainment). What changes across the panels is the precise formula used to transform someone's DNA sequence into a polygenic score. A person's polygenic score for a given trait is essentially an enormous weighted average of their genome, where the weights represent researchers' best guess of which DNA variants are associated with increases or decreases in the trait in question (and by how much). In 2004, at the start of the genomic era, it was virtually

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impossible to make a prediction about someone's educational attainment using DNA, and when the first major genomic study of educational attainment was published in 2013, polygenic prediction was just barely possible.[9] Now, almost a decade later, the formula underlying the educational attainment polygenic score has substantially improved. The latest version of the score is just as predictive of eventual years of schooling as all the textbook variables used by social scientists: a child's family income, their IQ test scores,[10] and their parents' education levels. (In nationally representative data sources, this polygenic score explains about one-sixth of the variation in years of schooling between individuals.)[11]

Figure 1 also highlights the quite limited ability of a polygenic score (or any variable, for that matter) to predict a specific person's educational attainment. Notice the often-large vertical gap between the dark black line, which represents a person's predicted education (given their polygenic score), and the gray dots, which represent a person's realized education. Even the best predictors leave the vast majority of variation unexplained. Plenty of people with high polygenic scores for educational attainment do not graduate high school, and plenty of people with low polygenic scores for educational attainment end up graduating from college.

Still, perhaps because of internalized genetic myths and biases, people can often erroneously think that the information gleaned from a polygenic score is as definitive as learning they are a carrier for Huntington's disease. They may also believe that polygenic scores have managed to somehow disentangle the effects of a person's DNA from their environmental context. Concerns over what exactly a polygenic score captures and how they should be used continue to mount. This book shows just how complicated these so-called complex traits (and the polygenic scores that try to predict them) really are and discusses how to navigate these complexities.

Polygenic scores are making swift inroads into society, leaving some excited and others on edge. Hoping to build a world in which more people experience better health and social outcomes,

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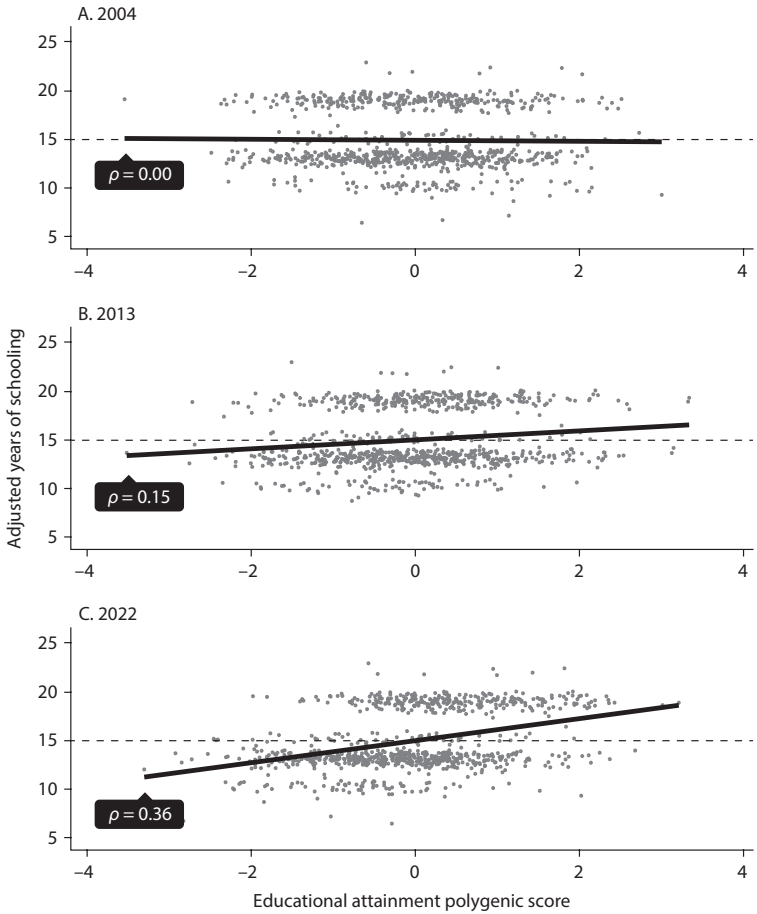

FIGURE 1. Polygenic Prediction of Educational Attainment: 2004 through 2023. Each panel of the figure displays a scatterplot containing the same 874 individuals of European ancestries from the National Longitudinal Study of Adolescent to Adult Health. Years of schooling is measured using the International Standard Classification of Education (ISCED) 1997 classification system and is collected at Wave IV of the study. Years of schooling is statistically adjusted for sex, age, and ten genomic principal components.[12] The same years of schooling variable is used in all three panels, but each panel plots a different polygenic score variable. Panel A utilizes a randomly generated standard normal variable, representing polygenic prediction during the candidate gene era. Panel B utilizes a polygenic score generated from the results of the very first genome-wide association study (GWAS) of educational attainment ($N = 126{,}559$ individuals[13]). Panel C utilizes a polygenic score from the most recent GWAS of educational attainment ($N = 3{,}037{,}499$ individuals[14]).

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proponents of polygenic scores believe these new tools have the potential to advance collective understanding of how well certain interventions or treatments work (and for whom). They see polygenic scores contributing to the ongoing projects of mapping the complexities of being human and improving people's well-being. Still, the United States has a long and fraught history of connecting DNA to human behavior. For this reason, polygenic scores for social and behavioral traits like educational attainment carry a particular historical baggage and set of social risks; they are sometimes viewed differently from, say, polygenic scores for diseases like breast cancer.[15] While many of the arguments made in this book apply to a broad range of genetics research, an understanding and acknowledgment of this ugly history inspires a particular focus on *social genomics*, a research field that seeks to connect a person's DNA sequence to social and behavioral traits, like a person's sexual orientation or occupation. Such research is at the highest risk of perpetuating the damaging types of myths described in this book.[16]

Rapid increases in polygenic prediction, coupled with the growing number of traits for which polygenic scores are available, raise a range of questions: What sort of information do polygenic scores provide? What are their risks and benefits? How should new DNA-based tools be regulated in society? As debates over these questions rage on, polygenic scores are increasingly being used by companies and institutions with surprisingly little oversight. Society needs to brace for a future where polygenic prediction is readily available. Now is the time to start having tough conversations about how to best navigate society's bumpy landing into this new genomic era.

Genes—whether through the literal acid of DNA or the conceptual acid of genetic myths—are sources of polarization and pride. They comprise what the physician and writer Siddhartha Mukherjee calls "one of the most powerful and dangerous ideas in the history

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of science."[17] DNA provides the fundamental means by which all humans differ from one another. Layered over variation in people's individual DNA profiles are the social myths created, narrated, and passed down through generations. These myths frame a collective understanding of how genes function and what they say about who people are.

This book pushes forward a much-needed discussion about how society should and should not use genetic information like polygenic scores. Today, people with a few hundred dollars to spare can spit into a tube or swab their cheeks using an at-home test; they'll receive information on their polygenic scores for traits that range from Type 2 diabetes and prostate cancer to math ability and intelligence. Increasing access to these new genomic technologies presents both opportunities and challenges, many of which inflect age-old debates about genetic difference. Real-world applications of polygenic scores are growing, but regulation lags behind—in part because of how sensitive and charged conversations about DNA can be.

At the same time, influential genetic myths about DNA are shaping people's perceptions of polygenic scores. These myths threaten to grow in power and influence due to the ever-increasing presence of genomic research and genomic tools. The two specific genetic myths unpacked in this book—the *Destiny Myth* and the *Race Myth*—are both socially inherited from historical eras marred by the powerful legacies of eugenics and scientific racism.[18] These myths have been passed down through books and laws, folklore and oral tradition. This book aims to pull them apart, bit by bit, until they are exposed for what they truly are: fictions that serve to distract from real issues like racial and class-based social inequalities,[19] the unsavory realities of an unequal world.

What We Inherit consists of three parts, each of which includes three chapters. Part one (chapters 1–3) provides background and context on the history of genomic research and genetic myths. Chapter 2 helps to put the infamous "nature vs. nurture" debate to bed by explaining what it means for DNA to influence a person's

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health, behavior, or life outcomes. The environment and DNA interact in complex ways, and phrases like "genetic effects" or the "effects of DNA" can mistakenly lead to overstatements of the role of DNA. The chapter concludes by debunking the Destiny Myth: the flawed idea that **the effects of DNA are immutable and inevitable**. (Note that causal relationships between DNA and outcomes are often referred to as "genetic effects," but this book uses the term the "effects of DNA" because this terminology choice helps to distinguish them from the effects of genetic myths.)

Chapter 3 disentangles race and ancestry. Race is a sociopolitical construct designed to benefit some and harm others; it differs from the ancestral information captured by the large Family Tree of humanity. In the twenty-first century, direct-to-consumer genetic testing companies can provide information about where a person's ancestors may have originated, but that information needs interpretation through an informed lens (and with a cautionary grain of salt). In disentangling race and ancestry, the chapter reveals the lies behind the Race Myth: the false belief that **DNA differences divide humans into discrete and biologically distinct racial groups**.

In a perfect world, half of the published copies would've listed Daphne first on the cover, and the other half would've listed Sam first. (In our actual world, we flipped a coin and Sam won.) However, while the book largely takes on one, united voice, the authors do not agree on everything. Their disagreements come through particularly in part two (chapters 4–6), which focuses on debates regarding genetic myths and genomic research. Chapter 4 explores the historical impacts of genetic myths on society, as well as the persistence of these myths. Chapter 5 covers debates about the promises and pitfalls of social genomics and discusses who gets to weigh the risks and benefits of such research. This chapter also examines how genetic myths can repurpose modern-day genomic research. Chapter 6 explores disagreements about whether DNA is relevant for understanding and ameliorating social inequality; at the heart of this debate are different understandings and

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definitions of social inequality. This chapter provides a conceptual basis for thinking about the regulation of polygenic scores (covered in part 3). As noted in the preface, in order to clarify and illustrate our disagreements, each chapter in part 2 begins and ends with dialogues between Sam and Daphne: for instance, on how genetic myths impact American society, the risks and benefits of social genomic research, and whether DNA "matters" when considering social inequality.

Part 3 provides policy recommendations for navigating a modern world rife with polygenic scores, focusing on three specific applications of the DNA-based tool. Chapter 7 considers the regulation of polygenic embryo selection, a technology that allows prospective parents to choose certain genetic characteristics of their future children. Utilizing existing polygenic scores in the fertility clinic can produce increases in height of 2 ½ inches, but the technology is expensive and—at present—not very effective for most people. Chapter 8 discusses the regulation of direct-to-consumer genetic testing and polygenic-informed screening programs. As online genetic tests proliferate and as polygenic scores are beginning to stratify care in hospitals and clinics, new frameworks must guide the ethical and responsible use of genomic information. In both chapters, the goal of the policy recommendations is to prevent these polygenic scores applications from, at the very least, widening preexisting structural inequalities. Chapter 9 presents concluding remarks and key takeaways, offering next steps for researchers, policymakers, and members of the public.

Now more than ever, reaching across the aisle and having conversations with those you disagree with may feel fraught and unproductive. This book aims to convince you that many such debates are necessary—and even urgent. Society faces a welter of big, challenging questions: Is it possible to balance social equality and efficiency in the face of rapid technological changes? What might it look like to conduct scientific research in a way that delivers vital discoveries without repeating past mistakes? To what extent is it appropriate to shape the biology of future generations of

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humans? Creating a world in which genomic data is used in a socially responsible way requires that these questions be answered.

As you will see, however, there are plenty of areas where the authors disagree with one another. Writing this book reflects a shared commitment to the belief that, even as scientific advances increase researchers' abilities to zoom in and view processes at the molecular level, efforts to also seek macro-level explanations of human processes cannot fall by the wayside. Working together, the authors slowly begin to digest the rapid development of new genomic technological changes and the age-old genetic myths that accompany them; along the way, this book aims to humanize the people affected by both. Navigating new genomic technologies while putting to rest old genetic myths is not going to be easy, but the only path forward is to learn how to listen and talk *with* rather than *past* each other.

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