The Genetic Lottery

Why DNA Matters for Social Equality

Kathryn Paige Harden
Narrated by Katherine Fenton

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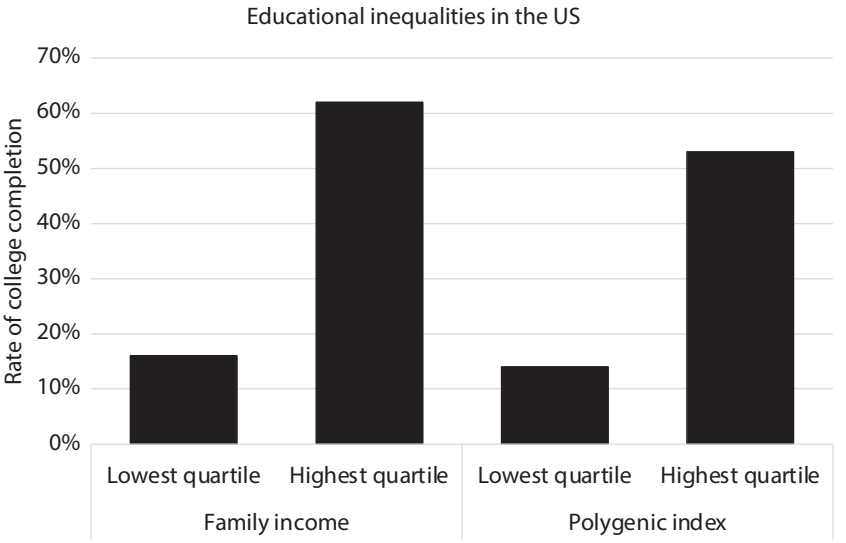


FIGURE 1.1. Inequalities in rates of college completion in the US based on differences in family income versus differences in measured genetics. Data on college completion by income drawn from Margaret W. Cahalan et al., *Indicators of Higher Education Equity in the United States: 2020 Historical Trend Report* (Washington, DC: The Pell Institute for the Study of Opportunity in Higher Education, Council for Opportunity in Education (COE), and Alliance for Higher Education and Democracy of the University of Pennsylvania (PennAHEAD), 2020), https://eric.ed.gov/?id=ED606010. Data on college completion by polygenic index from James J. Lee et al., "Gene Discovery and Polygenic Prediction from a Genome-Wide Association Study of Educational Attainment in 1.1 Million Individuals," *Nature Genetics* 50, no. 8 (August 2018): 1112–21, https://doi.org/10.1038/s41588-018-0147-3; additional analyses courtesy of Robbee Wedow. Polygenic index analyses include only individuals who share genetic ancestry characteristic of people whose recent ancestors all resided in Europe; in the US, these people are very likely to be racially identified as White. The distinction between race and genetic ancestry will be described in more detail in chapter 4.

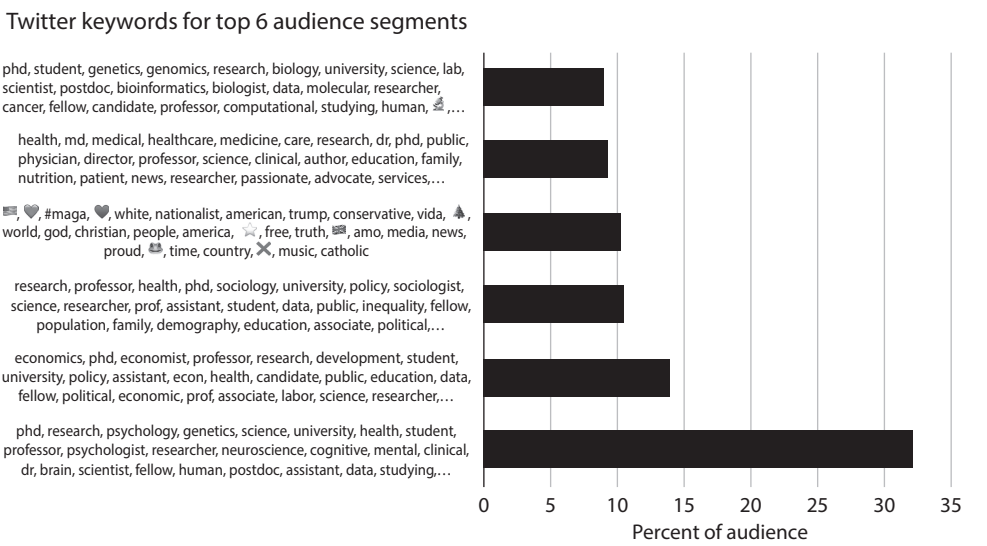


FIGURE 1.2. Top 6 largest social media audiences for scientific paper on genetics and noncognitive skills. Audience analysis methods reported in Jedidiah Carlson and Kelley Harris, "Quantifying and Contextualizing the Impact of bioRxiv Preprints through Automated Social Media Audience Segmentation," *PLOS Biology* 18, no. 9 (September 22, 2020): e3000860, https://doi.org/10.1371/journal.pbio.3000860. Audiences are presented for preprint of Perline Demange et al., "Investigating the Genetic Architecture of Noncognitive Skills Using GWAS-by-Subtraction," *Nature Genetics* 53, no. 1 (January 2021): 35–44, https://doi.org/10.1038/s41588-020-00754-2.

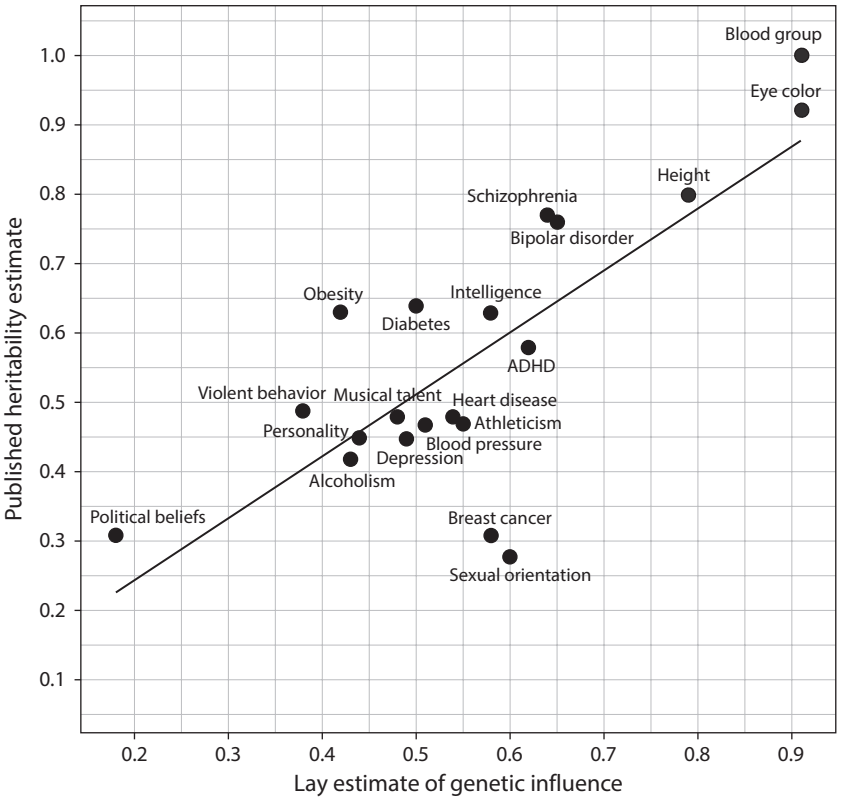


FIGURE 2.1. People's estimates of how much genetic factors contribute to human differences (horizontal axis) versus scientific estimates of heritability from twin studies (vertical axis). The correspondence between lay estimates and scientific estimates is r = .77. Figure reprinted by permission of Springer Nature from Emily A. Willoughby et al., "Free Will, Determinism, and Intuitive Judgments about the Heritability of Behavior," *Behavior Genetics* 49, no. 2 (March 2019): 136–53, https://doi.org/10.1007/s10519-018-9931-1.

FIGURE 2.2. A Galton board, showing how a normal distribution results from the accumulation of many random events. Photo by Mark Hebner.

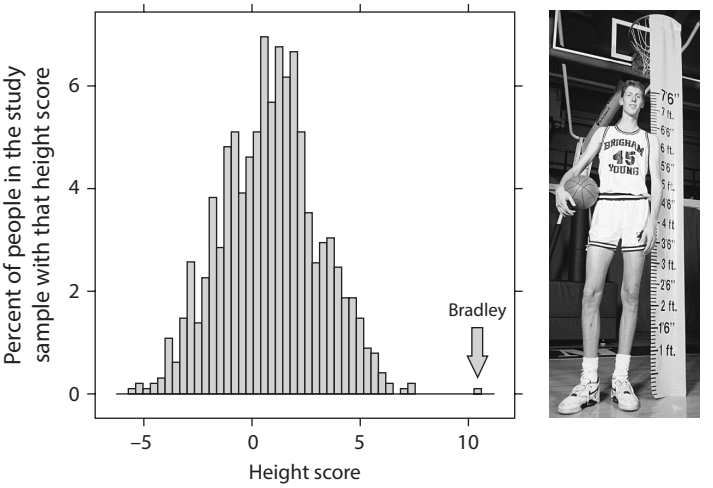


FIGURE 2.3. Height-increasing genetic variants in an individual of extreme height. On the right is a photo of Shawn Bradley next to a ruler showing that he is 7′6″ tall. On the left is the distribution of "genetic scores" (i.e., polygenic indices) constructed from 2,910 genetic variants associated with human height. Mr. Bradley's score was 10.32, whereas the average score in the sample of people being studied was 0.98, with a standard deviation of 2.22. Mr. Bradley's score was 4.2 standard deviations above the mean. Figure adapted from Corinne E. Sexton et al., "Common DNA Variants Accurately Rank an Individual of Extreme Height," *International Journal of Genomics* 2018 (September 4, 2018): 5121540, https://doi.org/10.1155/2018/5121540.

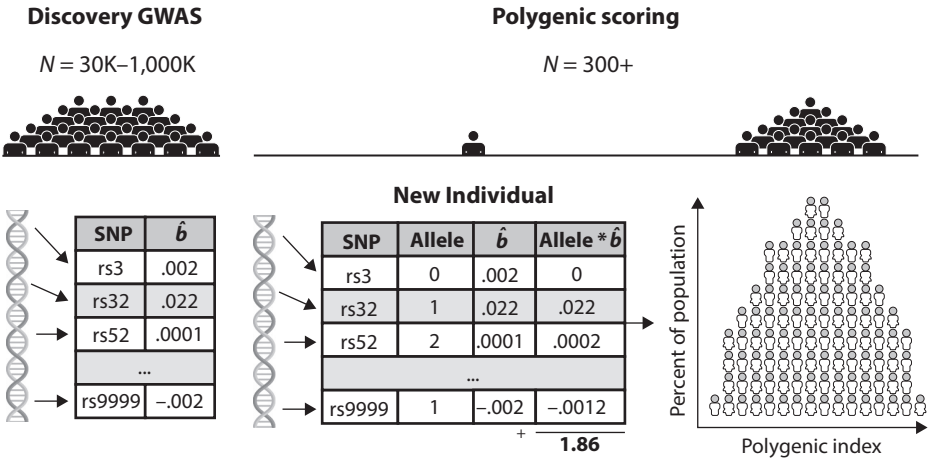


FIGURE 3.1. Creating a polygenic index. Figure reproduced from Daniel W. Belsky and K. Paige Harden, "Phenotypic Annotation: Using Polygenic Scores to Translate Discoveries from Genome-Wide Association Studies from the Top Down," *Current Directions in Psychological Science* 28, no. 1 (February 2019): 82–90, https://doi.org/10.1177/0963721418807729. Correlations between individual SNPs and a phenotype are estimated in a "Discovery GWAS" with a large sample size. Many GWAS have samples that exceed millions of people. Then, a new person's DNA is measured. The number of minor alleles (0, 1, or 2) in this individual's genome is counted for each SNP, and this number is weighted by the GWAS estimate of the correlation between the SNP and the phenotype, yielding a polygenic index. This polygenic index will be normally distributed: most people will have an average polygenic index, but a few people will have very low or very high scores. Reprinted by permission of SAGE Publications, Inc.

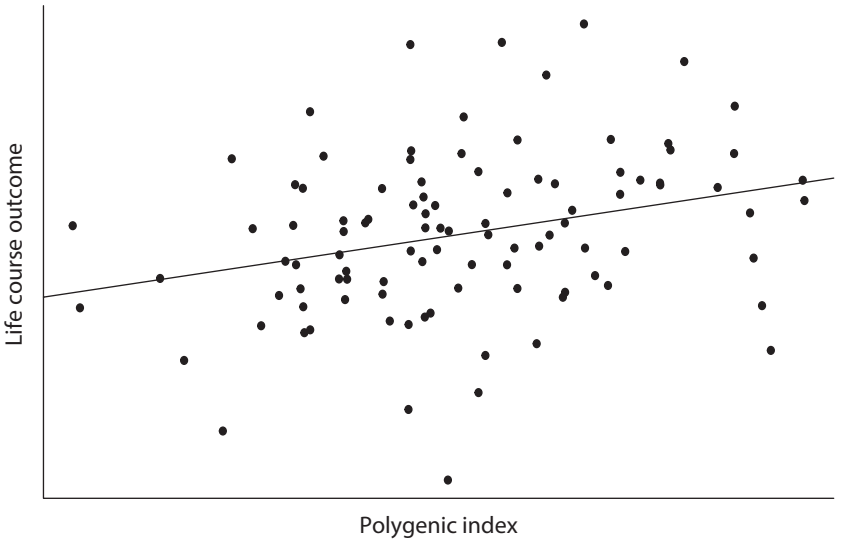


FIGURE 3.2. Hypothetical polygenic index that captures 10% of the variance in a life course outcome. Polygenic index on the horizontal axis; hypothetical life outcome, such as educational attainment, on the vertical axis. Each dot represents an individual person. For each value of the polygenic index, there is considerable variability in people's life outcomes.

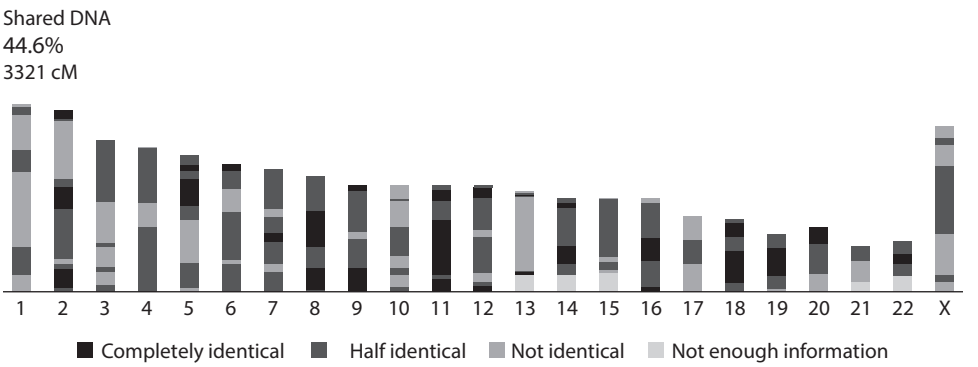


FIGURE 6.1. Identity-by-descent sharing of segments of 23 chromosomes between a pair of full siblings. Image from author's 23andMe® profile. The author and her brother share segments of DNA that have a total length of 3321 centimorgans (cMs), which is 44.6% of the author's genome.

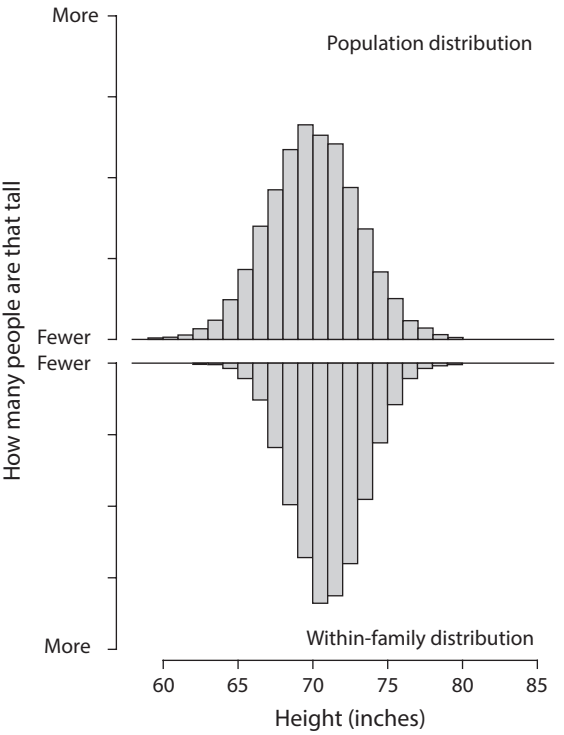


FIGURE 6.2. Expected distribution of heights in the general population (top) versus within potential offspring of a single pair of parents (bottom). Population distribution is based on mean of 70 inches with a standard deviation of 3 inches. Within-family distribution, i.e., the distribution of heights among all possible offspring of a single pair of parents, based on heritability of 0.8. Example and calculations adapted from Peter M. Visscher, William G. Hill, and Naomi R. Wray, "Heritability in the Genomics Era—Concepts and Misconceptions," *Nature Reviews Genetics* 9, no. 4 (April 2008): 255–66, https://doi.org/10.1038/nrg2322.

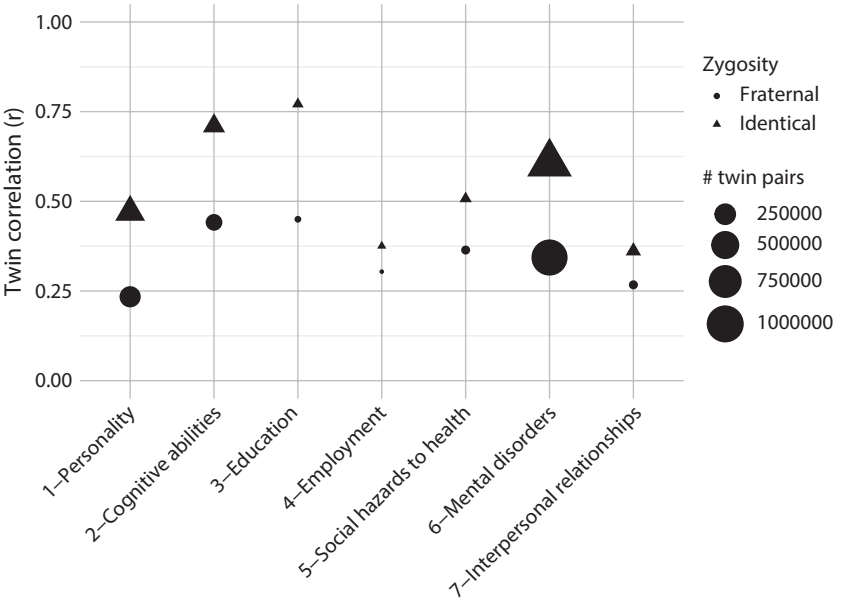


FIGURE 6.3. Identical and fraternal twin correlations for seven domains of inequality. Author's analysis of data from Tinca J. C. Polderman et al., "Meta-Analysis of the Heritability of Human Traits Based on Fifty Years of Twin Studies," *Nature Genetics* 47, no. 7 (July 2015): 702–9, https://doi.org/10.1038/ng.3285.

The case of the missing heritability

Heritability estimates from measured DNA studies might be too low

- DNA studies don't have enough people to reliably estimate the small effects of genes?
- DNA studies don't measure every genetic variant, and unmeasured variants might have big(ger) effects?

Heritability estimates from twin studies might be too high

- Genes and environments are correlated in ways that are difficult to measure and account for?
- Identical twins might be treated more similarly than fraternal twins?

FIGURE 6.4. The case of the missing heritability. Image reproduced by permission of Springer Nature from Brendan Maher, "Personal Genomes: The Case of the Missing Heritability," *Nature* 456, no. 7218 (November 1, 2008): 18–21, https://doi.org/10.1038/456018a.

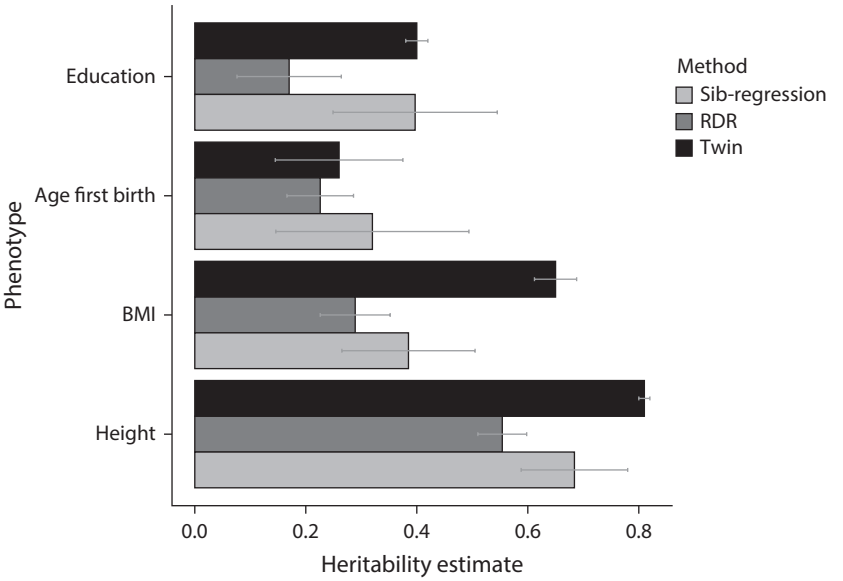


FIGURE 6.5. Heritability estimates for four human phenotypes from three different methods. "Education" = educational attainment (years of formal schooling). "Age first birth" = women's age at first childbirth. "BMI" = body mass index. "Height" = height in adulthood. "Twin" method estimates heritability by comparing similarity of monozygotic twins reared together to similarity of dizygotic twins reared together. "Sib-regression" method estimates heritability by leveraging random variation among sibling pairs in extent of identity-by-descent sharing. "RDR" (relatedness disequilibrium regression) method extends the sib-regression method to other pairs of relatives, where the relatedness of the pair is conditioned on the relatedness of their parents. Error bars represent standard errors. All heritability estimates drawn from Alexander I. Young et al., "Relatedness Disequilibrium Regression Estimates Heritability without Environmental Bias," *Nature Genetics* 50, no. 9 (September 2018): 1304–10, https://doi.org/10.1038/s41588-018-0178-9, except for twin estimate of heritability for educational attainment, which is drawn from Amelia R. Branigan, Kenneth J. McCallum, and Jeremy Freese, "Variation in the Heritability of Educational Attainment: An International Meta-Analysis," *Social Forces* 92, no. 1 (2013): 109–140; and twin estimate of heritability for age at first birth in women, which is drawn from Felix C. Tropf et al., "Genetic Influence on Age at First Birth of Female Twins Born in the UK, 1919–68," *Population Studies* 69, no. 2 (May 4, 2015): 129–45, https://doi.org/10.1080/00324728.2015.1056823.

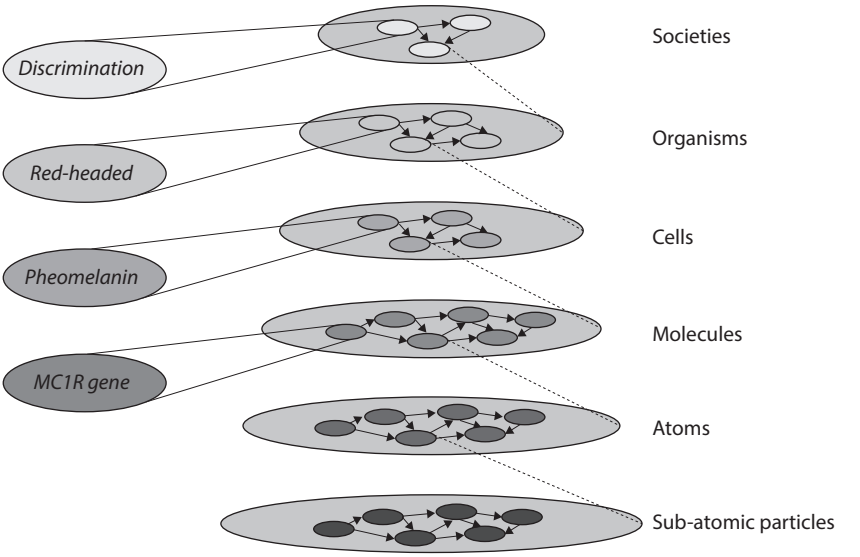


FIGURE 7.1. Levels of scientific analysis. Figure incorporates ideas from Carl F. Craver, *Explaining the Brain: Mechanisms and the Mosaic Unity of Neuroscience* (Oxford: Oxford University Press, 2007); Paul Oppenheim and Hilary Putnam, "Unity of Science as a Working Hypothesis," 1958, http://conservancy.umn.edu/handle/11299/184622; and Christopher Jencks et al., *Inequality: A Reassessment of the Effect of Family and Schooling in America* (New York: Basic Books, 1972).

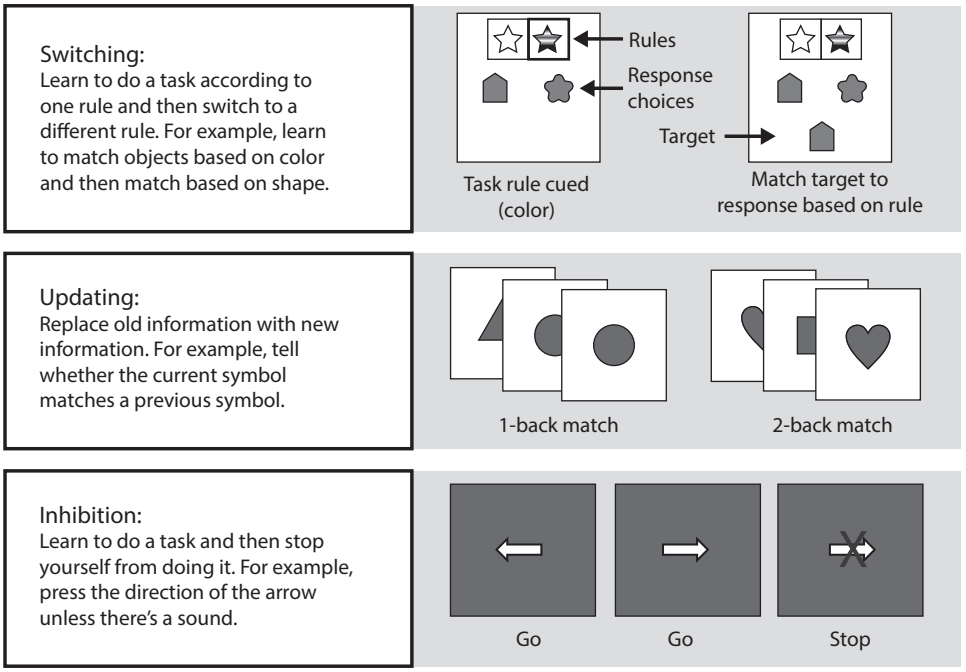


FIGURE 7.2. Examples of tests of executive functions in children. Described in Laura E. Engelhardt et al., "Genes Unite Executive Functions in Childhood," *Psychological Science* 26, no. 8 (August 1, 2015): 1151–63, https://doi.org/10.1177/0956797615577209.

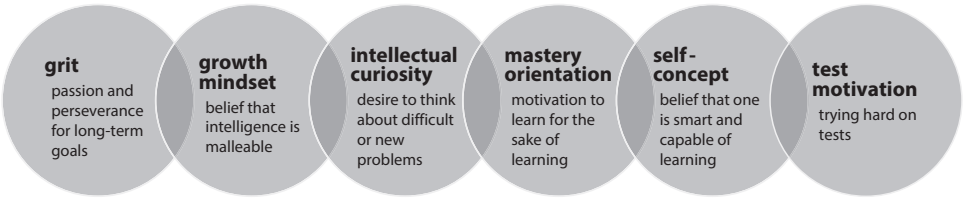


FIGURE 7.3. Different types of non-cognitive skills. Described in Elliot M. Tucker-Drob et al., "Genetically Mediated Associations between Measures of Childhood Character and Academic Achievement," *Journal of Personality and Social Psychology* 111, no. 5 (2016): 790–815, https://doi.org/10.1037/pspp0000098.

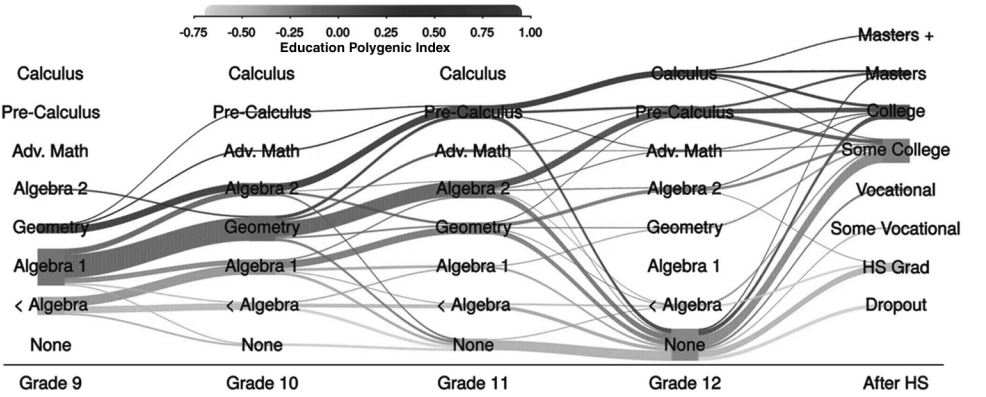


FIGURE 7.4. Flow of students through the high school math curriculum by educational attainment polygenic index. Width of the line represents number of students enrolled in each math course in each year of high school (secondary school). Darkness of the line represents the average education polygenic index of students enrolled in that course. Values of the polygenic index are in standard deviation units. Data are from European-ancestry students from the National Longitudinal Study of Adolescent Health who were enrolled in US high schools in the mid-1990s. Reproduced from K. Paige Harden et al., "Genetic Associations with Mathematics Tracking and Persistence in Secondary School," *Npj Science of Learning* 5 (February 5, 2020): 1–8, https://doi.org/10.1038/s41539-020-0060-2.

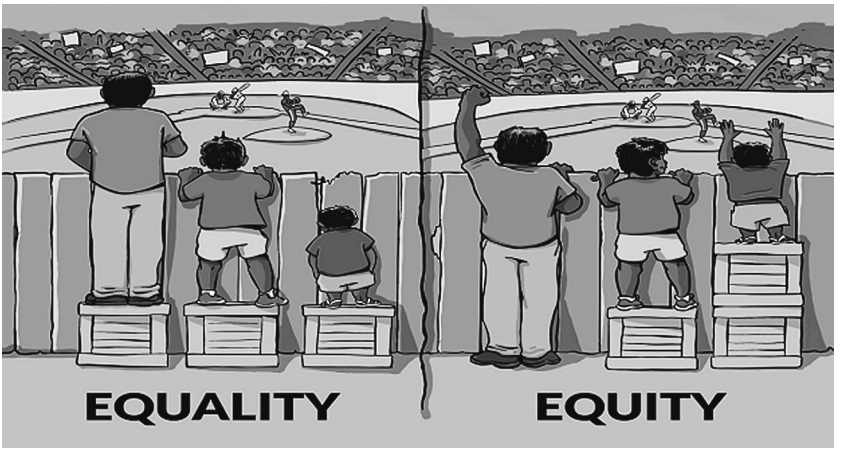


FIGURE 8.1. Equality versus equity. Image from Interaction Institute for Social Change. Artist: Angus Maguire.

FIGURE 8.2. Pre-kindergarten classroom sign about fairness. Photo by author.

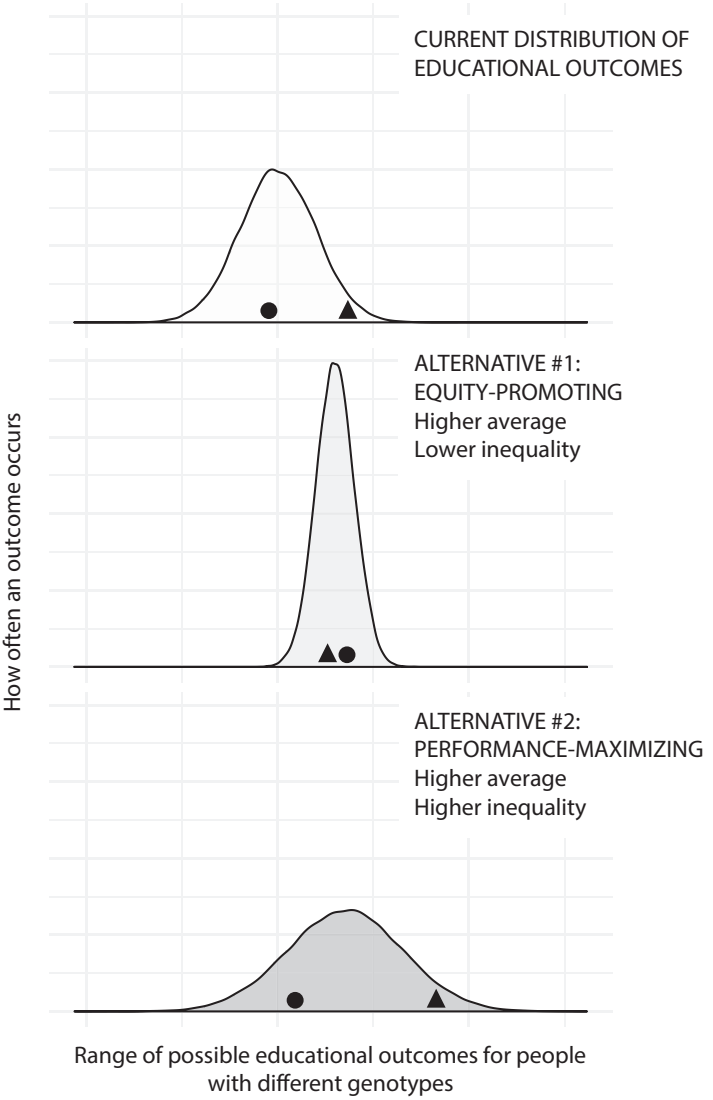


FIGURE 8.3. Distribution of educational outcomes for people with different genotypes in alternative environments. The circle and triangle represent two hypothetical individuals with two different genotypes. Relative to the current situation, the environment that is equity-promoting (alternative #1) improves the educational outcome of the individual represented by the circle, but makes little difference for the individual represented by the triangle, reducing inequality of outcome. In contrast, the environment that is performance-maximizing (alternative #2) improves the educational outcome of the individual represented by the triangle but not the individual represented by the circle, thus increasing the inequality of outcome between them but also leading to the highest individual outcome achieved across alternatives.

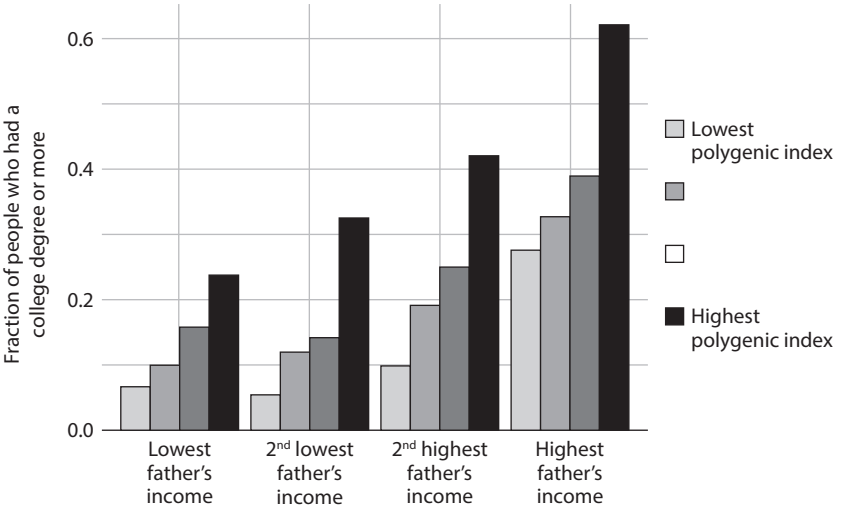


FIGURE 9.1. College graduation rates in White Americans born between 1905 and 1964, by paternal income and by polygenic index created from GWAS of educational attainment. Data courtesy of Nicholas Papageorge and Kevin Thom; results described in Nicholas W. Papageorge and Kevin Thom, "Genes, Education, and Labor Market Outcomes: Evidence from the Health and Retirement Study," NBER Working Paper 25114 (National Bureau of Economic Research, September 2018), https://doi.org/10.3386/w25114.

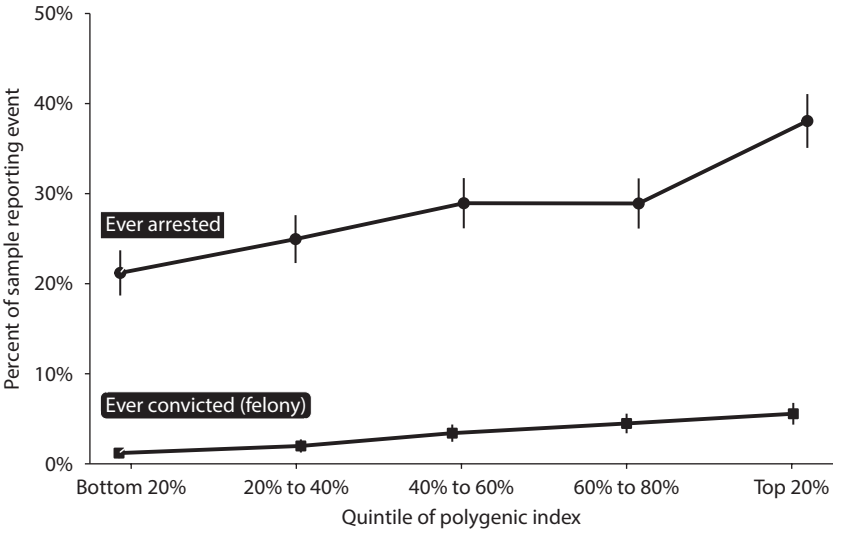
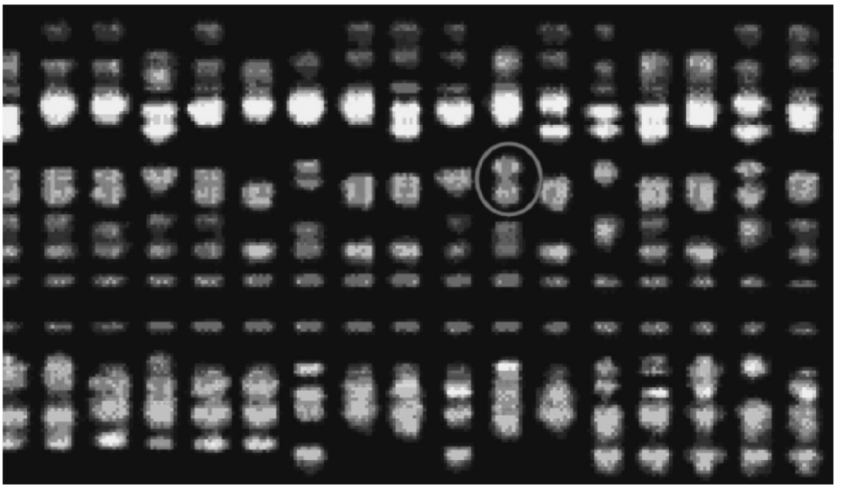


FIGURE 10.1. Rates of criminal justice system involvement and antisocial behavior by polygenic index created from GWAS of externalizing in 1.5 million people. Figure adapted from Richard Karlsson Linnér et al., "Multivariate Genomic Analysis of 1.5 Million People Identifies Genes Related to Addiction, Antisocial Behavior, and Health," bioRxiv, October 16, 2020, https://doi.org/10.1101/2020.10.16.342501.



"Scientists have found that people can have genes that lead them to behave this way. Here is a graphic that illustrates the area of the genome where these genes are found. According to recent testing, Jane has these genes. In other words, Jane's genetic makeup—the DNA that she inherited from her parents—leads her to behave the way she does in situations like these."

FIGURE 10.2. Genetic explanation of behavior. Image and text provided to participants in Matthew S. Lebowitz, Kathryn Tabb, and Paul S. Appelbaum, "Asymmetrical Genetic Attributions for Prosocial versus Antisocial Behaviour," *Nature Human Behaviour* 3, no. 9 (September 2019): 940–49, https://doi.org/10.1038/s41562-019-0651-1; image originally from Nicholas Scurich and Paul Appelbaum, "The Blunt-Edged Sword: Genetic Explanations of Misbehavior Neither Mitigate nor Aggravate Punishment," *Journal of Law and the Biosciences* 3, no. 1 (April 2016): 140–57, https://doi.org/10.1093/jlb/lsv053, by permission of Oxford University Press.

ACKNOWLEDGMENTS

The epigraph for the book comes from *The Witch Elm* by Tana French, copyright ©2018 by Tana French, reproduced with permission of Penguin Books, an imprint of Penguin Publishing Group, a division of Penguin Random House LLC. All rights reserved.

The original idea to write a book on genetics and equality was sparked by conversations I had with scholars at the Russell Sage Foundation, where I was on sabbatical for the 2015–2016 academic year. Since then, I've had the opportunity to discuss this work and learn from my colleagues in several interdisciplinary forums, including the meetings of the Genetics and Human Agency project, organized by Eric Turkheimer and funded by the John Templeton Foundation; the Hastings Center working group "Wrestling with Social and Behavioral Genomics: Risks, Potential Benefits, and Ethical Responsibility," organized by Erik Parens and Michelle Meyer, with funding by the Robert Wood Johnson Foundation, Russell Sage Foundation, and JPB Foundation; a workshop on interpreting the genetic basis of differences between populations and on the interactions among concepts used for research in social and natural sciences, organized by Danielle Allen, Anna Di Rienzo, Evelynn Hammonds, Molly Przeworski, and Alondra Nelson, sponsored by Harvard University's Edmond J. Safra Center for Ethics; a workshop, "Genes, Schools, and Interventions That Address Educational Inequality," co-organized with David Yeager and sponsored by the Human Capital and Economic Opportunity Global Working Group at the University of Chicago; and a residency on Genes and Development, co-organized with Dan Belsky and sponsored by the Jacobs Foundation. I am indebted to all of the participants of these

workshops and meetings for their incisive comments. Research for this book was further supported by grants from the Templeton Foundation and the Jacobs Foundation.

I have had the opportunity to present ideas from this book to a number of audiences, including the Duke University Population Research Institute, the Office of Population Research at Princeton University, the Department of Psychology at the University of Wisconsin, the Global Education and Skills Forum, and the Département d'Études Cognitives at the École Normale Supérieure, as well as attendees of meetings of the American Philosophical Association, Philosophy of Science Association, Behavior Genetics Association, the Integrating Genetics and the Social Sciences conference, Association for Psychological Science, American Society of Human Genetics, and American Society for Bioethics and Humanities. Thank you to these audiences for their illuminating questions and comments.

Writing a book means being distracted from one's ordinary responsibilities for a long time, and no one has borne the brunt of that distraction more than my trainees. Megan Patterson, Stephanie Savicki, Margherita Malanchini, James Madole, Laurel Raffington, Andrew Grotzinger, Travis Mallard, Aditi Sabhlok, and Peter Tanksley have been extraordinary junior colleagues whom I look forward to knowing, and working with, for years to come.

David Yeager generously facilitated a semester course release, and Jamie Pennebaker stepped in to co-teach my class, providing invaluable writing time.

Eric Turkheimer has been my mentor for nearly two decades, and nearly every page of this book bears the imprint of his influence (even if he disagrees with much of it). I also benefited from conversations with Benjamin Riley, Carl Shulman, Graham Coop, Doc Edge, John Novembre, Stuart Ritchie, Jasmin Wertz, and Razib Khan. Patrick Turley, Sanjay Srivastava, Ben Domingue, George Davey Smith and several anonymous reviewers helpfully reviewed earlier drafts of the manuscript. Alison Kalett was a careful editor and enthusiastic advocate. Innumerable people responded helpfully to my inchoate thoughts on Twitter.

I am especially thankful for the support of friends who have provided snacks, wine, advice, and encouragement throughout this long process, including Dan Belsky, Colter Mitchell, Philipp Koellinger, Nico Dosenbach, Sam Gosling, Joe Pflieger, Jane Mendle, Samantha Pinto, Jen Doleac, Sara Beckmann, and Natalia Wulfe. Every day is improved by the companionship of Travis Avery: "You and a bird flu could make me believe in fate." Micah Harden agreed to be genotyped for this book, one of many acts of brotherly love. Elliot Tucker-Drob has been, through good times and bad, my collaborator and friend; he and Barbara Wendelberger Drob are devoted coparents, and this book would not be possible without their teamwork. Finally, I am most grateful for my children, my natural experiments in within-family genetic diversity, my most precious preoccupations, and my reasons to hope for a better world.

NOTES

Chapter 1: Introduction

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7. Consistent with the American Psychological Association's style guidelines, I capitalize racial terms like Black and White. While there is not consensus regarding this issue, the Center for the Study of Social Policy argued that capitalizing Black "refers to not just a color but signifies a history and the racial identity of Black Americans." Moreover, they argued that "to not name 'White' as a race is, in fact, an anti-Black act which frames Whiteness as both neutral and the standard. . . . While we condemn those who capitalize 'W' for the sake of evoking violence, we intentionally capitalize 'White' in part to invite people, and ourselves, to think deeply about the ways Whiteness survives—and is supported both explicitly and implicitly." "Racial and Ethnic Identity," APA Style, accessed February 8, 2021, https://apastyle.apa.org/style-grammar-guidelines/bias-free-language/racial-ethnic-minorities; Ann Thúy Nguyễn and Maya Pendleton, "Recognizing Race in Language: Why We Capitalize 'Black' and 'White,'" Center for the Study of Social Policy, March 23, 2020,

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Chapter 5: A Lottery of Life Chances

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Teather-Posadas, "The Unprincipled Randomization Principle in Economics and Medicine," in *The Oxford Handbook of Professional Economic Ethics, ed.* George F. DeMartino and Deirdre N. McCloskey (New York: Oxford University Press, 2016).

7. Charles A. Nelson et al., "Cognitive Recovery in Socially Deprived Young Children: The Bucharest Early Intervention Project," *Science* 318, no. 5858 (December 21, 2007): 1937–40, https://doi.org/10.1126/science.1143921.

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9. David Lewis, "Causation," *Journal of Philosophy* 70, no. 17 (October 1973): 556–67, https://people.stfx.ca/cbyrne/Byrne/Lewis%20-%20Causation.pdf.

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11. Donald B. Rubin, "Estimating Causal Effects of Treatments in Randomized and Nonrandomized Studies," *Journal of Educational Psychology* 66, no. 5 (1974): 688–701, https://doi.org/10.1037/h0037350.

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13. More specifically, this method allows you to estimate the *average treatment effect* (ATE). However, the ATE is not the only quantity that researchers might be interested in estimating. For example, they might be specifically interested in heterogeneity in treatment response. For further discussion, see Angus Deaton and Nancy Cartwright, "Understanding and Misunderstanding Randomized Controlled Trials," *Social Science & Medicine* 210, special issue: Randomized Controlled Trials and Evidence-based Policy: A Multidisciplinary Dialogue (August 2018): 2–21, https://doi.org/10.1016/j.socscimed.2017.12.005.

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15. The evolutionary biologist Richard Dawkins made the point that genetic causes should be defined as difference makers even for relatively simple phenotypes that are intuitively "genetic," such as eye color. He wrote, "The 'effect' of any would-be cause can be given meaning only in terms of a comparison, even if only an implied comparison, with at least one alternative cause. It is strictly incomplete to speak of blue eyes as 'the effect' of a given gene *G1*. If we say such a thing, we really imply the potential existence of at least one alternative allele, call it *G2*, and at least one alternative phenotype, *P2*, in this case, say, brown eyes."

He continues with an example of two genes both related to skin pigmentation: "To be sure, *A*, the gene whose protein product is the black pigment, is necessary in order for an individual to be black. . . . But I shall not call *A* a gene for blackness unless some of the variation in the population is due to lack of *A*. . . . The point that is relevant here is that both *A* and *B* are potentially entitled to be called genes for blackness, *depending on the alternatives that exist in the population* (emphasis

added). The fact that the causal chain linking *A* to the production of the black pigment molecule is short, while that for *B* is long and tortuous, is irrelevant."

Finally, Dawkins pointed out that natural selection is concerned with differences: some versions of genes become more common than others because those versions cause differences in fitness. Evolution requires a comparison.

Failure to appreciate the fact that genetic causes, like all other causes, are difference makers that imply a comparison to some alternative is one major flaw in the reasoning of a still widely cited essay by the philosopher Ned Block. He wrote (emphasis added), "Genetic determination is a matter of *what causes a characteristic*: number of toes is genetically determined because our genes cause us to have five toes. Heritability, by contrast, is a matter of what *causes differences in a characteristic*: heritability of number of toes is a matter of the extent to which genetic differences cause variation in number of toes (that some cats have five toes, and some have six)." Block's error should be readily apparent. What causes a characteristic *is*, by definition, what causes differences in a characteristic. To say that a gene *G1* causes us to have five toes is to imply the existence of an alternative allele and an alternative phenotype—having a gene other than *G1* would cause you to have a different number of toes.

In fact, the fact that genes are difference makers can be empirically illustrated using Block's example of having five toes. Two of the genes that determine toe number are *EVC1* and *EVC2*. Rare mutations in these genes cause polydactyly (extra fingers and toes), as well as short ribs, teeth abnormalities, and cardiac defects, a syndrome known as Ellis–van Creveld syndrome. The *EVC* genes code for a protein that is found on the little hairlike projections that surround each cell; the protein helps cells communicate with each other so that they can arrange themselves into the right shapes. The *EVC1* and *EVC2* genes were discovered by studying nine Amish families in which some family members were born with extra fingers and toes. Scientists in this study focused on the exact question that Block, wrongly, identified as distinct from the question of genetic causation: they asked, What genes are associated with a difference in whether or not you have five fingers and five toes? Those who inherited two copies of a mutated form of the *EVC1* or *EVC2* genes had extra toes; those who didn't had five toes.

Richard Dawkins, *The Extended Phenotype: The Long Reach of the Gene*, rev. ed. (Oxford and New York: Oxford University Press, 1999); Ned Block, "How Heritability Misleads about Race," *The Boston Review* 20, no. 6 (January 1996): 30–35; Victor A. McKusick, "Ellis-van Creveld Syndrome and the Amish," *Nature Genetics* 24, no. 3 (March 2000): 203–4, https://doi.org/10.1038/73389.

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19. Urie Bronfenbrenner and Pamela L. Morris, "The Bioecological Model of Human Development," in *Handbook of Child Psychology*, vol. 1, *Theoretical Models of Human Development,* ed. Richard M. Lerner and William Damon, 6th ed. (Hoboken, NJ: John Wiley and Sons, 2007), https://onlinelibrary.wiley.com/doi/abs/10.1002/9780470147658.chpsy0114.

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21. Richard C. Lewontin, "The Analysis of Variance and the Analysis of Causes," *International Journal of Epidemiology* 35, no. 3 (June 2006): 520–25, https://doi.org/10.1093/ije/dyl062.

22. Clifford Geertz, "Thick Description: Toward an Interpretive Theory of Culture," in *The Interpretation of Culture* (New York: Basic Books, 1973), https://philpapers.org/archive/geettd.pdf. I am grateful to Benjamin Domingue for pointing out the similarities between my language here and Geertz's distinction between "thin" and "thick" description of behavior, e.g., "rapidly contracting his right eyelids" versus "practicing a burlesque of a friend faking a wink to deceive an innocent into thinking a conspiracy is in motion."

Chapter 6: Random Assignment by Nature

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4. Tinca J. C. Polderman et al., "Meta-Analysis of the Heritability of Human Traits Based on Fifty Years of Twin Studies," *Nature Genetics* 47, no. 7 (July 2015): 702–9, https://doi.org/10.1038/ng.3285.

5. Sophie von Stumm, Benedikt Hell, and Tomas Chamorro-Premuzic, "The Hungry Mind: Intellectual Curiosity Is the Third Pillar of Academic Performance," *Perspectives on Psychological Science* 6, no. 6 (November 1, 2011): 574–88, https://doi.org/10.1177/1745691611421204.

6. Richard C. Lewontin, "The Analysis of Variance and the Analysis of Causes," *International Journal of Epidemiology* 35, no. 3 (June 2006): 520–25, https://doi.org/10.1093/ije/dyl062

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9. Another objection: it doesn't matter that these traits are heritable because everything is heritable. That is, everything you can measure about a person that differs within a population shows some evidence of heritable variation. This extends to even silly traits, like how much TV you watch or how much Marmite you like to eat. Silly examples are useful in pushing back against the intuition, which I discussed in the last chapter, that genetic *causation* implies something like a biodeterminist *mechanism*. We are not going to understand Marmite-liking and TV-watching "at the level of the genome." But we don't care about the heritability of Marmite-liking, not because heritability is a useless and "metaphorical" statistic, but because we don't care whether people like Marmite or not. We do care, however, whether or not people graduate from college. The scientific and philosophical importance of heritability statistics is derived from the scientific and philosophical importance of the phenotype. Eric Turkheimer, "Three Laws of Behavior Genetics and What They Mean," *Current Directions in Psychological Science* 9, no. 5 (October 1, 2000), 160–64, https://journals.sagepub.com/doi/10.1111/1467-8721.00084.

10. The connection between heritability and genetic causation can be further clarified by considering how heritability coefficients are used in agricultural selection programs. The so-called "breeder's equation" is given as: $R = h^2 \times S$, where h^2 is the heritability coefficient in a population, R is the response to selection, defined as the change in the mean phenotype between generations, and S is how different the parents who are selected for breeding are from the mean in the population.

In the United States in 2019, the mean height for men is 5′9″ (176 cm). Imagine that a dystopian dictatorship ruled that only men who were taller than a certain threshold were allowed to father children. As a result, the average height among fathers selected for breeding was 6′0″. The difference between parents selected for breeding and the mean in the population is, in this instance, 3 inches. Assuming mothers were subject to selection of similar magnitude, how much taller will the next generation of male children be, on average, than they would have been in the absence of selection on the parents, assuming that everything about the environment is kept exactly the same? The heritability of height, according to the Visscher study that I described at the beginning of this chapter, was estimated to be 0.80. That's not 1.0—the next generation of sons won't also be 3 inches taller, on average. But a high heritability means that the offspring of the selectively bred parents will, in fact, be substantially taller—just over 2 inches on average. A shift in the mean of the population has implications for how frequently "extreme" values are observed. In a population with a mean height of 69 inches, about 1 percent of men are taller than 6′6″. Shift the mean height up 2 inches to 71 inches, and now about 4 percent of men are that tall.

Because it determines response to selection, the causal relevance of heritability can be further understood within the framework of the *manipulationist theory of causation*. Related to the theories of causation as counterfactual dependence that I described in the previous chapter, the manipulationist theory is not centered on the question, "What would have happened to Y if X had not happened?," but is rather centered on the question, "What would happen to Y if you changed X?"

The philosopher Jim Woodward describes this more precisely in *Making Things Happen*: "The claim that X causes Y means that for at least some individuals, there is a possible manipulation of some value of X that they possess, which, given other appropriate conditions (perhaps including manipulations that fix other variables distinct from X at certain values), will change the value of Y or the probability distribution of Y for those individuals" (p. 40).

Selection experiments are an interesting twist on this requirement. The claim that genes (X) cause the phenotype (Y) means that for at least some individuals, there is a possible manipulation of some value of X that they possess. In the case of selection, this manipulation is to restrict the range of genotypes allowed to reproduce. Given other appropriate conditions, including fixing other variables distinct from X (i.e., environmental conditions) at certain values, this will change the probability distribution of Y for those individuals' offspring.

If selection experiments demonstrate the causal power of genes for the phenotype, and heritability determines the response to selection, it is impossible to conclude that heritability is somehow irrelevant to causation. As Peter Visscher described in another paper, "Heritability is a fundamental parameter in genetics . . . it is key to selection in evolutionary biology and agriculture, and to the prediction of disease risk in medicine."

James Woodward, *Making Things Happen: A Theory of Causal Explanation*, Oxford Studies in Philosophy of Science (Oxford: Oxford University Press, 2003); Peter M. Visscher, William G. Hill, and Naomi R. Wray, "Heritability in the Genomics Era—Concepts and Misconceptions," *Nature Reviews Genetics* 9, no. 4 (April 2008): 255–66, https://doi.org/10.1038/nrg2322.

11. The equal environments assumption has been the subject of much scrutiny, and newer studies taking advantage of measured DNA have largely found support for it. One notable study took advantage of the fact that parents, pediatricians, and even twins themselves frequently misclassify zygosity—they think they are identical when they are actually fraternal, or vice versa. One study of about 300 Dutch twins found that parents were wrong about their children's zygosity 19 percent of the time. I find a similar thing in the twin study that I run in Texas: college students who have met a set of twins once are better than the twins' parents at guessing whether DNA results will show the twins to be identical or fraternal. The sociologist Dalton Conley and his colleagues leveraged this parental bias in order to test the equal environments assumption, reasoning that if identical twins are more similar than fraternal twins because their parents treat them more similarly (a violation of the equal environments assumption), then twin pairs that are *really* fraternal, but who have been misclassified as identical, will be more similar to one another than are twin pairs who have been correctly classified as fraternal. This is, in fact, what Conley was hoping to find. To a sociologist trained to view the results of behavior genetics with fear and loathing, the design seemed like a clever way to undermine the steadily mounting evidence that genes mattered for understanding social inequality. But that's exactly what he *didn't* find! Instead, the study found that twins' phenotypic similarity (i.e., how similar twins are for their outcomes) tracked their actual genetic relationship, not what their parents thought their zygosity was—evidence in support of the equal environments assumption.

Dalton Conley et al., "Heritability and the Equal Environments Assumption: Evidence from Multiple Samples of Misclassified Twins," *Behavior Genetics* 43, no. 5 (September 2013): 415–26, https://doi.org/10.1007/s10519-013-9602-1.

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Chapter 7: The Mystery of How

1. Christopher Jencks et al., *Inequality: A Reassessment of the Effect of Family and Schooling in America* (New York: Basic Books, 1972).

2. Complicated human behaviors are not the only phenotypes that are connected to genotypes via long causal chains. As the evolutionary biologist Richard

Dawkins argued, "What on earth [is] any genetic trait . . . morphological, physiological, or behavioural, if not a 'byproduct' of something more fundamental? If we think the matter through we find that all genetic effects are 'byproducts' except protein molecules." Similarly, it is now becoming clear that even apparently simple environmental interventions can also depend on long causal chains involving complex social processes, such as peer norms and teacher effects, in order to be effective. Richard Dawkins, *The Extended Phenotype: The Long Reach of the Gene*, rev. ed. (Oxford and New York: Oxford University Press, 1999)

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4. Carl F. Craver and Lindley Darden, *In Search of Mechanisms: Discoveries across the Life Sciences* (Chicago: University of Chicago Press, 2013).

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6. Charles Murray, *Human Diversity: The Biology of Gender, Race, and Class* (New York: Twelve, 2020).

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9. It's important to remember that the problem of unknown mechanisms, which perhaps operate through unintuitive mediators, is not a problem specific to genetic causes. In fact, this problem can attend *any* causal inference made from a randomized controlled trial (RCT). In their review of the strengths and weaknesses of RCTs, the Nobel prize-winning economist Angus Deaton and the philosopher of science Nancy Cartwright argued that "a great deal of other work—empirical, theoretical, and conceptual—needs to be done to make the results of an RCT serviceable." You might know that intervening in this one way under this one set of controlled conditions has this average treatment effect, but what are the boundary conditions? What is the chain of causal events between intervention and eventual outcome? How do people differ in their response to the intervention? So, too, is it insufficient merely to test the average treatment effect of a set of genetic variants on an outcome using nature's randomization. There is empirical, theoretical, and conceptual work to be done to make the results of that causal inference scientifically and practically useful. Deaton and Cartwright, "Understanding and Misunderstanding Randomized Controlled Trials," *Social Science & Medicine* 210, special issue: Randomized Controlled Trials and Evidence-based Policy: A Multidisciplinary Dialogue (August 2018): 2–21, https://doi.org/10.1016/j.socscimed.2017.12.005.

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13. Laura E. Engelhardt et al., "Genes Unite Executive Functions in Childhood," *Psychological Science* 26, no. 8 (August 1, 2015): 1151–63, https://doi.org/10.1177/0956797615577209.

14. One common criticism of twin studies is that they might underestimate the extent to which environmental factors shared by kids in the same home contribute to variation in their life outcomes, because the studies don't include sufficiently many families from disadvantaged backgrounds. Remember that heritability is a proportion, and the more environmental variation there is in the sample, the bigger the denominator and the smaller the heritability. In the case of the Texas Twin Project, however, our sample *does* represent a broad range of environmental adversity. One-third of our participating families have received some sort of public assistance (like SNAP, i.e., assistance buying food) since the kids were born. We also calculated the Gini index—a measure of income inequality—of our sample. It was 0.35, compared to 0.39 in the United States as a whole, indicating that we are doing a reasonable job, particularly given the geographical restriction of our sample, of capturing the broader pattern of income inequality that characterizes American society.

The composition of our sample is important, because it means that we don't see the very high heritability of EF just because we've only sampled children who all come from similarly affluent backgrounds. What's more, an independent lab in Colorado, run by the psychologist Naomi Friedman, found the *exact* same result of perfect heritability with a totally different sample of twins who were older at the time they were tested. Naomi P. Friedman et al., "Individual Differences in Executive Functions Are Almost Entirely Genetic in Origin," *Journal of Experimental Psychology: General* 137, no. 2 (May 2008): 201–25, https://doi.org/10.1037/0096-3445.137.2.201.

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