

Why Biology Is Not Destiny

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In *The Genetic Lottery*, Kathryn Harden disguises her radically subjective view of biological essentialism as an objective fact.

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Illustration by Vivienne Flesher

Reviewed:

The Genetic Lottery: Why DNA Matters for Social Equality

by Kathryn Paige Harden

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You must know the parable about the frog that sits in a pot of water being gradually heated, allowing itself to be boiled alive: because the change happens gradually, it never realizes it should leap out. Reading Kathryn Paige Harden's book *The Genetic Lottery: Why DNA Matters for Social*

Equality is a similar experience, as the author ingenuously points out. “Like a frog being slowly boiled alive,” she observes, readers follow her argument “from an uncontroversial premise to a highly controversial one.” Harden’s “uncontroversial premise” in this case is that siblings raised in the same family share a childhood environment and 50 percent of their DNA randomly assigned at conception, and are therefore like subjects in a controlled study of genetic differences. Ask anyone with a sibling whether their own childhood environment was the same as their sibling’s and you’ll quickly disprove Harden’s claim that her premise is uncontroversial. But putting that objection aside and sitting patiently as Harden increases the heat, we’ll arrive at her “highly controversial” assertion that “if siblings who differ genetically also have corresponding differences in their health or well-being or education, this is evidence that genes are *causing* these social inequalities.”

Harden is a dedicated frog boiler. She introduces many comfortably room-temperature premises: measurement is essential to science; people differ genetically; genes cause conditions such as deafness; a recipe for lemon chicken produces variable results but never leads to chocolate-chip cookies. Lulled to complacency by such anodyne and often homey observations, we soon find ourselves in a rolling boil of controversial claims: genes make you more or less intelligent, wealthier or poorer; every kind of inequality has a genetic basis.

Harden is right that such assertions are controversial, but they’re nothing new. The idea of a biological hierarchy of intelligence arose alongside the first theories of human evolution. It never goes away when discredited, just changes forms. In 1810, a year after the publication of the first modern evolutionary theory, two German doctors, Franz Joseph Gall and Johann Kaspar Spurzheim, inaugurated the science of phrenology by asserting that the parts of a person’s brain reflected, by their sizes, the degrees of the person’s mental powers, and that one could evaluate these by examining the shape of the skull.

That idea persisted through the nineteenth century. In 1869 Charles Darwin’s cousin Francis Galton grumbled that he had “no patience” with the empty platitude that “babies are born pretty much alike.” Rejecting “pretensions of natural equality” as morality tales for children, Galton asserted that measurements of the “head, size of brain, weight of grey matter, number of brain fibres, &c.” followed “the law of deviation from an average” and so did innate “mental capacity.” Galton was a founder of modern statistics, which he developed in conjunction with his new science of eugenics. Meanwhile, in 1876, Herbert Spencer, the English popular science writer and evolutionist, told the members of the Anthropological Institute that humans differed in the volume, complexity, and plasticity of “mental mass,” and accordingly in “quality of thought.”

Eventually, the idea of correlating the physical characteristics of the brain and skull with mental capacity or quality of thought went out of fashion, appearing naive as people turned to more modern methods. In the early twentieth century, psychologists in France, Germany, and America began developing cognitive tests. This approach became most influential in America, principally through developments at Stanford University (where we teach). In 1916 the Stanford education professor Lewis Terman published his version of an intelligence test, which quickly pervaded the worlds of education, public policy, and the professions. Terman said his test reflected not learning or culture but innate intelligence. “The common opinion that the child from a cultured home does better in tests solely by reason of his superior home advantages” was, he declared, “entirely gratuitous”: these children tested higher “for the simple reason that their heredity is better.”

By “heredity,” Terman meant biological inheritance, though he didn’t know what it was or how it worked. Five years earlier, the Danish botanist Wilhelm Johannsen had coined the term “gene” to designate a still-hypothetical “element of inheritance.” Genes soon became central to biological theories of intelligence, especially after the identification of the structure of DNA in 1953. Following the mapping of the human genome around the turn of the twenty-first century, these theories focused upon individual genes, or sequences of nucleotides in the DNA molecule. But two decades later, attempts to correlate mental traits with so-called candidate genes have gone the way of skull bumps and brain fibers.

Harden, a professor of psychology at the University of Texas at Austin, admits this. “OK,” she confides cheerfully, “so the candidate gene thing didn’t work.” No matter! Biological essentialism, aimed at demonstrating an innate hierarchy of intelligence, is going strong after more than two centuries of empirical failure. There’s always a new approach waiting in the wings. This time it’s “genome-wide association studies” of people’s “single-nucleotide polymorphisms.”

A single-nucleotide polymorphism (SNP) is a spot on the genome where people can have different variants: alternative nucleotides in their DNA. An average human has about 3.2 billion nucleotides and four million to five million single-nucleotide polymorphisms in their genome, and the genomes of any two people are about 99.9 percent the same. A genome-wide association study (GWAS) calculates a statistical correlation between patterns of DNA variants and a particular phenotype, or observable characteristic, among the sampled people. In one of the first genome-wide association studies, from 2005, researchers compared the genomes of people suffering from macular degeneration (a disease of the retina) with a control group of people who had healthy vision. They found two sets of single-nucleotide polymorphisms where the groups differed significantly. For complex diseases such as schizophrenia or bipolar disorder, however, genome studies haven’t revealed any spots showing statistically important differences between the focus and control groups; but there are thousands of spots showing statistically tiny

differences. In 2007, three geneticists proposed that for such diseases one could add up the statistical effects of all such spots in a given person's genome to produce an overall risk score for the disease.

So far, these so-called polygenic indices haven't indicated any therapeutic interventions, and their value is a matter of debate. But meanwhile, a growing number of social scientists, primarily in economics, psychology, and sociology, have seized upon the technique as a way of studying their own subjects. Social scientists engaging in "sociogenomic" research exploit existing genetic databases, which have recently become cheap to produce and readily accessible, to conduct genome-wide association studies for "social-science-relevant outcomes" such as the one Harden features most prominently in her book, "educational attainment." For a given life outcome—dropping out of high school, earning a Ph.D., having a teen pregnancy, becoming wealthy, going bankrupt—these writers claim they can use a genome-wide association study to generate a "polygenic index," or overall genetic score revealing a person's likelihood of having that outcome.

Among other phenotypes associated with "educational attainment" for which Harden cites genome studies are "grit," "growth mindset," "intellectual curiosity," "mastery orientation," "self-concept," "test motivation," and especially "a trait called Openness to Experience, which captures being curious, eager to learn, and open to novel experiences." Harden doesn't reveal just who calls this important trait "Openness to Experience" or how they measure it. Surely, there must be disagreement among researchers about what constitutes this phenotype or others in the list, such as "grit." More so, at any rate, than about what constitutes macular degeneration.

Explaining how social scientists make genome-wide association studies and polygenic scores, Harden writes:

Correlations between individual SNPs and a phenotype are estimated in a "Discovery GWAS" with a large sample size.... 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 alphabet soup in the passive voice implies that no one actively does all this estimating, measuring, counting, weighting, correlating—or that these are such technical processes that any human presence in them is irrelevant. But people are making interpretive decisions at every stage: how to define a phenotype and select people to represent it, how to count these people, which single-nucleotide polymorphisms to consider, how to weight and aggregate them. Interpretive decisions are of course essential to all science, but here there are a great many opinions dressed up in facts' clothing. "This polygenic index will be normally distributed," Harden continues, now disguising an assumption—that there are intrinsic cognitive and

personality traits whose distribution in a population follows a bell-shaped curve, a founding axiom of eugenics—as an objective fact. Harden then tells us that “a polygenic index created from the educational attainment GWAS typically captures about 10–15 percent of the variance in outcomes.” All these trappings of scientific objectivity notwithstanding, a polygenic index “captures” differences in educational outcomes the way Jackson Pollock’s *Summertime* painting captures the season: as a reflection of its creator’s radically subjective view of things (which is just fine for abstract expressionism).

If you find a magical hammer that, whenever you swing it, rewards you with funding and professional advancement, you look at your research area and see nothing but nails. Genome-wide association studies are the social sciences’ new magical hammer. Macular degeneration seems plausibly to be a nail: genomic analysis revealed two sets of single-nucleotide polymorphisms that were importantly associated with having the disease. Schizophrenia appears *not* to be a nail, though it might have some structural features a hammer could help with. The things social scientists have been swinging at aren’t just non-nails. They are to nails as ships to sealing wax, as cabbages to kings. To suggest that macular degeneration has genetic causes is to make an empirically testable proposal; to suggest that “grit” or “openness to experience” has genetic causes is to make a category mistake. These are interpretive descriptions, made of ideas, opinions, and practices, not molecules.

If we’re to have genome-wide association studies for “growth mindset” and “mastery orientation,” the possibilities are legion. How about a genome-wide association study for a trait called “corporate-speak susceptibility,” which captures the tendency to adopt terms often found in motivational pamphlets on leadership? Or one for “bogus scientism,” which captures the tendency to present interpretive opinions as objective facts? Or one for “spurious reductionism,” which captures the tendency to assume that all phenomena are reducible to nucleotides? Reducing complex phenomena to simple parts can be enlightening, but it can also be spurious. This is not to say that genes are inessential to social life. It was essential for Shakespeare to derive energy from respiration to write his plays, but a diagram of the Krebs cycle sheds no light on *King Lear*.

Before there were genome-wide association studies, people arguing for the genetic basis of social differences conducted studies comparing fraternal and identical twins raised together and apart. Harden continues in this tradition: she codirects the Twin Project at the University of Texas and invokes analyses of twin data as evidence that “genes cause differences in educational outcomes.” She cites a notorious 1969 paper by the American psychologist Arthur Jensen, who maintained that races differed in IQ and who used twin studies to argue that social interventions couldn’t overcome genetic deficiencies in scholastic achievement.

Harden condemns Jensen's racism and rejects his assertion that social interventions are futile, but she doesn't question his basic claim that genetic differences produce an innate hierarchy of scholastic achievement. She also doesn't acknowledge his dependence on fraudulent data from a 1966 paper by the English psychologist and geneticist Cyril Burt purporting to compare identical twins raised together and apart. And nowhere does she cite the Princeton psychologist Leon Kamin's 1974 devastating debunking of Jensen and Burt or engage with the critical problems Kamin raised there regarding twin studies in general, because of the impossibility of isolating genetic factors from environmental ones. While Harden, who describes herself as a political progressive, repudiates Jensen's overt racism, she resurrects the misconceived science underlying it.

Harden's purpose in *The Genetic Lottery* is to popularize the claim that social inequalities have genetic causes, and to argue that if progressives want to address inequality, they'd better confront this fact. In presenting her case, Harden revives central features of the earlier, now-discredited biological theories of intelligence: the presentation of interpretive opinions as objective facts, as we've seen; spurious reduction to a biological mechanism that is not only hypothetical but unspecified; and a claim to be writing in the interest of social progress.

Regarding spurious reduction to an unspecified mechanism: although Harden pays lip service to the principle that correlation is not causation, she both implies and explicitly argues that correlations of genetic differences with social ones indicate genetic causes of social differences. When merely implying causation, she uses weasel words: genes are "relevant" for educational attainment; they are "associated with" first having sex at an earlier age; they "matter" for aggression and violence; social and economic inequalities "stem from" genetics. Harden also says it directly: genes "cause" differences in educational outcomes; genetic differences "cause" differences in social and behavioral outcomes; a "causal chain" links a genotype with the social behavior of going to school, and another such chain joins genetics to performance on intelligence tests.

The confusion between correlation and causation in fact first arose in connection with arguments for the biological, hereditary basis of intelligence. The mathematical concept of correlation—a measure of the degree to which two variables are associated—came into existence as a linchpin of the conjoined sciences of statistics and eugenics in the 1880s. Galton developed fundamental concepts of statistics, including correlation, deviation, and regression, to provide the mathematical basis for a new "science of improving stock," for which he coined the term "eugenics." This mathematics of heredity, Galton believed, revealed evolutionary patterns in "human qualities and faculties"—for example that they naturally followed a "normal distribution," or bell-shaped curve.

Galton's younger colleague Karl Pearson, another pioneer of statistics and eugenics, further developed the mathematics of correlation. Occasionally, Pearson emphasized the distinction between correlation and causation, but more often he blurred it, for example by arguing that causes were unknowable other than through correlations. Pearson's eugenic arguments in fact worked precisely by obscuring this distinction, as when he argued that a good home environment had "practically no influence on the intelligence of boys" whereas "parentage"—heredity—did. By correlating intelligence with "parentage," Pearson continued, "you realise at once how great is the importance of the hereditary as compared with the environmental factor!" Numerical correlations, Pearson claimed, revealed the "*first* fundamental principle of practical Eugenics": "It is five to ten times as advantageous to improve the condition of the race through parentage as through change of environment."

Almost a century and a half later, it's déjà vu all over again. Harden acknowledges and disavows the eugenic origins of statistics and concludes her book with a chapter advocating what she calls "anti-eugenic science and policy," meaning policies to counter natural inequalities. Yet she also reproduces the old statistical illogic of eugenics, with the correlation/causation confusion at its core. She devotes a whole chapter to blowing smoke over the question "What does it mean to be a cause?" Here Harden, like Pearson, implies that causes are essentially unknowable other than through correlations.

She describes the Bucharest Early Intervention Project, an experiment that began in 2000 following revelations of the terrible neglect of children in Romanian orphanages. During the dictatorship of Nicolae Ceaușescu, from the 1960s through the 1980s, contraception and abortion were mostly illegal. The increasingly crowded orphanages became sites of misery where children lay unattended in metal cribs. After the fall of Ceaușescu's regime, visitors to the orphanages found hundreds of silent, listless children. American psychologists and psychiatrists selected a group of the children and randomly assigned half of them to foster care, leaving the other half institutionalized, then compared the two groups. Leaving children in such conditions in the name of rational inquiry seems a good example of the miscarriage of science. Unsurprisingly, the researchers discovered that it was better to be in foster care, where children showed an increase in IQ over those who remained institutionalized.

Harden tells this story to illustrate the fundamentally mysterious nature of causation: we don't know the mechanism, she says, by which the foster children's IQ increased. It may have been a reduction in "physiological reactivity" in a caring environment, "preventing glucocorticoids from interfering with the development of synaptic connections," or increased iodine in the diet, or a "proliferation of synapses" due to greater language exposure. Nevertheless, Harden writes, the

researchers weren't just "claiming that foster care was *associated* with higher IQ or *correlated* with higher IQ" but that it "*caused* an increase in IQ." People accepted that "being moved out of institutional care causes an increase in IQ, but how? *No one really knows.*"

Well, of course we know why it was better to be in foster care. Any or all the mechanisms Harden lists may have been involved, but the essential explanation is simple: care causes children to thrive; neglect causes them to languish. Harden's insistence that *no one really knows* how it worked reproduces some important steps in the old eugenic circular logic: first, the claim that social situations can be reduced to extremely technical, deeply hidden natural causes; and second, the assertion that these causes are fundamentally unknowable, so the best we can do is to consider their effects statistically. These prepare the third step, in which statistical analysis confirms that the social world derives its hierarchical configuration from innate differences in biology.

"In the course of ordinary social science and medicine," Harden writes,

we are quite comfortable calling something a *cause*, even when (a) we don't understand the mechanisms by which the cause exerts its effects, (b) the cause is probabilistically but not deterministically associated with effects, and (c) the cause is of uncertain portability across time and space.... I'm going to call this a "thin" model of causation.

Harden's and her colleagues' comfort level notwithstanding, her "thin causation" is really correlation, and barely even that, given the "uncertain portability" of item (c), meaning that results obtained in one setting might not be reproducible in another.

Ultimately, Harden offers no explanation for how, say, an adenine rather than a guanine in a certain spot in a person's genome makes them likelier to get an 800 on their SAT, any more than Gall and Spurzheim could specify how a bulging skull gave a person cognitive powers, or Galton could show how more "brain fibres" made for enhanced mental capacity, or Spencer could describe the connection between "mental mass" and "quality of thought," or Terman could specify what he meant by "better heredity." Harden moreover writes that each single-nucleotide polymorphism makes a minuscule difference, amounting to at most "a few extra weeks of schooling," and in some cases—as with "a SNP named rs11584700"—only "an extra two days." What sort of difference could help someone to stay in school an extra *two days*?

Of course, these measures represent statistical averages, not individuals. Still, if we're to believe the statistics reflect meaningful differences among people, we must accept not only the idea of a genetic profile for remaining in school, but also the idea that this genetic profile is composed of hundreds or thousands of infinitesimal advantages whose specific natures and mechanisms are

unknown—indeed, are so tiny as to be unknowable other than statistically. With no causal explanation for how tiny fluctuations in the genome might produce percentages of variance in years of schooling, there's no reason to think sociogenomics researchers are counting anything but their own projections. Forty-eight percent of constellations are animals, while only 33 percent are objects; is this evidence that the most successful stars arrange themselves into animal-like shapes? Or that people like to see animals in the stars? An old, protean tradition has taken on a new form: genomic astrology.

Harden's reductionism is of the "I'm no reductionist, but" variety: there's no gene for intelligence, but there's a polygenic score based upon all of your genes; "genetics might not *determine* your life outcomes, but they are still associated, among other things, with being hundreds of thousands of dollars wealthier"; genetic and environmental differences are "entangled" and "braided together," but, she argues, we should make it our business to disentangle and unbraided them.

Such talk of entanglements and braids is misleading, implying that genetics and environment are discrete strands, when in fact living things are in continual interaction with their environments in ways that transform both at every level. The late Harvard evolutionary biologist and geneticist Richard Lewontin used the concept of the "reaction norm"—a curve expressing the relation between genotype and phenotype as a function of the environment—to describe this interaction and its implications. Lewontin showed that since the relationship between genotype and phenotype depends on the environment in which the phenotype is measured, one can't infer genetic causes from correlation and regression calculations. Harden mentions Lewontin as a critic of behavioral genetics, but she implies that he didn't approve of the field simply on ideological grounds. She never mentions or engages with his substantive refutation of the core assumption that genetic and environmental causes of behavior are separable.

With an admirable poker face, Harden writes that what behavioral geneticists really care about is *environment*: they want to identify the genetic causes of different life outcomes just to get them "*out of the way*, so that the environment is easier to see." This is impossible, even as an ideal, because the environment is in the genome and the genome is in the environment. We can no more unbraided genetics and environment than we can unbraided history and culture, or climate and landscape, or language and thought.

Progressives, Harden says, shouldn't be afraid to acknowledge genetic causes of inequality; instead, they should work to narrow "genetically associated inequalities" with programs specially benefiting the genetically disadvantaged. She implies it's a new departure for a political progressive to espouse the idea of inherent differences in intelligence, but in fact scientists

arguing for a biological hierarchy of intelligence have traditionally invoked progressive values. Harden indeed sounds like Spencer, who said his science would help rectify “ignorant legislation” and “rationalize our perverse methods of education.”

Just how can behavioral genetics serve the interest of social progress toward greater equality? Harden never says. She does mention three examples of programs or policies that she claims have helped to rectify natural imbalances in intelligence, but none involve genomic analysis. The first is the 1957 law in the UK requiring children to remain in school until age sixteen; the second is an intervention program based at the University of Oregon to reduce teen drinking called the Family Check-Up; and the third is the approach to math instruction in “advantaged high schools.” All three, Harden writes, have particularly benefited those she says are genetically disadvantaged.

Regarding the better math performance by students in rich high schools, Harden says it’s “not clear yet why this is”: it might be because of tutoring and mentorship, or a social norm valuing math performance. Isn’t it likely both of these and other factors too, such as smaller class sizes, a less distracting environment, more qualified teachers? There’s no mystery about why it’s better to study math in a rich high school, nor does it require sequencing the students’ genomes to explain it. Harden also recommends social policies equalizing “access to clean water and nutritious food and health care and freedom from physical pain.” Right on! What has any of this to do with genomics?

Speaking of Harden’s progressive, egalitarian values, we come finally to the elephant in the book: race.

Harden’s statements about race don’t hang together.

First, she endorses the growing consensus among biologists that human races are social categories, not natural kinds, and that the concept of race “does not stand up scientifically.” Biologists have mostly turned from talking about races to talking about genetic populations based on genetic ancestry. The genetic populations they study don’t line up with social categories of race, which don’t even line up with one another across time and place—a sure way to tell they’re social categories and not natural kinds.

Metropolitan Museum of Art
Woodblock by Itō Jakuchū, circa 1900

Having distinguished genetic ancestry from race, however, Harden continually elides the two, as when she says that genomic research has so far been based almost entirely on “people whose recent genetic ancestry is exclusively European and who are overwhelmingly likely to identify as White.” Harden mentions this fact about genomic research in order to explain that her claims about the genetic basis of differences in intelligence apply only to differences among white-identifying people rather than to differences between whites as a group and people of other racial identities.

Here again Harden echoes her predecessors: Galton wrote in 1869, “The range of mental power between—I will not say the highest Caucasian and the lowest savage—but between the greatest and least of English intellects, is enormous.” Social class, as much as race, provided the focus of Galton’s eugenic writings; he too argued for an innate biological hierarchy of intelligence among white people. Harden’s assertion that “genetics can be causes of stratification in society” accords well with Galton’s view that social classes were based in biology.

Regarding race, Harden’s message is to relax: She has nothing to say about genetics and intelligence in nonwhite people, so how can her argument have racist implications? Moreover, she writes that the genome studies of white people will likely not be “portable” to other races, which will differ in frequencies and co-occurrences of genetic variants, precluding interracial comparisons based on such studies. What happened to the idea that races aren’t natural kinds? Once again Harden elides the crucial distinction between genetic populations and races when she writes that genome studies will probably not apply across “genetic ancestries *or* socially defined races.” Her use of italics seems to emphasize a distinction between ancestry and race, yet she continually treats them as equivalent, offering no explanation for why race would pose a significant barrier to applying genome study results across populations defined by genetic ancestry.

Harden’s distinction between genetic ancestry as a scientific category and race as a social one gets even blurrier when she writes that “socially constructed race differences are systematically related to genetic ancestry,” which seems to contradict her endorsement six pages earlier of the view that the concept of race “does not stand up scientifically.” Or again when she observes that “people’s moral commitments to racial equality are on shaky ground if they depend on exact genetic sameness across human populations.” No one alleges exact genetic sameness across human populations. The central point regarding genetics and race is that the defining criteria for races are social, not genetic, and the social categories of race don’t correspond with genetic differences among populations. In her efforts to assure the reader that there’s no racism here, Harden tacitly—and sometimes not so tacitly—endorses the founding axiom of scientific racism since its inception in the eighteenth century, that human races are biologically distinct.

“Let us not flinch,” Harden writes finally,

from considering what seems like the worst-case scenario: What if, next year, there suddenly emerged scientific evidence showing that European-ancestry populations evolved in ways that made them genetically more prone, on average, to develop cognitive abilities of the sort that earn high test scores in school?

Her answer is that we’ll need to confront this source of inequality and “arrange society” to correct for it. She doesn’t say how. Would we sequence children’s genomes as they enter preschool and put the genetically disadvantaged ones of mostly non-European ancestry in remedial programs? It’s a lot easier to imagine the disadvantages than the benefits to children of non-European ancestry placed in remedial programs for the genetically challenged. She also doesn’t say how we’d correct for the problem of confirmation bias and self-fulfilling prophecies: teachers and others would surely view those designated as genetically disadvantaged differently, and treat them accordingly, which would create differences to match the designation.

These objections come not from head-in-the-sand progressives, but from logic. Genome studies can illuminate things that genes cause, but genes don’t cause everything. Whatever scientific evidence emerges regarding genetic populations, it won’t explain why some students do well on tests any more than it will explain why some social scientists construct essentialist theories of intelligence. Educational success and biological essentialism are social and cultural phenomena, not genetic phenomena. True, genes help shape people, and people make up social and cultural situations. Likewise, grammar helps shape sentences and sentences make up Harden’s book. But we can’t reduce her contention that genetic differences cause social differences to the syntactical rules of an English sentence. Meanwhile, beneath Harden’s protestations that she’s an egalitarian hides a stealthy affirmation of the old, tenacious view that races and classes are natural kinds.

Back finally to frog boiling: this practice, it turns out, is directly, not just metaphorically, related to arguments for a biological hierarchy of intelligence. The first experiments testing the reflexes of frogs in gradually heated water took place in the mid-nineteenth century, around the same time as the first theories of the biological basis of intelligence. Experimenters also lobotomized frogs, severed their spinal cords, heated and chilled their brains, and subjected them to strychnine poisoning, among other forms of torture, to see how these affected their reflex responses. Spencer cited these experiments in support of his theory that intelligence arose by infinitesimal degrees from the rudimentary reflexes of lower animals, through higher animals, “the inferior human races,” “the villager,” and “the man of ordinary education,” to “the advanced man of science”—i.e., Spencer himself.

Harden was right to compare her reasoning to the reasoning of the frog boilers. Both the logic and the experimental program of frog boiling exemplify the essentialist tradition in which she is a participant. But the theory doesn't hold up in experiments: the frog, if intact and in a vessel it can escape, will actually jump out rather than be boiled alive. Our message to you, reader, is accordingly simple: jump out.

Letters:

Kathryn Paige Harden

'Why Biology Is Not Destiny': An Exchange

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