

# **'Why Biology Is Not Destiny': An Exchange**

**Kathryn Paige Harden, Nick Patterson, Victor I. Reus, and Henry D. Schlinger Jr., reply by M.W. Feldman and Jessica Riskin**

June 9, 2022 issue

**In response to:**

*Why Biology Is Not Destiny* from the April 21, 2022 issue

*To the Editors:*

Marcus Feldman and Jessica Riskin did not like my book. Or rather, they did not like a book called *The Genetic Lottery* by an author named “Kathryn Paige Harden,” but their review [NYR, April 21] so badly distorts my arguments that I have the curious impression that Feldman and Riskin somehow got their hands on another book entirely, an evil doppelgänger to mine. Therapists, parents, and the unhappily married would recognize the feeling I had upon reading their review the first time: it’s both vexing and bewildering when someone is spoiling for a fight about something you never said.

As a longtime reader of *The New York Review of Books*, I am surprised and disappointed that this review was published in such an esteemed outlet. Yes, “we all enjoy an intemperate paragraph of syntactically inspired bile,” to quote Zadie Smith, and Feldman and Riskin do deliver the bile. But I assume that, besides wanting to be entertained by vitriol, readers of book reviews are also interested in learning what a book is about. Here, Feldman and Riskin’s review fails entirely. That quote of Smith’s is from her review of *My Prizes* by Thomas Bernhard, about which she wrote: “The gap between what actually happened and how Bernhard writes of it can be interpreted variously as postmodern playfulness or deceitful paranoia.” The gap between what *The Genetic Lottery* says and how Feldman and Riskin write of it is very large indeed, and no tone of playfulness is to be found.

Feldman and Riskin set out to slay three dragons: genetic determinism (“why biology is not destiny”), genetic essentialism (“you are not your genes”), and genetic reductionism (“all phenomena” are not “reducible to nucleotides”). They argue that these three “isms” are not supported by the scientific evidence—and I wholeheartedly agree. In fact, *The Genetic Lottery* devotes considerable space to describing why these three “isms” are wrong. As I summarize in the last chapter:

Much of this book has been taken up with these arguments. We should *not* interpret genetic influences as deterministic. We should *not* give up on the possibility of social policy to bring about social change. We should *not* confuse an outcome being socially *valued* with a person being *valuable*.

Passages like this one, which directly contradict their characterization of the book’s alleged determinism, reductionism, and essentialism, are easy to find! But Feldman and Riskin are not here to engage with what *The Genetic Lottery* says. Their aim, instead, is to expose what they see as the sinister, repressed meanings of my book, which are cloaked, in their view, by my cluelessness and my duplicitousness. Throughout the review, I am portrayed as the unwitting dupe of false consciousness (“beneath Harden’s protestations...”) or worse, as a liar (“with an admirable poker face, Harden writes...”). The possibility that I might mean what I say does not enter the picture. The hermeneutics of suspicion has its place, I suppose, but as Paul Ricoeur said, hermeneutics is animated by a “double motivation: willingness to suspect, willingness to listen.” They forgot the listening part.

In addition to grossly distorting the arguments of my book, Feldman and Riskin also turn their suspicious gaze to a variety of ordinary scientific concepts. In their telling, the “normal” distribution, which was first described, decades before Galton, by a mathematician studying errors in astronomical observations, is a “founding axiom of eugenics.” They write about the measurement of personality differences in a conspiratorial tone more commonly associated with Reddit discussions about the Deep State. They dismiss the importance of random assignment for causal inference as “blowing smoke.” There would be little left of the behavioral and social sciences if everything that Feldman and Riskin considered dubious were removed.

But perhaps they would like to be rid of all of the behavioral and social sciences? We could return to that more innocent time, when scientists didn’t go about confusing cabbages with kings, when behavior was understood solely in terms of “social practices” and free from any reference to biology. You know, the good old days, when autism was

blamed on “refrigerator mothers” and being gay was blamed on domineering ones, when schizophrenia was the result of familial “double binds,” when patients with psychosis from late-stage syphilis suffered without any treatment options.

There is no arguing with someone committed to a paranoid reading, and doubtless Feldman and Riskin will respond to this letter with further distortions about how my book should “really” be interpreted. Fortunately, I trust readers. Based on the hundreds of e-mails and comments I have received about *The Genetic Lottery*, people are more than able to appreciate and engage with the book’s arguments. To quote one (anonymous) reader comment:

As someone with a chronic illness influenced by genes (T1D), and the mother of a child with a genetic syndrome and its attendant disabilities, I have wished for someone to wade into the fraught waters between eugenics on the right and the refusal to consider genes as important to life outcomes on the left. It is impossible to live lives where genes have such obvious and significant impact without feeling abandoned by both sides, frankly; that, on the one hand, we should accept our lesser status and try not to further contaminate the human race with our dirty genes, or on the other, we have certain accessibility rights defined by physical limitations but are never to link them to genetic causes in polite company, or mention the gaps that such a refusal creates in our ability to live good lives. *The Genetic Lottery*...elegantly discuss[es] how to address genetic causes for social outcomes within a framework of equity and human rights.

Near the end of their review, Feldman and Riskin acknowledge that, “True, genes shape people, and people make up social and cultural situations.” They don’t take that idea any further, leaving unanswered questions such as: How do we know that genes shape people? What aspects of people do genes shape? And what should we do with any new scientific insights about how genes shape people, especially if we want to avoid the evils of eugenics? These are exactly the questions that *The Genetic Lottery* addresses. Read it for yourself and decide.

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*To the Editors:*

In the April 21, 2022, issue of *The New York Review*, M.W. Feldman and Jessica Riskin publish a hostile review of Kathryn Paige Harden’s recent book *The Genetic Lottery: Why DNA Matters for Social Equality*. The review makes some arguments that make no sense

to me and deserve rebuttal.

Let us discuss the genetics of Educational Attainment (EA), defined as the number of years of schooling and measured in adults over thirty years old. EA takes up a good part of both the book and the review. Using genetic data from more than 300,000 individuals of European ancestry, it was possible to develop a “score” using the genomes of the people in the study. The details of the score are of lesser importance, but it’s important to realize that the score is a single number calculated from a genomic sample, by a fixed recipe. The score is correlated with EA at an enormously significant statistical level. This result was then replicated in an Icelandic study, using entirely different individuals from the first study. Again enormously significant results were obtained.

How do Feldman and Riskin explain these results? After a somewhat rambling diatribe complaining that choices were made in the details of the score, and how exactly the EA phenotype was chosen to study, they conclude that “researchers are [not] counting anything but their own projections.” How is this reasonable? A recipe is given, checked in a different study, and the results replicated. (Incidentally, a much larger study with more than 3 million (!) individuals was completed just this month and the results were again replicated.) Are we somehow to believe that experimental error in Iceland is correlated with EA of a sample? This is truly absurd.

The score has other interesting features. The average has been decreasing in Iceland since at least 1910, and the score strongly correlates between mating couples, an effect much stronger than correlation of EA. This argues that the score is meaningful without making the meaning clear. The work on EA is technically little different from studies of the genetics of height, and if we took the criticisms of Feldman and Riskin seriously that would invalidate an enormous amount of modern genetics, in which it is routine to find that complex traits are associated weakly with multiple genetic loci.

Feldman and Riskin also attack Harden for stating that the score will be normally distributed. Claiming that a trait follows a bell-shaped curve is “a founding axiom of eugenics.” This is an argument of guilt by association. The score is a sum of small values mostly independent and any geneticist, or statistician, will expect the distribution to be approximately normal. In a given study it is trivial to check normality of the computed scores.

This review is baffling. Feldman is a leading mathematical biologist at Stanford who I would have assumed understands statistical genetics, yet if I didn’t know who the reviewers were I would have thought that they were incompetent or ignorant. Perhaps

Feldman and Riskin think that any argument is acceptable if it goes against results that they dislike?

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*To the Editors:*

M.W. Feldman and Jessica Riskin rightfully point out the many fallacies and misinterpretations in Kathryn Paige Harden's new book, but appear dismissive of all efforts to identify genetic contributions to complex behaviors, going so far as to suggest that investigating genetic contributions to social life is somehow equivalent to comparing a copy of the Krebs cycle to Shakespeare's *King Lear*.

How then to understand such experiments of nature as Williams-Beuren syndrome, a rare congenital genetic disorder caused by a deletion of twenty-six to twenty-eight genes on chromosome 7, and characterized by distinctive facial features, cognitive and cardiovascular dysfunction, and, most interestingly, extreme social affability and an engaging extroverted personality? Structural variants of the genes identified in the syndrome furthermore have been found to be associated with stereotypical hypersociability in dogs and implicated in the behavioral divergence of dogs and wolves.

It is clear that unraveling complex behavioral phenotypes into component parts that relate best to genetic risk is a difficult undertaking, but it is not a folly, and a more sophisticated analysis of the literature would accord greater respect for the field.

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*To the Editors:*

M.W. Feldman and Jessica Riskin, in their excellent, well-written, and very entertaining review of *The Genetic Lottery* by Kathryn Harden, allude to but do not specify how one's environment can be defined functionally and scientifically. Such a definition is the crux of

the debate about nature and nurture and any discussion of genetic influences on behavior.

I believe that Feldman and Riskin, not to mention everyone else who talks about environment, would benefit from a behavioral view of *environment* as “all stimuli that affect behavior at any given moment” rather than the more widely accepted and general one which is “the surrounding conditions in which a person, animal, or plant lives or operates.”

The behavioral view of environment has three important implications: 1) the environment is not just our surroundings but is inside of us as well because there are stimuli (pain, proprioceptive, etc.) that are located inside our bodies that affect our behavior; 2) the environment is constantly in flux as it comprises “all stimuli that affect an individual’s behavior *at any given moment*,” which means that one’s environment changes moment to moment; and 3) no two individuals can ever have the same environment, not even monozygotic twins.

To the last point, my mother used to ask me how my two siblings and I could be so different even though we were raised in the same environment. Now I know that she meant the same parents, house, etc. But according to a behavioral view of environment, we weren’t raised in the same environment because one’s environment consists of all stimuli that affect behavior moment to moment. And even though that environment begins negligibly in the last trimester before birth, the number of responses and stimuli explode and expand exponentially at birth. When one considers the staggeringly high number of possible behavioral responses and stimuli those responses can interact with, any genetic explanation of human behavior takes a back seat to environmental ones. As Feldman and Riskin wrote, “living things are in continual interaction with their environments in ways that transform both at every level.”

To the behavioral view of environment we can add the discovery of how stimuli affect behavior as antecedent events (e.g., discriminative stimuli) and, more importantly, as (reinforcing) consequences, which themselves determine whether and how antecedent stimuli evoke (or abate) behavior. This conception of environment and a selectionist view of how consequences cause behavior have proven to be a much more productive avenue for research and, perhaps more importantly, interventions to change behavior in sometimes dramatic ways.

Of course, genetics cannot be completely discounted because the capacity of an organism's behavior to be affected by the environment is a function of the species' evolutionary history and genetic endowment. Nonetheless, a functional and scientific conception of environment can counteract to a large degree any biological or genetic claims of behavioral causation.

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**M. W. Feldman and Jessica Riskin reply:**

Kathryn Paige Harden accuses us of selective reading, but she herself is an uncareful reader. To begin with, she writes that we've charged her with "genetic determinism." But we haven't done this anywhere in our review. We were as careful to acknowledge her disavowals of genetic determinism as she was to place them at regular intervals throughout her book. Although we didn't quote the one she excerpts in her letter, we did quote an equivalent one, which occurs on page 46: "One's genetics might not *determine* your life outcomes, but they are still associated, among other things, with being hundreds of thousands of dollars wealthier at the end of one's working life." As we said in connection with this passage and others like it, Harden's reductionism is of an "I'm no reductionist but" variety; or we might just as well have said that her essentialism is of an "I'm no essentialist but" variety. Another example: "genetic luck is braided together with other differences" (page 43), but "Can we really say that genes *cause* you to be wealthier? (Short answer: yes)" (page 44).

When she says we described the "normal" distribution as "a founding axiom of eugenics," Harden misreads us. In fact, we wrote that the assumption "that there are intrinsic cognitive and personality traits whose distribution in a population follows a bell-shaped curve" was a founding axiom of eugenics—not, of course, the normal distribution itself, which isn't an axiom of any kind, eugenic or otherwise.

Harden also misquotes us when she writes that we "acknowledge that, 'True, genes shape people, and people make up social and cultural situations.'" She leaves out a crucial word. What we wrote was that "genes *help* shape people," hardly a momentous "acknowledgment"; neither we nor anyone else would be likely to deny it.

Oddly, Harden says we seem nostalgic for a “more innocent time” in the “good old days.” This is surprising: we wouldn’t have guessed our historical sketch of eugenics and scientific racism in the nineteenth and early twentieth centuries could seem nostalgic. At the same time, it’s interesting to note that the very conditions and characteristics she lists as having received much worse treatment in those old days—autism, homosexuality, schizophrenia, psychosis, and syphilis—all seem like good examples of a principle we advanced in our review: genes don’t cause everything. We agree that many today regard these human phenomena in a better light than previously, but this is not due to behavioral genetics or sociogenomics.

Finally, Harden asks whether we reject the psychological and behavioral sciences altogether. We don’t: we reject those areas of the psychological and behavioral sciences that claim a spurious reduction of complex social phenomena to genes. Happily, we know that some researchers in the area are pursuing better, less reductionist lines of research. The intrinsic and extrinsic aspects of people in social situations aren’t fixed and distinct but are continually shaping and transforming one another, so that there’s no fact of the matter about which causes what. It’s not that these situations are too complex for us to figure out how much is intrinsic and how much extrinsic, but that the question itself is meaningless. How much is the California coastline due to the Pacific Ocean and how much to the North American continent?

Overall, Harden’s letter confirms what we said in our review: while disavowing essentialist, racist, and eugenic notions she affirms a new version of the old illogic that has long supported them.

Nick Patterson’s arguments are beside the point. First, regarding polygenic scores for “educational attainment,” he writes that these are calculated “by a fixed recipe.” OK, but assuming this recipe didn’t come from a burning bush, someone must have written it, making many interpretive choices along the way, as we said in our review, such as which single-nucleotide polymorphisms to consider, how to weight and aggregate these, and what definition of “educational attainment” to adopt. Patterson mentions “years of schooling.” Dissatisfaction with this parameter led various institutions, including the US Census Bureau, to replace it with “highest level of school completed or highest degree achieved.”

These parameters too (like all parameters) carry ambiguities and interpretive assumptions. For instance, what kinds of school? The Census Bureau generally includes “professional” but not “vocational” training, allowing training in chiropractic but not automotive mechanics or airline maintenance. The main study that Patterson cites



(Okbay et al. 2016) draws on many surveys conducted in different countries, all with different educational systems and definitions of “educational attainment,” and uses a UN framework to normalize the results to “years of education.” In short, Patterson’s “fixed recipe” hides many layers of implicit social theory.

Next, Patterson emphasizes that polygenic risk scores correlate with “educational attainment” to a statistically significant degree; he in fact says these correlations are “enormously significant.” Since “enormous” is not a technical term it’s hard to guess what he might understand by it. “Significant” does have a technical meaning in statistics. Just as correlation is not causation, “significant” does not mean “important” or even “relevant.” A “significant” effect is one whose p-value—or probability of resulting from pure chance—is less than a certain specified value. Accordingly, it can still be tiny and/or causally irrelevant.

In fact, data mining can even extract highly “significant” correlations that “explain” a set of randomly generated dependent variables using a set of randomly generated independent variables. The three studies Patterson mentions do indeed use data-mining techniques to, as he says, develop polygenic indices for “educational attainment” and to make various weak correlations of the latter with the former. Of course, we never disputed that such studies do this; as we said in our review, the field of sociogenomics is devoted to creating statistical correlations between social outcomes according to various interpretive definitions and aggregates of up to many thousands of single-nucleotide polymorphisms. Our point is that there’s no reason to think these statistical correlations have any causal meaning.

Patterson writes that our criticisms “would invalidate an enormous amount of modern genetics.” Again, we won’t interrogate what he means by “enormous,” but will just assure readers that our review of *The Genetic Lottery* is not meant to invalidate modern genetics. For some physical traits, such as height or body mass index, polygenic scores are associated with a sizeable fraction of the variation. Even in these cases, the traits are clearly environmentally malleable, and there is no fact of the matter about how much of the variation is due to genetics versus environmental factors, as Richard Lewontin showed, because of the interaction between the two. Therefore, we would indeed question any claim to show specifically genetic causes of such traits on the basis of polygenic scores. Happily, such claims constitute what we would judge to be a less than enormous portion of the field.

Finally, Patterson is incredulous that we would question the idea of a polygenic score following a bell-shaped curve, writing that “the score is a sum of small values mostly independent and any geneticist, or statistician, will expect the distribution to be approximately normal.” He says that only someone incompetent or ignorant of statistics could deny this. But our point is ontological, not statistical. The question is not whether a sum of small, independent values would be likely to follow a normal distribution. The question is whether there’s any reason to think such a sum can bear a meaningful relation to cognitive, psychological, social, or behavioral aspects of people.

Here’s an example to illustrate what we mean: we find some letter-writers to *The New York Review* to be cogent while others are tendentious. No doubt, given the right genetic database and using our definitions of cogent and tendentious letter-writing, we could produce polygenic scores for each, made up of many independent variables. On that basis, a competent statistician might expect the distributions of cogent and tendentious letter-writers to the *The New York Review of Books* to be bell-shaped. But such a score would be a fabrication with no causal meaning and would not help to explain the occurrence of tendentious letters to the editor.

**V**ictor Reus invokes the rare disease Williams-Beuren syndrome, which has a known and relatively simple genetic cause. There is no doubt that even simple genetic abnormalities may cause many phenotypic consequences; this is called pleiotropy. But this is a very different situation, even something like the opposite situation, from what the authors of polygenic scores are claiming. In Williams-Beuren and other such syndromes, differences or deletions in one area in the genome cause many effects throughout the body. Conversely, the authors of polygenic scores claim that aggregates of tiny differences throughout the genome have specific effects on complex phenotypes.

Polygenic risk scores therefore do not “[unravel] complex behavioral phenotypes into component parts.” Rather, they associate complex behavioral phenotypes with aggregates of thousands of single-nucleotide differences. Moreover, genome-wide association studies of social-behavioral phenomena such as “educational attainment” associate only a tiny fraction of the variation among individuals with single-nucleotide differences, and there is no reason to think these differences are causal.

Henry Schlinger raises an important point, not only for human behavioral traits but for evolution in general. Richard Lewontin challenged the neo-Darwinian assumption of a fixed environment, pointing out that organisms are continually constructing and transforming their environments through their own behaviors and social interactions. This idea was formalized in a series of articles and a 2003 book, *Niche Construction*, by F.

John Odling-Smee, Kevin N. Laland, and Marcus W. Feldman, and taken further in articles in *Nature* and the *Proceedings of the Royal Society B*. The “extended evolutionary synthesis” framework incorporates this continual, mutual transformation of organism and environment into the evolutionary picture.