

Article

In Defence of Behavioral Genetics

Peter M. Visscher^{1,2} 

¹The University of Queensland, Australia and ²Nuffield Department of Population Health, University of Oxford, UK

Abstract

The author presents a commentary on the trilogy of papers by Damien Morris entitled ‘Behavioral Genetics and Human Agency: How Selectively Deterministic Theories of Free Will Drive Unwarranted Opposition to Behavioral Genetic Research and Undermine Our Moral and Legal Conventions’, published recently in this journal [PMID 40457806, 40963213, 41107251]. In addition to commenting on this specific set of papers, the author offers a broader perspective and defense of the field of behavioral genetics.

Keywords: Behavioral genetics; Human trait exceptionalism; Human agency; Determinism; Free will; Moral and legal conventions

(Received 7 April 2026; revise received 7 April 2026; accepted 8 April 2026)

I was asked to write a commentary on a series of recent articles in *Twin Research and Human Genetics* by Damien Morris on ‘Behavioral Genetics and Human Agency’ (Morris, 2025a, 2025b, 2025c). The three articles are a criticism of published work by Eric Turkheimer (ET) and, to a lesser extent, K. Paige Harden (PH). The hope was that ET and/or PH would formally respond to the Morris’ papers and that I would wrap it up with a summary of the arguments, some conclusions and, perhaps, some wise words. Alas, both ET and PH declined the invitation. I did not want to let the Editor down so hereby a commentary on the work by Morris, which, as will become clear, is more a general defense of the field of behavioral genetics (BG) than a dissection and discussion of the philosophical, factual and logical arguments in the articles from Morris.

I have not read the primary ET papers that Morris cites. In the past I read and cited (Visscher et al., 2008) ET’s interesting empirical work on the estimates of heritability of IQ in 7-year-old children stratified by socioeconomic status (Turkheimer et al., 2003). The conclusion, that estimates of heritability of IQ were larger in higher SES groups, echoes what has been found in other species (Visscher et al., 2008), including studies in dairy cattle (my PhD thesis), in that heritability tends to be higher in ‘better’ environments. I read PH’s book *The Genetic Lottery: Why DNA Matters for Social Equality* (Harden, 2021) and thought it was a brave but futile attempt to appease both polarized sides of the political spectrum in the US in making a case that genetic variation in behavioral traits matters when it comes to understanding individual differences between people and understanding society, but also that such variation is neither deterministic nor absent. I was appalled by the unfair reviews I read of her book because they provided a what seemed deliberately misleading and biased

summary of the discoveries in human genetics studies in the last few decades.

As a quantitative geneticist (Walsh et al., 2025) I do not consider myself as a bona fide behavioral geneticist. However, behavioral phenotypes are also quantitative (complex) traits, and the study of the genetics of such traits is in principle no different than the study of, say, human height or liability to cardiovascular disease. I have been involved in a number of ‘core’ BG studies, notably four genomewide association studies (GWAS) on educational attainment (EA; Lee et al., 2018; Okbay et al., 2016; Okbay et al., 2022; Rietveld et al., 2014). Another disclosure is that in 2022 I was accused by ET in a blog of using eugenic language because I stated in an invited News & Views type commentary in the journal *NPJ Science of Learning* that it is a lottery who your biological parents are. In particular, I wrote (Visscher, 2022) what I still believe is self-evident:

Genetic variation underlying human traits is the result of two genetic lotteries. Firstly, it is the lottery of who your biological parents are and secondly it is the lottery of which genetic variants you have inherited from your parents. The first explains a proportion of the differences between families whereas the latter explains a proportion of the differences between brothers and sisters. Nature is not fair, hence the title of Harden’s book. (p. 1)

A third disclosure is that I wrote to PH to say that I enjoyed reading her book.

Now to the three articles from Damien Morris. The very long title of Morris’ trilogy more or less summarizes the main point he wishes to make: ‘Behavioral genetics and human agency: How selectively deterministic theories of free will drive unwarranted opposition to behavioral genetic research and undermine our moral and legal conventions’. The main arguments are about human agency, free will and what, if anything, studies in humans about behavioral traits can say about this. Apparently, it has been suggested in the social sciences literature that the magnitude of a correlation between monozygotic (MZ) twins for a behavioral trait can be used to quantify free will, human agency and criminal

Corresponding author: Peter M. Visscher; Email: peter.visscher@ndph.ox.ac.uk

Cite this article: Visscher PM. In Defence of Behavioral Genetics. *Twin Research and Human Genetics* <https://doi.org/10.1017/thg.2026.10065>

responsibility. Along the lines of ‘if the MZ phenotypic correlation is 1 then there is no free will’. I had never come across this proposed link between an empirical phenotypic correlation coefficient and free will before, and at face value it would appear rather far-fetched. Yet this may reflect my ignorance, and others must have taken it seriously, as evidenced by the references and notes in Morris’ series of articles and because presumably Morris would not have written his opus otherwise.

Morris forensically and cogently dissects the writings of ET (and to a lesser extent PH), demonstrates internal inconsistencies and contradictions in their published work and argues against their overall conclusions about human agency and free will. Those conclusions are classified by Morris as belonging to the philosophical camp of ‘libertarian incompatibilism’, which states that humans are not responsible for behavior that is caused by extrinsic factors, yet that free will exists because some behavior is self-caused. As an example of an inconsistency and contradiction, Morris shows that ET has argued that ‘nonshared environmental variance’ (which is twin research jargon for the variance of unknown residual effects that are not correlated among siblings) is a measure of free will, yet also argued that genetic variation (and heritability) does not signal causation. The criticism from Morris is not just general and philosophical, but also to the level of individual sentences from published papers. The latter might be necessary and perhaps common in the psychology and philosophy literature, but I am sure if someone did the same by forensically going through all of my publications that contradictions and inconsistencies would be found too! Since I am not a philosopher and do not know the relevant literature on the topic of human agency, free will and criminal law, I cannot really comment on these parts of the Morris trilogy. However, as an interested reader not skilled in philosophy and arguments around free will, I thought that Morris’ arguments were cogent. It is in my opinion a scholarly piece of work. I will instead comment on the other parts in the Morris papers, which are essentially a strong defense of behavioral genetics methods, empirical analyses and statistical inference, and an overall defense of the research area of behavior genetics.

As Morris articulated (part I), the notion that variation in behavior is not in part explained by genetic variation is surely untenable. We only have to look at other mammalian species to show that genetics matter. Dog breeds have been heavily selected on multiple traits, including behaviors such as herding, retrieving, pulling sledges, guarding, guiding, and so on. Notably, most of the genetic variation in those traits was present in the ancestral (wolf) population. Silver foxes can be selected to become more docile and ‘dog-like’ after multiple generations of selection, again showing that the initial unselected population harbors plenty of genetic variation associated with behavior (Trut et al., 2009). Cows show variation in ‘temperament’ (anxiety, flight response). There is variation among inbred mouse lines in behavior, and so on. So, the notion that variation between humans for behavioral traits is not at least in part due to genetic variation is simply untenable. Note that this genetic variation is also causal, in the sense that the population mean changes with artificial selection.

In humans, there are multiple scientific questions around behavioral traits which are, in my opinion, interesting to pursue just out of curiosity alone. They include the quantification of genetic variation associated with behavioral traits, dissection of genetic trait variation between and within families and to the level of individual DNA variants, and the quantification of genetic and environmental correlations with other traits, including disease. In other words, what are the causes and consequences of genetic

variation associated with human behavior? There are researchers who object to the study of genetic variation underlying behavioral traits (in particular, intelligence) for historical (Kevles, 1998) and ideological reasons (Meyer et al., 2023) and for reasons of utility (‘there is no practical benefit or application’).

There are also apparent scientific objections to empirical studies in BG, which is surprising because the same criticism is not levelled at the study of other human complex traits. Visscher’s rule of ‘human trait exceptionalism’ could be phrased as:

All traits in all species follow the same theoretical laws and rules of evolutionary, population and quantitative genetics and are subject to the same experimental designs and analytical methods, apart from behavioral traits in humans.

I have never fully understood why such outstanding scientists such as Kempthorne, Jacquard and Lewontin so objected to and criticized the study of genetic variation in human behavioral traits with empirical data. At best, they were presenting themselves as guardians of human morality and defenders of the truth; at worst, their opinions were driven at least in part by political ideology. The present-day champions of anti-BG sentiment and writing seem to fall into the same exceptionalism trap, confusing what ‘is’ with what ‘ought to be’ (according to them), also known as the moralistic fallacy. These scientists may wish to remember that JBS Haldane, the famous and outstanding evolutionary and population geneticist (and devoted communist), refused to condemn Lysenko and Stalin, and thereby failed to defend Mendelian genetics (the truth), in a 1948 radio interview with RA Fisher and CD Darlington (Subramanian, 2019). Plant geneticist Vavilov, whom Haldane knew, died in a gulag in 1943, an early example of being cancelled (‘deplatformed’), literally. (Haldane later acknowledged that Vavilov was destroyed by ideology and regretted not defending him). Haldane also stayed closer to eugenics than is commonly believed (see the excellent book review from Brian Charlesworth, 2021). In true Orwellian style, at UCL in London, Haldane has multiple spaces named after him whereas there is no trace left of Galton’s scientific legacy. And Fisher’s stained-glass window tribute at Caius College in Cambridge was removed after protests by activists. Chillingly, I was at a major genetics conference in 2025 where a plenary speaker seemed to suggest that we should stop teaching Mendelian genetics at school because it gives a misleading view of genetic variation in human traits and disease. It is easy to imagine a dystopian future curriculum without Mendelian genetics, something along the lines of: all traits are so polygenic and so socio-environmentally context specific that it is impossible to quantify their effect sizes individually or cumulatively, and in any case environment-induced epigenetic effects are more important in explaining differences between people than effects due to DNA sequence differences. The leap (or creep) back to Lysenko (and Lamarck) is not hard to make. Note that even replacing the teaching of Mendelian genetics with polygenic inheritance is missing the main message of Fisher’s landmark 1918 paper on complex traits, the clue is in the title (Fisher, 1918).

As discussed by Morris, one frequent punchbag in anti-BG rhetoric is the utility of the estimation of genetic variation and the estimation of the population parameter ‘heritability’ in particular. Trait heritability is not the panacea for all scientific questions around trait variation in populations, but it can be very useful. It allows a unit-less comparison across traits within species, a comparison of the same trait between species, provides an upper limit for how much trait variation can ultimately be resolved by individual DNA variants, and provides an upper limit to prediction accuracy in

polygenic (genomic) prediction. Since genomes are established before phenotypes are observed, genetic variation provides a powerful anchor to study causality, even if causal genetic factors are mechanistically mediated through socio-environmental factors. The recent uses of molecular data to quantify and dissect the effect sizes of DNA variants between and within families, to quantify the imprints of assortative mating on the genome, and to test hypotheses of causation with instrument variable analyses are all surely interesting and robust ways to better understand the causes and consequences of trait variation in the human population.

Morris also mentions the criticism of GWAS for BG traits and the shifting goalposts (from it won't work to it works too well). This criticism is clearly neither logical nor fair, as my colleagues and I have tried to point out in a number of perspective-review articles in *American Journal of Human Genetics* (Abdellaoui et al., 2023; Visscher et al., 2012; Visscher et al., 2017). When exactly the same experimental designs and analyses methods are used for BG traits as for diseases and model traits such as height, and the conclusion is that the same certain cell types in the brain that are enriched in genetic variation for schizophrenia and other psychiatric disorders are also enriched in educational attainment (Yao et al., 2025), then how could one possibly argue that genetics does not matter or that the GWAS associations for such traits have nothing to do with biology and is mostly/all due to the environment?

In conclusion, Morris provides a 3-paper deconstruction of the idea that results from twin studies can be used to draw conclusions about free will, human agency and criminal responsibility. They are a long but necessary treatise on this topic because the erroneous conclusions have been used to undermine the scientific field of behavioral genetics. It is unfortunate and a missed opportunity that neither ET nor PH had the bandwidth to respond formally, but perhaps they will in the future using different media.

Acknowledgments. The outline of this paper was 'written' (composed in my head) while hiking the Tour du Mont Blanc in 2025. Yes, that's sad but it was a great hike. I thank Matt Keller and Loic Yengo for helpful comments.

References

- Abdellaoui, A., Yengo, L., Verweij, K. J. H., & Visscher, P. M. (2023). 15 years of GWAS discovery: Realizing the promise. *American Journal of Human Genetics*, 110, 179–194. <https://doi.org/10.1016/j.ajhg.2022.12.011>
- Charlesworth, B. (2021). *A Dominant Character: The Radical Science and Restless Politics of J. B. S. Haldane*. Samanth Subramanian, New York, NY: W. W. Norton, 2019. *The FASEB Journal*, 35, e21984. <https://doi.org/10.1096/fj.202101533>
- Fisher, R. A. (1918). The correlation between relatives on the supposition of Mendelian inheritance. *Transactions of the Royal Society of Edinburgh*, 53, 399–433.
- Harden, K. P. (2021). *The genetic lottery: Why DNA matters for social equality*. Princeton University Press.
- Kevles, D. J. (1998). *In the name of eugenics*. Harvard University Press.
- Lee, J. J., Wedow, R., Okbay, A., Kong, E., Maghzian, O., Zacher, M., Nguyen-Viet, T. A., Bowers, P., Sidorenko, J., Karlsson Linner, R., Fontana, M. A., Kundu, T., Lee, C., Li, H., Li, R., Royer, R., Timshel, P. N., Walters, R. K., Willoughby, E. A., . . . Cesarini, D. (2018). Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. *Nature Genetics*, 50, 1112–1121. <https://doi.org/10.1038/s41588-018-0147-3>
- Meyer, M. N., Appelbaum, P. S., Benjamin, D. J., Callier, S. L., Comfort, N., Conley, D., Freese, J., Garrison, N. A., Hammonds, E. M., Harden, K. P., Lee, S. S.-J., Martin, A. R., Martschenko, D. O., Neale, B. M., Palmer, R. H. C., Tabery, J., Turkheimer, E., Turley, P., & Parens, E. (2023). Wrestling with social and behavioral genomics: Risks, potential benefits, and ethical responsibility. *Hastings Center Report*, 53, 2–49. <https://doi.org/10.1002/hast.1477>
- Morris, D. (2025a). Behavioral genetics and human agency: How selectively deterministic theories of free will drive unwarranted opposition to behavioral genetic research and undermine our moral and legal conventions, Part I. *Twin Research and Human Genetics*, 28, 219–233. <https://doi.org/10.1017/thg.2025.22>
- Morris, D. (2025b). Behavioral genetics and human agency: How selectively deterministic theories of free will drive unwarranted opposition to behavioral genetic research and undermine our moral and legal conventions, Part II. *Twin Research and Human Genetics*, 28, 305–319. <https://doi.org/10.1017/thg.2025.10015>
- Morris, D. (2025c). Behavioral genetics and human agency: How selectively deterministic theories of free will drive unwarranted opposition to behavioral genetic research and undermine our moral and legal conventions, Part III. *Twin Research and Human Genetics*, 28, 385–400. <https://doi.org/10.1017/thg.2025.10016>
- Okbay, A., Beauchamp, J. P., Fontana, M. A., Lee, J. J., Pers, T. H., Rietveld, C. A., Turley, P., Chen, G. B., Emilsson, V., Meddens, S. F., Oskarsson, S., Pickrell, J. K., Thom, K., Timshel, P., de Vlaming, R., Abdellaoui, A., Ahluwalia, T. S., Bacelis, J., Baumbach, C., . . . Benjamin, D. J. (2016). Genome-wide association study identifies 74 loci associated with educational attainment. *Nature*, 533, 539–542. <https://doi.org/10.1038/nature17671>
- Okbay, A., Wu, Y., Wang, N., Jayashankar, H., Bennett, M., Nehzati, S. M., Sidorenko, J., Kweon, H., Goldman, G., Gjorgjieva, T., Jiang, Y., Hicks, B., Tian, C., Hinds, D. A., Ahlskog, R., Magnusson, P. K. E., Oskarsson, S., Hayward, C., Campbell, A., . . . Young, A. I. (2022). Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals. *Nature Genetics*, 54, 437–449. <https://doi.org/10.1038/s41588-022-01016-z>
- Rietveld, C. A., Conley, D., Eriksson, N., Esko, T., Medland, S. E., Vinkhuyzen, A. A. E., Yang, J., Boardman, J. D., Chabris, C. F., Dawes, C. T., Domingue, B. W., Hinds, D. A., Johannesson, M., Kiefer, A. K., Laibson, D., Magnusson, P. K. E., Mountain, J. L., Oskarsson, S., Rostapshova, O., . . . Hatteme, J. M. (2014). Replicability and robustness of genome-wide-association studies for behavioral traits. *Psychological Science*, 25, 1975–1986. <https://doi.org/10.1177/0956797614545132>
- Subramanian, S. (2019). *A dominant character: The science and politics of J.B.S. Haldane*. Simon and Schuster.
- Trut, L., Oskina, I., & Kharlamova, A. (2009). Animal evolution during domestication: The domesticated fox as a model. *Bioessays*, 31, 349–360. <https://doi.org/10.1002/bies.200800070>
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological Science*, 14, 623–628. <https://doi.org/10.1046/j.0956-7976.2003.psci.1475.x>
- Visscher, P. M. (2022). Genetics of cognitive performance, education and learning: From research to policy? *NPJ Science of Learning*, 7, 8. <https://doi.org/10.1038/s41539-022-00124-z>
- Visscher, P. M., Brown, M. A., McCarthy, M. I., & Yang, J. (2012). Five years of GWAS discovery. *American Journal of Human Genetics*, 90, 7–24. <https://doi.org/10.1016/j.ajhg.2011.11.029>
- Visscher, P. M., Hill, W. G., & Wray, N. R. (2008). Heritability in the genomics era — Concepts and misconceptions. *Nature Reviews Genetics*, 9, 255–266. <https://doi.org/10.1038/Nrg2322>
- Visscher, P. M., Wray, N. R., Zhang, Q., Sklar, P., McCarthy, M. I., Brown, M. A., & Yang, J. (2017). 10 years of GWAS discovery: Biology, function, and translation. *American Journal of Human Genetics*, 101, 5–22. <https://doi.org/10.1016/j.ajhg.2017.06.005>
- Walsh, B., Visscher, P. M., & Lynch, M. (2025). *Genetics and analysis of quantitative traits: Foundations*. Oxford University Press.
- Yao, S., Harder, A., Darki, F., Chang, Y. W., Li, A., Nikouei, K., Volpe, G., Lundstrom, J. N., Zeng, J., Wray, N. R., Lu, Y., Sullivan, P. F., & Hjerling-Leffler, J. (2025). Connecting genomic results for psychiatric disorders to human brain cell types and regions reveals convergence with functional connectivity. *Nature Communications*, 16, 395. <https://doi.org/10.1038/s41467-024-55611-1>