

influences like colorism; instead, the example of skin tone and colorism highlights that such research designs identify contextual causal processes which often operate through the sociocultural features of our world (and therefore may have low external validity). We agree with her point that GWAS “cannot disentangle genetic from environmental,” but the limitations are not only practical – they are conceptual. Burt’s distinction between upward and downward genetic causation privileges sociocultural processes as somehow ontologically and causally prior to genetic factors, which is equally mistaken as viewing genetic factors as ontologically and causally prior to environments. Ironically, in attempting to wrest some of the counterfactual effects of genes back into the environmental fold, Burt thrusts the conversation again into a phony horseshoe between genes and environments, wherein opposing sides engage in a bean-counting exercise over how much outcome variation counts as genetic. We’ve been there before; it’s an intellectual dead end.

Financial support. This work was partially supported by NHGRI grant T32HG008953 (D.O.M.) and the Center for Health and Wellbeing at Princeton University (S.T.).

Competing Interest. None.

References

- Domingue, B., Trejo, S., Armstrong-Carter, E., & Tucker-Drob, E. (2020). Interactions between polygenic scores and environments: Methodological and conceptual challenges. *Sociological Science*, 7, 365–386. <https://doi.org/10.15195/v7.a19>
- Heine, S. J. (2017). *DNA is not destiny: The remarkable, completely misunderstood relationship between you and your genes*. Norton.
- Holland, P. W. (1986). Statistics and causal inference. *Journal of the American Statistical Association*, 81(396), 945–960.
- Howe, L. J., Nivard, M. G., Morris, T. T., Hansen, A. F., Rasheed, H., Cho, Y., ... van der Zee, M. D. (2021). Within-sibship GWAS improve estimates of direct genetic effects. *BioRxiv*.
- Laidley, T., Domingue, B., Sinsub, P., Harris, K. M., & Conley, D. (2019). New evidence of skin color bias and health outcomes using sibling difference models: A research note. *Demography*, 56(2), 753–762. <https://doi.org/10.1007/s13524-018-0756-6>
- Martschenko, D., Trejo, S., & Domingue, B. W. (2019). Genetics and education: Recent developments in the context of an ugly history and an uncertain future. *AERA Open*, 5(1), 2332858418810516.
- Meyer, M., Turley, P., & Benjamin, D. (2020). Genetic scoring presents opportunity, peril. *The Wall Street Journal*. <https://medium.com/@michellenmeyer/response-to-charles-murray-on-polygenic-scores-e768cf145cc>
- Murray, C. (2020). Genetics will revolutionize social science. *The Wall Street Journal*. <https://www.wsj.com/articles/genetics-will-revolutionize-social-science-11580169106>

Polygenic scores and social science

Walter Veit^a  and Heather Browning^{b,c} 

^aSchool of History and Philosophy of Science, The University of Sydney, Sydney, NSW, Australia; ^bCentre for Philosophy of Natural and Social Science, London School of Economics and Political Science, London, UK and ^cDepartment of Philosophy, University of Southampton, Avenue Campus, Southampton, UK

wveit@gmail.com; <https://walterveit.com/>
DrHeatherBrowning@gmail.com; <https://www.heatherbrowning.net/>

doi:10.1017/S0140525X22002345, e229

Abstract

It is a hotly contested issue whether polygenic scores should play a major role in the social sciences. Here, we defend a methodologically pluralist stance in which sociogenomics should abandon its hype and recognize that it suffers from all the methodological difficulties of the social sciences, yet nevertheless maintain an optimistic stance toward a more cautious use.

It is a hotly contested issue whether polygenic scores (PGSs) and genome-wide association studies (GWASs) should play a major role in the social sciences. As described in the target article, what we see is both (over)hype and a staunch opposition, with harsh accusations thrown around, straw man arguments, and *ad hominem* attacks. All this makes it difficult to not only evaluate the positions, but even to ask important methodological questions about the potential uses of these genetic tools within the social sciences.

Here, Burt offers an elegant methodological target article with the aim of addressing just this problem. In it, Burt objectively criticizes the hype that has often accompanied heritability research, without committing any of the above sins, drawing attention to the methodological limits and challenges of adding genetics research to the social sciences. Although we agree with many of Burt’s points, however, we can’t help but feel that she ends up overstating her conclusions and overplaying the differences between sociogenomics and traditional research within the social sciences.

In her conclusion, Burt states that “GWASs and PGSs may be powerful tools for identifying genetic associations, but they are not the right tools for understanding complex social traits” (target article, sect. 9, para. 3). Naturally, we wholeheartedly agree. However, our reasons for accepting this claim aren’t a belief that these tools cannot at all help us to understand genetic influences or social outcomes, but rather that there is no such thing as *the* right tools for understanding complex social traits. That is, we do not think that there is some kind of unique or privileged combination of scientific tools for investigation of whatever complex social trait we are interested in, whether that is poverty, educational attainment, or criminal behavior. Let us elaborate.

As philosophers of science (and in particular, philosophers of the social sciences) have long recognized, complex phenomena are not to be understood through the competition of various methods with the aim of finding the ideal one, but rather through use of a broad range of tools that complement each other in various ways (Mitchell, 2009; Veit, 2019, 2021; Wimsatt, 2007; Ylikoski & Aydinonat, 2014). Although there are often conflicts within scientific disciplines regarding what sets of methods, models, experiments, and the like should be employed, these often appear to be driven by “indoctrination” into the methodology of a lab and ideological disputes over the correct methods. As the saying goes: If all you learn is how to swing a hammer, all problems will start to look like nails. But from a higher-level perspective, it is precisely because of the pluralism of different methods that science has flourished. And this conclusion, we think, likewise applies to the use of GWASs and PGSs.

These methods should not act as a replacement for standard social science tools, nor should they be seen as competitors to randomized controlled trials (RCTs) that investigate environmental

factors. Instead, we argue that they can provide us with a useful complement for research into the main targets of the social sciences, that is: complex causal systems with great heterogeneity and no strong generalizations. Just as the study of genome-wide associations bears the danger of falsely attributing causality to observed correlations, so too does standard social science. Burt is right in her criticism of the hype around PGSs: That they are often seen as deterministic, fail to control for a wide range of potential confounds, risk reviving the unfortunate gene-culture war, and so forth. But it is possible to arrive at such a critical stance by highlighting that sociogenomics will of course suffer from all the methodological difficulties of the social sciences – causal indeterminacy, the complexity of the social world, looping effects, and so forth. Within such an alternative picture, however, sociogenomics could still play a valuable role, within its own limited sphere.

Rather than simplifying the complexity of social phenomena, we argue that sociogenomics can help us to highlight how complex and causally interdependent social phenomena truly are. That is, we can buy into the main criticisms of the usefulness of PGSs in the social sciences, without being led to the strong conclusion that sociogenomics is methodologically doomed. Rather than returning to old and unhelpful discussions of social versus genetic causes, we think that sociogenomics might in fact help us toward a recognition of the complexity of our social traits and their myriad bases. This is how one should understand the argument that PGSs may improve RCTs by finding further variables to be controlled for (Harden, 2021). It's an embrace of a supplementary and pluralistic stance in the face of complexity. Rather than eliminating sociogenomics, or buying into the mistaken hype that it is going to replace and revolutionize standard social science, we can see its role instead as a complementary method to be added to the vast toolkit of the social sciences. Burt rightly points out that the methods as they are currently used too often fail to appreciate their own limitations, but this can be used as a starting-point, with these careful criticisms forming the basis for refining and strengthening the methods to better fit the contexts of use.

We therefore think that neither the majority of advocates nor the majority of critics of PGSs hold an adequate epistemic stance toward their use in the social sciences. Instead, we have here advocated for something of a mid-level approach, in which proponents of sociogenomics are urged to recognize the methodological difficulties of social science research and familiarize themselves with the philosophy of the social sciences in order to improve their own methods. Once the hype dies down, what remains will be better science, one practiced with adequate attention paid to the current problems and limitations of the methods. At the moment, without knowing exactly how this will unfold, we would like to avoid making any firm predictions regarding the likely payoffs of sociogenomics; however, we hold a (cautiously) optimistic stance regarding its future use.

Financial support. W.V.'s research was supported under Australian Research Council's Discovery Projects funding scheme (project number FL170100160).

Competing Interest. None.

References

Harden, K. P. (2021). *The genetic lottery: Why DNA matters for social equality*. Princeton University Press.

Mitchell, S. D. (2009). *Unsimple truths: Science, complexity, and policy*. University of Chicago Press.

Veit, W. (2019). Model pluralism. *Philosophy of the Social Sciences*, 50(2), 91–114.

Veit, W. (2021). Model diversity and the embarrassment of riches. *Journal of Economic Methodology*, 28(3), 291–303.

Wimsatt, W. C. (2007). *Re-engineering philosophy for limited beings: Piecewise approximations to reality*. Harvard University Press.

Ylikoski, P., & Aydinonat, N. E. (2014). Understanding with theoretical models. *Journal of Economic Methodology*, 21(1), 19–36.

Vertical pleiotropy explains the heritability of social science traits

Charley Xia^{a,b,*} and W. David Hill^{a,b,*} 

^aLothian Birth Cohort studies, University of Edinburgh, Edinburgh, UK and

^bDepartment of Psychology, University of Edinburgh, Edinburgh, UK

Charley.Xia@ed.ac.uk; <https://www.ed.ac.uk/profile/dr-charley-xia>

David.Hill@ed.ac.uk; <https://www.ed.ac.uk/profile/david-hill>

doi:10.1017/S0140525X22002382, e230

Abstract

We contend that social science variables are the product of multiple partly heritable traits. Genetic associations with socioeconomic status (SES) may differ across populations, but this is a consequence of the intermediary traits associated with SES differences also varying. Furthermore, genetic data allow social scientists to make causal statements regarding the aetiology and consequences of SES.

Burt describes the signal captured by a polygenic score (PGS) derived from a genome-wide association study (GWAS) on social science traits such as education as being “artificial” and a product of social differences rather than genetic processes. As an example of downward causation, Burt provides the thought experiment posed by Jencks et al. (1972) where, in a hypothetical scenario, red-headed individuals are denied access to an education.

We argue that, just as a PGS captures the aggregate effect of each individual single-nucleotide polymorphism (SNP) used in its construction, each SNP from a GWAS conducted on education captures the aggregate effect of each heritable trait associated with differences in education. This process, referred to as vertical pleiotropy (also known a mediator variable) describes incidences where phenotype A (e.g., intelligence) is associated with phenotype B (education) and so a genetic variant found to be associated with phenotype A will also be associated with phenotype B (Fig. 1).

In Burt's hypothetical example, red hair would emerge as an intermediary phenotype between genetic inheritance and phenotypic consequence but in real data, childhood intelligence ($r_g = 0.72$, $SE = 0.09$) (Hill, Davies, Liewald, McIntosh, & Deary, 2016), health ($r_g = 0.56$, $SE = 0.03$) (Hill et al., 2019b), attention-deficit/hyperactivity disorder (ADHD) ($r_g = -0.54$, $SE = 0.03$) (Hill et al., 2019b), and neuroticism ($r_g = -0.23$, $SE = 0.02$) (Hill et al., 2020) show consistent and substantial genetic correlations with education and give an indication as to what

*Both authors contributed equally.