All that Glisters is not Gold: Genetics in Social Science[1]

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Abstract

In their target article, Madole and Harden offer an account of "what it means for genes to be causes" of social outcomes to bolster their claim that genetics should be incorporated into social science with practical implications. Here I object to several key features of their arguments, their representation of the state of science, and claims about the utility of genetics for social science and society.

[1] Forthcoming commentary in *Behavioral and Brain Sciences*; comment on Madole and Harden's (2022) target article: Building Causal Knowledge in Behavior Genetics.

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In their target article, Madole and Harden (MH) provide a lengthy exposition of genetic causation to undergird their argument that social scientists not only can but should incorporate genetics into their research on human behavior. Pointing to the recent 'discovery of genes' associated with social outcomes and the potential utility for social science, the authors argue that careful analysis of what it means for genes to be causes of social behaviors is needed. I agree.

The devil is, as usual, in the details. With much to say and limited space, I first identify and set aside points of agreement between MH and me while highlighting key disagreements. I then discuss my objections to their arguments. With unlimited space, I would challenge methodological issues related to their model of genetic causation, e.g., violation of SUTVA, the non-generalizability of causes of sibling differences to causes of differences between unrelated people in the population, and so on. Here, I focus on what I view as more fundamental challenges.

Shared Understandings and Points of Departure

There is, in my reading, much upon which MH and I agree about the role of genetics in human behavioral differences. Our disagreement is rooted in what we can know about causes of human behavior, given the nature of development and given limitations of current methods and biological knowledge, and thus whether genetics is useful for social science. In particular:

- We agree that genetic differences matter for human social outcomes—achievements, behavior, physical health, personality—in a complex, context-sensitive way. We disagree that complex, social, non-disease achievements or behaviors, like educational attainment, having ever had same-sex sex, or income, are appropriate 'traits' for genetic study.

- We agree that studying processes that are malleable prognostic markers of human outcomes and thus targets for intervention to reduce disparities and improve health and well-being is valuable for science and society. We disagree that studying putative *genetic* linkages to these intermediate processes is necessary or useful, especially at the current state of the science.

- We agree that genetic research has the potential to advance understanding of human health and disease. We disagree that genetic research is gainfully employed to enhance knowledge on the etiology of complex human *social* behavior.

- We agree that those previous iterations of social science genetics employed in the service of 'scientifically' demonstrating the genetic inferiority of socially subordinate groups should be rejected as flawed and denounced. We disagree that the problems with social science genetics are rooted in political orientation or limited to ethical issues and thus solved by publicly repudiating genetic determinism and recognizing context-dependency and gene-environment interactionism. Crucially, as I outlined in my target article, the problems are not just political but are conceptual (e.g., Burt, 2022; Kaplan & Turkheimer, 2021), methodological (Coop & Przeworski, 2022; Morris et al., 2020; Richardson & Jones, 2019), and biological (Crouch & Bodmer, 2020; McClellan & King, 2010).

I do not doubt that MH have good intentions. But good intentions —including an explicit 'anti-eugenics' approach—are not enough.

Having sketched key sources of disagreement rooted in shared understandings, I turn to four issues that deserve response, challenge, and/or clarification.

Oversimplifications and Obfuscations

Throughout MH's target article, biological complexity is downplayed, and terminology is misleading, which obscures substantial difficulties and biological unknowns. For example, MH frame their study as being about 'genes' building on "the discovery of genes associated with human phenotypes like educational attainment and substance use disorders" (p.3). To be sure, given that the intended audience includes social scientists who lack genomic expertise including familiarity with genetic terminology, the gains in readability by referencing "genes identified" versus a more accurate, "genetic variants that are non-causal markers of some unknown causal SNPs likely in proximity" are substantial. However, this simplifying language is misleading. Many social science readers might reasonably interpret this use of 'genes' to indicate identified different versions of genes with defined functions that affect social outcomes through well-characterized biological pathways. The reality is nothing of the sort.[1] As I discuss at length in my target article, due to a host of limitations, current sociogenomics methodologies (i.e., GWASs and PGSs) are ill-suited for identifying specific "genes associated with" complex highly polygenic outcomes (Burt, 2022a; Charney, 2022; Kaplan & Turkheimer, 2021).

In glossing over the difficulty of biological interpretation, MH misrepresent the complexity and uncertainty in moving from risk loci to causal variants acting in genes with defined functions. They note that because "'genes' are studied at an intermediate level of resolution…Researchers can then use 'fine mapping' techniques to gain higher resolution" as if fine mapping were straightforward or unproblematic rather than highly sophisticated guesswork based on limited biological knowledge (see discussions in Burt, 2022; Charney, 2022; Crouch & Bodmer, 2020). Were fine mapping and gene identification so easy.

Irreducibly Social Behaviors Resist Genetic Reductionism

While their specific claims about utility are meager and usually vague, MH point the value of genetics to enhance social scientific understanding of "how genetic factors unfold along biological and behavioral pathways across development" (p.47). To be sure, the idea of tracing genetic pathways from molecules to behavior sounds compelling. In contrast to genetic diseases, however, tracing genetic variants to complex *social* behavioral differences is impracticable. Due to biological and conceptual limitations—including the fact that complex social traits are defined by social context and thus irreducibly social—mapping genetic variants to social outcomes is infeasible, even leaving aside statistical issues (e.g., environmental confounding, counterfactual model assumption violations) and ignoring the problem of misidentifying downward causation as upward genetic causation (see below).

Given this, the search for specific genetic causes of complex social (non-disease) traits like education and crime remains a misguided endeavor, even if our current methodologies facilitated such precise identification, which they do not.

[1] The authors' use of genetic disease examples with well-characterized biological pathways (cystic fibrosis) surely reinforces such simplified understandings.

To be clear, alleviation of this situation will not come from new genomic research tools, sophisticated statistical algorithms, larger sample sizes, or within-family studies. Complex social traits like educational attainment and crime, unlike cystic fibrosis and sickle cell anemia, are social traits not biological ones (Burt, 2022b; Dupre, 2012).

Lack of Utility

This inability to map specific (miniscule) genetic effects to social outcomes is, however, no great loss. We do not need genetics to "isolate intermediate processes that represent (a) prognostic markers of future outcomes and (b) targets for programmatic intervention" (e.g., early childhood eating habits, health behaviors, education-related behaviors) (MH, p.47).

Using BMI as a 'ready example' to illustrate the potential value of genetics for social science, MH note that studies linking genetic associations with 'phenotype annotation efforts' "have found that, by as early as age two, a child's eating behavior may demarcate genetic risk for adult [high] BMI" (p.48). Given what we know about the importance of early childhood and eating behaviors, do we need genetics to suggest that early childhood eating behaviors are important in shaping adult BMI? I think not. As I have argued elsewhere, the justification for incorporating genetics into social science to reveal well-established social patterns is lacking. This is particularly true since, as we all agree, *genetic does not mean unchangeable*, and however genetically influenced, environments always matter. The targets for interventions and policies are the intervening psychosocial mechanisms (diet, self-control) and or the environments (e.g., parenting) to reduce adverse health outcomes, reduce social inequalities, and enhance social flourishing.

In sum, from the fact that genetic differences matter for development and behavioral differences, it does not follow that incorporating measures of genetic differences (invariably imprecise and environmentally confounded) will advance social science models or policy.

Misidentifying Downward (Social) Causation as Upward (Genetic) Causation

Finally, even a rigorous counterfactual study of genetic causation cannot distinguish authentic ("upward") genetic causation (from genetic differences to trait differences through biological pathways) from downward social causation (e.g., Burt, 2022a). Although not now, hopefully one day we can all agree that a model that identifies downward causation as 'genetic' (and thus would identify darker skin pigmentation alleles as genetic causes of lower income shaped by socio-historical processes of racial/ethnic discrimination) is wrong and misleading.

In sum, while MH extoll the utility of these studies for social science and society, given limitations in what we know, can know, and can measure, what genetics offers social science remains vague, misguided, and/or overhyped. Moreover, this is to say nothing of the potential dark side, including reifying an oversimplified, flawed, and catchy notion of a 'genetic predisposition' for complex social traits stratified along class, racial/ethnic, and other axes of inequality.

In conclusion, complex social phenotypes like educational attainment are not biological phenotypes; human behaviors are irreducibly social and contingent; and their causes are heterogeneous, intertwined, and unable to be 'unbraided' by observational methods with even the best statistical genetic methodologies. Given this, the endeavor to identify "genes" or genetic causes of normal variation in educational attainment given both the current state of science and the nature of the

“phenotype” is misguided. Such endeavors also waste time, energy, and the skills of talented scientists, such as Madole and Harden.

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Acknowledgements: The author is grateful to Kara Hannula for helpful discussions, valuable feedback, and other support.

Funding Statement: Callie Burt is supported by funding from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (5K01HD094999).

Conflict of Interest Statement: I have no conflicts of interest.