



Research



Cite this article: Benning JW, Carlson J, Smith OS, Shaw RG, Harpak A. 2025 Confounding fuels misinterpretation in human genetics. *Proc. R. Soc. B* **292**: 20251615.

<https://doi.org/10.1098/rspb.2025.1615>

Received: 23 June 2025

Accepted: 8 September 2025

Subject Category:

Genetics and genomics

Subject Areas:

behaviour, genomics, evolution

Keywords:

confounding, human, genetics, social science
genomics, association studies, genomic prediction

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Electronic supplementary material is available online at <https://doi.org/10.6084/m9.figshare.c.8082266>.

Confounding fuels misinterpretation in human genetics

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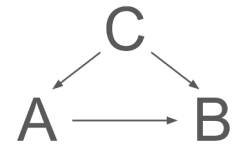
The scientific literature has seen a resurgence of interest in genetic influences on human behaviour and socioeconomic outcomes. Such studies face the central difficulty of distinguishing possible causal influences, in particular genetic and non-genetic ones. When confounding between possible influences is not rigorously addressed, it invites over- and misinterpretation of data. We illustrate the breadth of this problem through a discussion of the literature and a reanalysis of two examples. The first paper we discuss suggested that patterns of similarity in social status between relatives indicate that social status is largely determined by one's DNA. Our reanalysis shows that the paper's conclusions are based on the conflation of genetic and non-genetic transmission (for example, of wealth) within families. The second paper we discuss posited that genetic variants underlying bisexual behaviour are maintained in the population because they also affect risk-taking behaviour and thereby confer an evolutionary fitness advantage through increased sexual promiscuity. In this case, too, our reanalysis shows that, though possible explanations cannot be distinguished, only one is chosen and presented as a conclusion. We discuss how issues of confounding apply more broadly to studies that claim to establish genetic underpinnings to human behaviour and societal outcomes.

1. Introduction

People vary remarkably in behaviour and social outcomes. This variation sparks curiosity about its causes, and for the past 150 years, scholars have debated the extent to which it arises owing to underlying genetic differences. In the nineteenth century, Galton [1] found strong resemblance between parents and their offspring in measures of social status and, on that basis, inferred that genetics is the most likely root cause, a school of thought described broadly as 'hereditarianism' [2]. As is now well appreciated, Galton's inference neglected the fact that parents transmit not only genetic material to their offspring, but also wealth, place of residence, knowledge, religion, culture and more. For such attributes, transmission within families can parallel genetic transmission (figure 1a; [4–21]), often leading genetic and non-genetic transmission to be indistinguishable in observational data. A long history of scholarship has highlighted this type of confounding and how it impedes inference of the causes of phenotypic variation [22–30]. Studies without molecular genetic data are particularly susceptible to confounding, because they offer little to no signal that could be argued to reflect only genetic or only non-genetic transmission [22,23,26,28,31].

Box 1: What is confounding?

Consider the hypothesis that a change in variable A causes a change in variable B, denoted $A \rightarrow B$. Another variable, C, that affects both A and B, is called a confounder: it renders the effect of A on B indistinguishable from the effect of C on B. In the presence of confounding, one cannot reliably estimate causal influences [32, 33].

**Confounding in data**

Sometimes, variables that are not confounders in the population can still act as confounders in a dataset. This can happen when the structure of the data prevents variation in one variable while holding another constant—so even if C does not cause A in the population, in the dataset, one cannot vary A independently of C. Confounding can also arise when multiple causal factors (e.g., A_1 and A_2) are aggregated into a single variable A. If only A is measured, and A_1 is interpreted as the causal driver, associations arising from A_2 may be mistakenly attributed to A_1 (this is one mode of what is sometimes termed “proxy confounding” [34, 35]). In both cases, the structure of the data—rather than solely the causal mechanisms that exist in the population—determines what is confounded.

Modes of confounding discussed in this work

case discussed in this manuscript	(A) putative cause	(B) outcome	(C) confounder
i. familial transmission (Clark [3]; figure 1a)	genetic variants	social status	non-genetic factors transmitted within families, such as wealth, community, and culture these influence covary with genetic variants across individuals, forming a single, inseparable ‘transmissible component’. Thus, this is a form of proxy confounding: even though the non-genetic factors do not influence genotypes in the population, the two are bundled together in the same variable (individual identifier) in the data
ii. genetic confounding (figure 1b) in a GWAS for height	focal genetic variant	height	other genetic variants influencing height that are correlated (in “linkage disequilibrium”) with the focal variant.
iii. environmental/social confounding (figure 1b) in a GWAS for asthma	focal genetic variant	asthma	environmental or social influences on asthma, such as exposure to pollutants, that correlate with the focal variant
iv. analytical confounding via choice of hypotheses to consider (Song and Zhang [36]; figure 3a,b)	effects of genetic variants, associated with bisexual behavior, on risk-taking behavior	effects of these genetic variants on number of children (as a proxy for evolutionary fitness)	effects of these variants on traits and outcomes other than risk taking (none were considered in the study)

In observational studies, confounding may be insurmountable

Pearl’s backdoor criterion [34, 37] provides a formal way to estimate the causal effect of A on B in observational data: we must block all “backdoor paths”—paths that connect A and B through common causes rather than through the effect of A on B—by conditioning on common causes of both A and B, while avoiding conditioning on variables affected by A.

Applying this criterion can be difficult or impossible in observational studies. One reason is that we may not observe, or know of, the relevant confounding variables. Even when we do, these variables may be, in addition to affecting A, affected by A themselves, meaning that we cannot apply Pearl’s backdoor criterion.

2. Confounding fuels hereditarian fallacies

A recent publication [3] analysed familial correlations in a dataset of socioeconomic measures (e.g. occupational status, house value, literacy) from a selection of records spanning the eighteenth to twenty-first centuries in England. In it, a quantitative genetic model is fitted to these observed correlations ([38,39]; electronic supplementary material, note S1). Based on this fit, [3] infers that social status persists intergenerationally because of strong assortative mating on a status-determining genotype (or ‘social genotype’ as the author has termed it in previous work [40]). Further, the paper argues that the persistence of social status within families—and persistence of differences in status among families—have been largely unaffected by changes in social policy in the last four centuries. In a subsequent commentary about this work [41], the author presents the results of [3] as providing strong support for a hereditarian interpretation. In doing so, he appeals to the metaphor of a ‘genetic lottery’ underlying social outcomes.

Here, we discuss the failure to account for the confounding of genetic and non-genetic transmission (figure 1a) that, together with other core flaws of the analysis (figure 2a,b), fuels the hereditarian claims in [3] (see our discussion of other misinterpretations, errors and incongruencies in [3] in the electronic supplementary material, notes S2–S7, tables S1–S3, and figures S1–S13). We also demonstrate that familial status correlations varied substantially over the time period examined, generally decreasing

(figure 2c). This finding contrasts with conclusions in [3], based on the same data, that social mobility has been stagnant. As we show below, the analyses in [3] do not establish the contribution of genetics to social status.

(a) Confounding between genetic and non-genetic transmission

Inferences in [3] are based on a linear regression model derived from quantitative-genetic theory developed by R.A. Fisher ([38,39]; electronic supplementary material, note S1) and the model

$$P = G + E, \quad (2.1)$$

where an individual's phenotype, P , is the sum of separable genotypic (G) and environmental (E) influences on it. Because genes are transmitted from parents to offspring, genetic parameters can be inferred from correlations between relatives, on the condition that environmental influences are random and independent of genotypes. Fisher [38] formally showed that under this model, the expected correlation in a trait between pairs of individuals of a defined relationship is a function of the genealogical relationship between the relatives, the trait's heritability (h^2), and the extent of assortative mating in the population (b). (h^2 is the fraction of phenotypic variance due to additive genetic variance, commonly referred to as 'narrow-sense' heritability.)

Crucially, to interpret the model parameters h^2 and b as relating to genetic effects, Fisher's model assumes that there are no non-genetic (material, environmental or cultural) influences on a trait that are systematically shared or transmitted between relatives. This assumption is valid in an experimental setting, for instance, in which genotypes are randomized with regard to environment. However, in humans that assumption is nonsensical. Non-genetic transmission is ubiquitous for social and behavioural traits. Traits may be transmitted directly between relatives (e.g. literate parents teaching their children how to read; [6]), or via indirect mechanisms such as 'ecological inheritance,' where the trait value of an offspring is influenced by the environmental conditions bestowed by their parents (e.g. familial wealth influencing educational opportunities; [9,42]). When genotypes cannot be randomized over environments, true genetic effects are much more difficult to separate from other factors underlying phenotypic resemblance between relatives [23]. In [3], for instance, the assumption of no systematic non-genetic transmission implies that similarity in house value among relatives (one of the measures of social status analysed) is solely owing to shared genes, and does not arise from similarity in parental wealth, the inheritance of wealth or property, or having learnt from one's relatives about investment.

In fact, we found evidence of strong confounding between genetic and non-genetic contributions to familial resemblance in the data used in [3]. The paper acknowledges the inheritance of material wealth from one's parents as an example of non-genetic transmission only when treating wealth itself as the focal status measure. For other measures studied, the effect of familial wealth on social status is ignored. However, familial wealth can obviously influence a wide range of conditions that affect offspring (e.g. healthcare, place of residence, access to tutors, social circles, etc.; [43–47]). Consistent with this intuition, we found that all seven status measures analysed in [3] are substantially correlated with paternal wealth (Pearson's r ranging from 0.19 to 0.66; mean $r = 0.36$; all $p < 2 \times 10^{-16}$; electronic supplementary material, table S2; figure 2a). Closer relatives tend to have more similar paternal wealth, and the similarity in paternal wealth between relatives predicts their similarity in occupational status extremely well (Pearson's $r = 0.91$; figure 2a). Thus, the effect of transmission of genes and that of parental wealth on familial similarity in social status are confounded in these data. Apart from wealth, numerous other non-genetic factors may contribute to familial correlations [48,49]. Two post hoc analyses are presented in [3] in an attempt to rule out non-genetic contributors to familial resemblance in social status. In the electronic supplementary material, note S4, we detail why these analyses are uninformative as to the strength of non-genetic effects on resemblance in social status between relatives (electronic supplementary material, figure S1).

This confounding invalidates the interpretation of the model parameters offered in [3] as pointing to identifiable genetic contributions (electronic supplementary material, note S1). In particular, in the presence of such confounding, the interpretation of G and E in equation (2.1) as transmissible genetic (*heritable*) and random non-genetic effects on a phenotype, respectively, no longer holds. Instead, they can be interpreted as a *transmissible* component and a random, non-transmissible component. Consequently, the parameter interpreted in [3] as narrow-sense heritability, h^2 , is in fact an estimate of the 'total transmissibility' of a trait, t^2 , the proportion of trait variance attributable to an unknown compound of transmissible influences on the traits, including genes, culture, wealth, environment, etc. [11,13]. The second key parameter, m , which [3] interpreted as the 'spousal correlation in the underlying genetics,' does not represent a genetic correlation between mates. It is instead the spousal correlation in the transmissible component of the trait. m is derived from the 'intergenerational persistence rate,' $b = \frac{1+m}{2}$, estimated from the regression model. The expected correlation for a given kinship pair is equal to $t^2 b^n$, where n denotes genealogical distance (figure 1a). Note that the parameterization of b for father–son and grandparent–grandchild relationships also depends on the degree of assortative mating with respect to the focal trait itself; see the electronic supplementary material, note S1. The conflation of genetic and non-genetic transmission helps to explain why the model parameters estimated in [3], which are claimed to represent quantitative genetic parameters h^2 and m , are much higher than estimates of these same parameters from studies that strive to account for confounding (e.g. [20,50,51]).

Conclusions in [3] about the insensitivity of social standing to policy and sociopolitical context rest on the similarity of estimates of the parameter b across status measures and across time. The claim [3] that this stability is owing to strong assortative mating on a genetic factor for 'social ability' does not hold, given that both genetic and non-genetic factors are transmitted within families. The estimate of m tells us nothing about genetic versus non-genetic contributions to assortment, and b tells us nothing about the cause of within-family persistence of social status (figure 1a; electronic supplementary material, note S1).

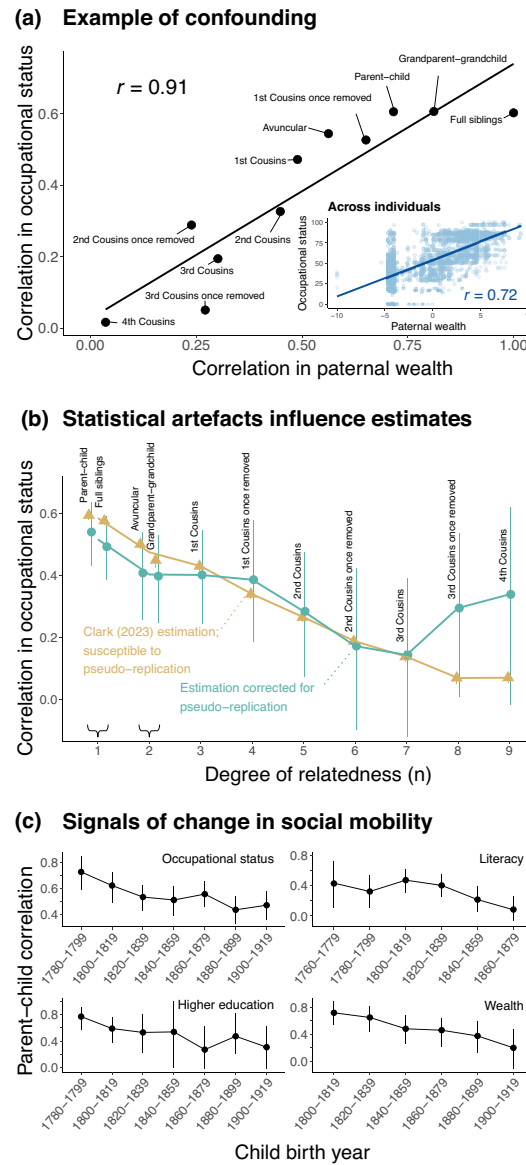


Figure 2. Reanalyses of data from Clark [3] challenge the paper's claims. (a) Example of confounding between genetic and non-genetic transmission. The relationship between paternal wealth and a measure of social status suggest at least one source of confounding between genetic and non-genetic transmission. Across relative pairs, correlation in occupational status is highly correlated (Pearson's $r = 0.91$) with those relatives' correlation in paternal wealth. Inset shows that individual occupational status is strongly correlated (Pearson's $r = 0.72$) with father's wealth. Excluding outliers in paternal wealth ($1.5\times$ interquartile range rule) had only a small impact (a change of less than 0.01) on the Pearson correlations reported here. Plots show data for individuals born 1780–1859 and their fathers. For the cohort of individuals born after 1859, the correlations are $r = 0.72$ for relative pairs and $r = 0.60$ for individuals. Clarke [3] estimated wealth from probate records. Log wealth at death was mean-centred by subtracting the mean log wealth for each decade. Individuals not probated owing to insufficient wealth were assigned a value of half the minimum probate requirement for the time period. (b) Pseudoreplication distorted estimates of familial correlations. Familial correlations (95% confidence interval (CI)) in occupational status (1780–1859) using the approach employed by [3] (in gold) involved pervasive, non-uniform pseudoreplication (electronic supplementary material, note S6). For example, the (1780–1859) occupational status correlation for fourth cousins is calculated from 17 382 pairs, derived from only 1878 unique individuals. In teal we show conservative estimates using only a single relative pair per surname (means and 95% CI over 1000 bootstrap samples are plotted for each familial correlation), which are therefore not susceptible to pseudoreplication. Distant cousins show dramatically higher correlations after adjusting for pseudoreplication. We note that additional biases may exist that are not addressed with this adjustment (electronic supplementary material, note S6). (c) Signals of change in social mobility. Parent-offspring correlations in multiple status measures generally decrease over time in Clark's [3] data, in contrast to claims of stagnant social mobility made in the original paper. To mitigate pseudoreplication, we calculated correlations using one pair from each surname (as in (b)). Shown are average correlations (95% CI) across 500 bootstrap iterations of correlation estimation. The electronic supplementary material, figure S13 shows two complementary analyses estimating correlations either without accounting for pseudoreplication, or using percentile ranks—both result in similar trends.

Regardless of whether owing to genetic causes or not, a striking claim in [3, pg 1] is that 'The vast social changes in England since the Industrial Revolution, including mass public schooling, have not increased, in any way, underlying rates of social mobility'. In point of fact, we found that the estimates of familial correlations in [3], and, in turn, estimates of the persistence rate, are heavily affected by statistical artefacts (figure 2b; electronic supplementary material, note S6). Furthermore, we show that across status measures, parent-offspring correlations—an established measure of social mobility [52,53]—generally decrease over time (figure 2c; electronic supplementary material, note S7). How could the new measure, 'persistence rate', used by [3] lead to such contrasting conclusions? Clark [3] offers neither justification for why this measure reflects social mobility, nor explanation for the discrepancies with established measures of mobility used in other literature (e.g. [54]) and applied to the same data.

Some readers have already taken arguments in [3] as compelling evidence that social status is largely caused by genetic factors [55–59]. Yet the assumptions and interpretations in [3] ignore a century of quantitative-genetic theory, previous empirical evidence for confounding, and the fallacies that arise when confounding is ignored [14,18,22,23,27,49,60–67], as well as patterns in the paper's own data that conflict with the interpretations presented. In this regard, we emphasize that [3] does not merely overstate the findings: the model parameters are misconstrued and the pervasive confounding of genetic and non-genetic transmission is not addressed.

3. Are modern genomic studies less susceptible to confounding?

In relying solely on observational phenotypic data and assuming that transmission in families is solely genetic, [3] is similar in spirit to studies carried out by Francis Galton a century and a half ago. One might hope that the inferential flaws described above are remedied in studies that use large genomic datasets and employ state-of-the-art statistical methods to adjust for confounding. As we outline, however, the same concerns remain broadly applicable; confounding is still poorly understood and often underplayed in the literature.

(a) Confounding in genomic studies is poorly understood

Human geneticists have long appreciated that there are myriad ways by which a genetic variant may be associated with a trait or outcome [27,68,69]. A key example is 'population stratification' in genomic data (e.g. in GWAS) wherein patterns of genetic similarity in a sample are correlated with the phenotype studied (figure 1b). Possible reasons for this correlation include social, environmental (box 1iii) or genetic (box 1ii) factors, contemporary and historical. Typically, the specific causes are unknown. These same axes of genetic similarity ('population structure') are reflected in the frequencies of numerous genetic markers that may be tested for association with a trait in a GWAS. Consequently, any such markers will tend to be correlated with the trait, even if only a subset (or in fact none) of the variants causally affect it (figure 1b).

Consider, for example, a GWAS conducted to identify genetic risk factors for asthma in a sample of people from the United States (US) of either primarily European American genetic ancestry or primarily African American genetic ancestry. There are many millions of variants in the genome that significantly differ in frequency between these groups. At the same time, African Americans in the US are systematically exposed to higher levels of air pollution [70], an environmental risk factor for asthma. If confounding is not adequately addressed, the GWAS would lead us to conclude erroneously that 'African American genetics' predispose one for asthma (box 1iii). Regardless of what drives population stratification, it can result in biased estimates of the individual effects of numerous genetic markers that tag these axes of population structure (figure 1b).

Human geneticists use various methods to adjust for confounded associations. However, confounding may persist, despite application of these methods ('residual confounding'). In 2019, we and other researchers discovered that genetic effect estimates in the largest GWASs for height—the most extensively studied polygenic trait—were biased owing to residual confounding [65,66]. It is plausible that this confounding is, at least in part, 'genetic confounding': the effect estimated for each individual variant was biased by the effects of many other variants (box 1iii). Regardless of the source of confounding, it became clear that while the bias for each individual genetic variant was slight, it was systematic across variants. Consequently, when researchers summed over signals from many genetic variants, they also summed over systematic biases. This led to erroneous conclusions in many studies (as detailed in [18,65,66]). Further research has suggested that residual confounding may affect many GWASs, in particular for social outcomes and traits that are heavily influenced by social context [27,67,69,71–73].

Even so, it is often impossible to explicitly demonstrate a lack of statistical identifiability or to pinpoint the specific confounders in a study. To our knowledge, no general method exists for the detection and quantification of confounding in GWAS data. In the absence of an omnibus litmus test for confounding, the answer cannot be to reduce the burden of proof for causal narratives.

(b) Confounding in genomic studies is downplayed

Papers often imply that confounding is completely remedied by current methods, despite evidence to the contrary [65–67,69,71,73–83]. Some methods to estimate genetic parameters have grown in popularity even after they were shown to be susceptible to confounding, with this susceptibility rarely mentioned as a caveat (see discussions in [65,72,73,77,84,85]). In other cases, confounding is acknowledged as a potential limitation, but its impact on the reported results (and their interpretation) is downplayed or obscured (see for instance, [86,87]).

As one example, consider the reporting of evidence for genetic effects from standard GWASs versus family-based studies. Family studies identify genotype-trait associations within, instead of among, families. This approach greatly mitigates many sources of confounding [67,69,88]. Family studies have yielded estimates of genetic effects on behaviour or social outcomes that are substantially weaker than those estimated from standard GWASs [50,67,71,73,89–92]. Reporting practices tend to downplay this point by using evidence from family studies as support for *existence* of a causal genetic effect, while continuing to rely on the magnitude of effects estimated using standard GWAS [62]. Such reporting choices mislead by presenting signals susceptible to confounding as measures of genetic causality.

(c) Confounding in complex traits genetics: aggregation of many small biases

Quantitative geneticists acknowledge residual confounding as an unsolved problem. However, in practice, researchers face incentives to publish their inferences of genetic associations that are vulnerable to confounding. With polygenic (or ‘complex’) traits, the usual focus of these studies, the genetic contribution to trait variation is in large part composed of numerous genetic variants with individually small effects. Researchers often wish to leverage weaker and weaker genetic associations to capture these highly polygenic signals. At the same time, confounding tends to be aggravated as more weakly associated variants are considered [67,81]. Thus, in the pursuit of understanding polygenic effects, researchers may face a trade-off between explaining a smaller part of the phenomenon under study in a causally rigorous way, versus accounting for a seemingly larger part, at the price of unknown biases introduced by confounding.

An example of this trade-off lies in genetic trait prediction with so-called ‘polygenic scores’ [93]. Polygenic scores based on more variants, including weakly-associated ones, may be preferred by researchers because they often attain higher prediction accuracy than polygenic scores that are limited to confident associations. However, polygenic scores that include many weakly-associated variants are plausibly more susceptible to underappreciated axes of confounding [67,81,94]. Subsequent ‘consumers’, including clinicians, researchers, policymakers and the general public, may then assume these polygenic scores capture strictly direct genetic effects; the possibility of confounding is rarely acknowledged. Consider, for instance, a hypothetical pre-implantation genetic test using a polygenic score based on the asthma GWAS we described above. In this extreme, embryos would mistakenly be prioritized for implantation according to whether or not they share genetic variants with people exposed to higher levels of air pollution in a previous generation.

Similarly, popular methods to estimate genetic correlations (the correlation between two groups of individuals in genetic effects on a trait, or the correlation of genetic effects on two traits) often indiscriminately aggregate across genome-wide associations [95,96]. Such methods are useful for characterizing how the genetic bases of complex traits are intertwined. However, they may inadvertently mask (or even introduce) additional axes of confounding. For example, confounding by population stratification that is shared between the two groups or two traits, or confounding with genetic effects on a third trait, can lead to biases in genetic correlation estimates [67,77,84,97,98]. The use of genetic correlations in causal inference (e.g. in ‘Mendelian Randomization’ [99–101]) therefore remains controversial [77,102]. However, when a study reports conclusions based on *genetic correlations*, it is likely to be interpreted—particularly by non-experts—as unambiguously reflecting genetic causality.

4. Further confounding introduced via researchers’ choices

Such unknown axes of confounding are plausibly a concern in a recent study that, based on an analysis of genetic correlations, purported to resolve an evolutionary paradox: why alleles associated with same-sex sexual behaviour are maintained, despite being ‘reproductively disadvantageous’ [32]. In what follows, we focus on what we refer to as ‘analytical confounding’, where potential confounding is worsened, or even introduced, through researchers’ analysis choices. In [32]: confusing model assumptions with evidence, ignoring the compatibility of data with confounded explanations, and introducing confounding through researchers’ classification choices. We posit that, while these problems are not unique to genomic studies, they can evade attention when couched in reports about how behaviours and outcomes are *genetically correlated*.

(a) Confusion of assumptions with evidence

In [32], a measure of bisexual behaviour is defined based on questionnaire data about total lifetime number of sexual partners and same-sex sexual partners (hereafter, we refer to this measure as BSB; see [103] and [104] for discussion of shortcomings of such measures). The paper reports a significant positive genetic correlation between BSB in males and the number of children. However, when adjusting this genetic correlation for genetic correlations of each measure with self-assessment as a ‘risk-taker’, the adjusted (or ‘partial’) genetic correlation between BSB in males and number of children was statistically indistinguishable from zero (figure 3a). It presents an interpretation of this finding as evidence that ‘the current genetic maintenance of male BSB is a byproduct of selection for male risk-taking behaviour.’ No explanation of the hypothesized mechanism by which risk-taking behaviour increases the number of offspring is given in [32], but in subsequent news coverage, one of the authors is quoted, ‘self-reported risk-taking [likely] includes unprotected sex and promiscuity, which could result in more children’ [105].

The study presents the contrast between these unadjusted and partial genetic correlations as support for a causal claim. However, the causal model is assumed *a priori*, and no evidence supporting this model is provided. Even under the assumption that the three measures considered are the only ones at play—and some causally affect others—the evidence is equally consistent with contradictory causal hypotheses (e.g. different directions of causality, arrows in fig. 2b of [32] and figure 3a; electronic supplementary material, figure S14 here; electronic supplementary material, note S8; c.f. [106]).

Furthermore, the study did not evaluate the support for any alternative model involving other factors, observed or latent. The authors justify their focus on risk-taking as a mechanistic explanation for the genetic maintenance of same-sex sexual behaviour by citing previous reports of genetic correlations of same-sex sexual behaviour and risk-taking [107,108]. However, these studies (and others [109]) reported multiple measures with similar (or even stronger) genetic correlations with same-sex sexual behaviour than risk-taking ([107]; electronic supplementary material, table S5). Sexual behaviour aside, [32] neither cites

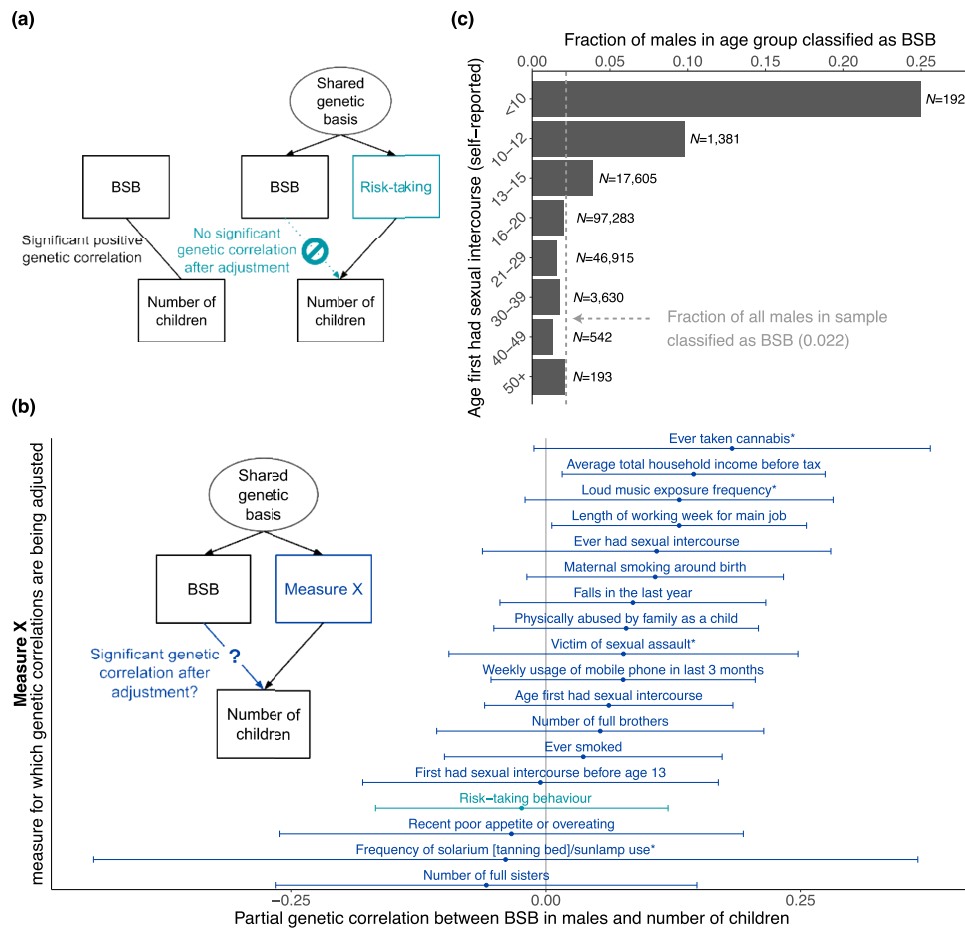


Figure 3. (a) Song & Zhang [32] show that the estimated genetic correlation between BSB (a measure of bisexual behaviour) in males and number of children is significantly different from zero (left diagram). They hypothesized the causal structure shown in the right diagram: genetic variants affecting BSB affect the number of children only through their simultaneous effect on risk-taking behaviour. When adjusting for genetic effects on risk-taking behaviour, the partial (residualized) genetic correlation between BSB and number of children is no longer significantly non-zero. They take this observation as evidence for their hypothesis. (b) When we repeat this analysis but replace risk taking with a variety of other measures ('measure X', blue in causal diagram), 16 out of 18 measures we examined yield a partial genetic correlation between BSB and number of children that does not significantly differ from zero (bars show 95% confidence intervals). Therefore, the data are equally consistent with the hypothesis that genetic variants driving BSB are maintained through evolution as a byproduct of selection on any of these 16 measures. We note that most of these measures—like risk-taking behaviour—have a significant partial genetic correlation with number of children. Four of the measures, denoted with an asterisk, are not. (c) Male participants in the study sample who reported having first had sex before the age of 13 (including victims of childhood sexual assault, many of which would have had same-sex perpetrators) are likelier to be classified as BSB by the criteria used in Song & Zhang [32]. *N*, total number of males in each group.

nor offers any evidence for the alleles associated with risk-taking being maintained over an evolutionary timescale. Additionally, this association is based on an answer to a single questionnaire question, 'Would you describe yourself as someone who takes risks?' [110, pg 5]. It is possible that responses to this question reflect a tendency towards practising unprotected sex and promiscuity, and that they simultaneously correlate with risk-taking tendencies that have been relevant for fitness throughout recent human evolution and across evolving societies; but, as acknowledged in [32], these key assumptions are hard to evaluate.

For these reasons, we asked: is there unique support for the assumed mechanistic model, in particular the role of risk-taking behaviour as a mediator? To answer this question, we considered models wherein a measure other than risk-taking mediates the genetic correlation between BSB and number of children ('measure X' in blue in figure 3b). If adjusting for this measure also results in a partial genetic correlation between BSB and number of children that is not significantly different from zero, the data are equally compatible with the hypothesis that there is a reproductive advantage for BSB-affecting alleles because of their simultaneous effect on measure X. We implemented this strategy with 18 measures, selected based on prior evidence of high genetic correlations with same-sex sexual behaviour, risk-taking behaviour and/or number of children ([109]; electronic supplementary material, tables S4, S5, note S8). All but two of these models yielded a partial genetic correlation between BSB and number of children that was not significantly different from zero (Genomic SEM [111] $p > 0.05$ before applying any correction for multiple testing; figure 3b). Hence, other causal narratives that do not involve risk taking could just as easily be constructed: the data are equally consistent with the hypothesis that genetic variants driving BSB are maintained through evolution as a byproduct of selection on the number of falls in the past year, usage of cell phone or any of these measures (figure 3b; electronic supplementary material, figure S15). The ease with which alternative hypotheses about genetic causality receive support using the method from [32] is revealing: the conclusion is highly reliant on the choice of a single hypothesis to test and a failure to consider confounding (box 1iv).

(b) Confounding introduced by researchers' classification choices

Measures relating to having experienced some form of nonconsensual sex ('victim of sexual assault' and 'first had sex before age 13') exhibited some of the strongest genetic correlations with BSB in males (electronic supplementary material, figure S15). This observation led us to be concerned about the ascertainment choices made in [32]. Indeed, we found that classification as a BSB individual is highly enriched among males who reported having first had sex before the age of 13 (in this regard, we note that children under this age cannot legally consent to any sexual activity in the UK [112]; figure 3c). Whereas 2.2% of males in the sample considered were classified as 'BSB individuals,' this classification rate was 9.8% among those who reported first having sex between the ages of 10–12 (inclusively), and 25% among those who reported first having had sex before the age of 10. For this dataset, there is no information about the age at which males classified as BSB first had same-sex sexual intercourse, or what fraction of victims of sexual assault had a perpetrator of the same sex. It is, however, established that the majority of reported sexual assaults on prepubescent male victims are carried out by male perpetrators [113–116]. This aggravates the concern that the BSB classification used in [32] conflated voluntary sexual behaviour and sexual assault, undermining the study's stated aim of advancing our understanding of human sexual preferences. Taken together, our reanalysis of [32] cautions yet again against causal inference based on preferential attention towards sensational hypotheses and analyses that seemingly support them.

5. Conclusion

The study of the genetics underlying human behaviour and social outcomes, with its fraught history and heightened potential for misinterpretation and misappropriation [62,63,72,103,117,118], demands the utmost rigour. The failure to reckon with confounding fuels misinterpretation of genetics research and impedes scientific progress. We are therefore concerned that a publishing culture which rewards sensationalism may instead promote a decline in standards [119,120]. In that respect, everyone has a role to play: it is crucial that researchers, reviewers and editors uphold the highest standards in their handling of these complex, far-reaching issues.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. All code for reproducing our analyses is available on Zenodo [121].

Supplementary material is available online [122].

Declaration of AI use. Artificial intelligence tools (Claude, ChatGPT) were used as coding assistants to build and check functions, unit test, format code, and improve documentation. All code was independently verified by the authors.

Authors' contributions. J.W.B.: conceptualization, formal analysis, investigation, writing—original draft, writing—review and editing; J.C.: conceptualization, formal analysis, investigation, writing—original draft, writing—review and editing; O.S.S.: formal analysis, investigation, writing—review and editing; R.G.S.: conceptualization, writing—original draft, writing—review and editing; A.H.: conceptualization, formal analysis, investigation, writing—original draft, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. The work was funded by NSF grant DBI-2010892 to J.W.B., NSF Graduate Research Fellowship DGE 2137420 to O.S.S., and NIH grant R35GM151108 and a Pew Scholarship in the Biomedical Sciences to A.H.

Acknowledgements. We thank Ipsita Agarwal, Kate Antonovics, Mark Borrello, Raj Chetty, Graham Coop, Doc Edge, Sasha Gusev, Kelley Harris, Mark Kirkpatrick, Magnus Nordborg, Nick Patterson, Molly Przeworski, Sohini Ramachandran, Noah Rosenberg, James Schmitz, Elizabeth Thompson, Elliot Tucker-Drob, Alex Viloria-Winnett and the Biological Interest Group of the Minnesota Center for Philosophy of Science for comments on the manuscript and helpful discussions. We thank anonymous reviewers for their critiques that helped improve this manuscript. We thank Gregory Clark for providing us with a corrected version of the occupational status data. We thank Siliang Song and Jianzhi (George) Zhang for providing us with detailed methods to replicate the GWAS they had performed. This research has been conducted using the UK Biobank Resource under Application Number 92741.

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