

## Genetics

### Ethics of Genetic Research on Same-Sex Sexual Behaviour

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**The ethical basis for research into the genetics of sexuality is not straightforward. A new study by Zietsch et al. investigates a hypothesis for the evolutionary basis of same-sex sexual behaviour. This increases our understanding of the genetics of complex behaviour, raising questions for whether and how such knowledge should be used.**

Studying the genetic basis of human sexual orientation has been fraught—socially, politically, and ethically. In the late 1800s, pioneering psychiatrists, bent on establishing their medical specialty, adopted the term ‘homosexual’ to describe a group of people seen as deviant: needing explanation, diagnosis and cure. Heterosexuals seemed to need no explanation, their patterns of sexual attraction and behavior taken to be natural and inevitable.[1] As the gay rights movement gained steam last century, search for a ‘gay gene’ became entwined with well-intentioned but ultimately fallacious moral arguments: if same-sex attraction was a matter of genetics, then gay people were born that way and so should no more be discriminated against on that basis than on the basis of sex or race.[2]

There is some evidence that people who think homosexuality is innate are more likely to support civil rights protections for gay people.[3] But from a moral and legal perspective, empirical facts about the causes of same-sex behaviour cannot, on their own, be the basis for civil rights.[4] It’s a genetic fallacy, a category error, to argue otherwise. Moreover, studies into the biological aspects of sexual orientation have historically resulted in truly gruesome efforts to ‘cure’ perfectly healthy people of their non-heterosexuality. More generally, LGBT+ people’s civil rights cannot be contingent on the latest scientific data or theories about human sexuality.

With the work of Zietsch et al.,[5] we return to the ‘gay gene’ narrative, but with a novel twist. Their work is part of a long history of research aimed at explaining homosexuality from an evolutionary perspective. Since exclusively same-sex behavior is not itself reproductive, how could such a disposition be maintained through generations (and occur in other animals)? Unlike most previous research, Zietsch et al. did not seek to explain a single sexual orientation (e.g. male homosexuality), rather they focused on non-heterosexual behavior, that is, any *same-sex sexual behavior* (SSB) in either males and females — something that cuts across many of LGBT+ social identity categories.

Their genome-wide association study confirmed what has been known for a long time: there is not a single gene that determines SSB, rather, they report “a very large number of genes” associated with it, amounting to a “very small effect spread across the genome.” Zietsch et al. report that those same gene-clusters also occur in some heterosexuals, where they seem to confer a “mating advantage” as measured by number of partners over a lifetime.[5] In simple terms: when genes associated with SSB occur in ‘straight’ people (whether male or female), the latter tend to have more partners. If this mating boost is enough to outweigh the ‘loss’ of reproduction among the same-sex group, this could explain the evolutionary puzzle.

The relevant genes are reportedly “pleiotropic,” which refers to genes or gene variants that have effects on multiple traits. The authors provide evidence that these same genes may be associated with psychological traits such as risk taking and openness to experience, and perhaps increased physical attractiveness, especially in men. They postulate that there could be other associations to “sex drive, orientation towards short vs. long-term relationships, and charisma.” There might not be genes specifically associated with sexual orientation, then, but rather genes for openness to experience (etc.).

Some will argue that – however scientifically interesting – this sort of research shouldn't be done, given how prone it is to societal abuse.[6] One can imagine technologically advanced repressive regimes where homosexuality is outlawed requiring genetic testing of embryos and foetuses, destroying those disposed to SSB or testing children early in life for their propensities. Others will respond that the world (or at least some parts of it) has become more accepting of homosexuality, so perhaps these worries are overblown.

Polygenic tests for various traits or dispositions (polygenic scores) such as SSB will likely become more widely available. Genomic Prediction offers polygenic scores for embryo selection in the US within the low-normal IQ range.[7] The fears expressed by particular groups of people - especially historically vulnerable groups who stand to be harmed - should be taken seriously. It is conceivable, for example, that genomic prediction of IQ and educational attainment might be heralded as useful for identifying those most likely to need more support (the original purpose of IQ tests themselves as developed by Binet); but the same genetic information is being used to select against 'low IQ' embryos (and more widely in Down Syndrome screening).

Of course, discrimination based on genes exists with single-gene prediction: in societies that discriminate against women and girls, knowing the likely sex of the foetus (based on the SRY gene) can lead to selective abortion of females. Dismantling structures of oppression is necessary, whatever genetic information becomes available.

The potential for polygenic scores to provide more detailed predictions about non-disease traits raises the spectre of eugenics and memories of Nazi atrocities. These involved involuntary sterilisation and murder of the supposedly genetically unfit, including homosexuals. A major lesson from Nazi eugenics was that if genetic information is used, this must be separated from any state apparatus, and should be voluntary for parents (non-directive counselling in clinical genetics). It must also be informed by principles of social justice and aimed at the *wellbeing of the future individual*. This raises novel questions for policy paradigms covering embryo testing based on disease categories.[7]

Polygenic associations will be an interconnected web of traits. Used in in-vitro fertilization, selection would involve choosing between embryos any of which could have been chosen by nature. Because the act of choosing cannot be said to harm the future child unless its life would be worse than non-existence, some ethical concerns on behalf of the future child are rendered moot.[8] In other words, the child cannot object that they would have been better off if another embryo had been chosen because a different child would have existed.[9] Nevertheless, individual selection choices may have collective effects, highlighting the need for socially conscious ethics.[10]

Current polygenic scores to predict disease and traits are all based upon associations. They do not detect causal genes. But in principle, such causal genes could be identified and modified through gene editing, intentionally reducing or increasing the probability of SSB. Gene editing, as opposed to selection, can directly harm the resulting person, both by creating off target mutations and bringing about pleiotropic modifications that make the person worse off than they would otherwise have been.

Pleiotropy shows that genetic packets are associated with multiple behaviours, some of which are valued by the individual or society, and some which aren't. The idea of good and bad genes in behavioural genetics is likely to be a fiction. To properly assess the ethics of genomic research and editing, we need not only knowledge of off-target mutations and pleiotropy, but answers to more philosophical questions: what makes a life good or meaningful? What matters is not only health, but our well-being, autonomy and identity.

Genes shape, limit, and provide opportunities for who we are and who we can be, both as individuals and as members of communities. To prepare for further research into pleiotropic behavioural traits including SSB, we must reshape society and ourselves to tackle historical injustices and structural inequalities. As part of this process, we will need to create a robust ethical framework for the responsible use of such knowledge.

## References

1. Katz, J. N. *The Invention of Heterosexuality* (University of Chicago Press, 2007).
2. Earp, B. D., & Vierra, A. Sexual orientation minority rights and high-tech conversion therapy. In D. Boonin (ed.), *The Palgrave Handbook of Philosophy and Public Policy* (pp. 535-550). Palgrave Macmillan, Cham (2018).
3. Lewis, G. B. Does believing homosexuality is innate increase support for gay rights?. *Policy Studies Journal* **37**, 669-693 (2009).
4. Stein, E. The relevance of scientific research about sexual orientation to lesbian and gay rights. *Journal of Homosexuality* **27**, 269-308 (2008).
5. Zietsch, B. P., Sidari, M., Abdellaoui, A. Maier, R., Långström, J. N., Guo, S., Beecham, G. W., Martin, E. R., Sanders, A. R. & Verweij, K. J. H. Genomic evidence consistent with antagonistic pleiotropy may help explain the evolutionary maintenance of same-sex sexual behaviour in humans. *Nature Human Behaviour* (in press)
6. Schuklenk, U., Stein, E., Byne, W. & Kerin, J. The ethics of genetic research on sexual orientation. *Hastings Center Report* **27**, 6-13 (1997).
7. Munday, S., Savulescu, J. Forthcoming. Three models for the regulation of polygenic scores in reproduction. *Journal of Medical Ethics* doi: 10.1136/medethics-2020-106588 (online first).
8. Parfit D. *Reasons and Persons* (Clarendon Press, 1984).
9. Savulescu, J., Hemsley, M., Newson, A. & Foddy, B. (2006). Behavioural genetics: Why eugenic selection is preferable to enhancement. *Journal of Applied Philosophy* **23**, 157-171 (2006).
10. Bavelier, D., Savulescu, J., Fried, L. P., Friedmann, T., Lathan, C.E., Schürle, S. & Beard, J.R. Rethinking human enhancement as collective welfarism. *Nature Human Behaviour* **3**, 204–206 (2019).

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