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COMMENTARY

Carving Non-Proximal Explanations for Same-Sex Sexual Orientation

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Introduction

VanderLaan et al. (2022) have produced a very interesting and thorough Target Article focusing on the biological development of same-sex sexual orientation in humans, providing a long-needed overview of what is known and what is still unclear concerning the "biological" (i.e., prenatal, see below) bases for this human behavioral trait. The overall picture is impressive, first for the phenotypic differences across sexual orientations, such as height, handedness, mental rotation tasks, etc., but also for the various mechanisms identified, from genes to hormones and the immune system. We found the Target Article extremely interesting and largely agree with their message. Yet, reading this paper from our evolutionary biologist background prompted several reactions that we wish to share as they might be of general interest to the study of this topic and of other aspects of human behavior.

The various determinants of the biodevelopment of same-sex sexual orientation are presented by VanderLaan et al. (2022) as an unordered list of proximal causes. Unless all the determinants presented are completely independent, some must be proximal relative to others. For example, the maternal immune hypothesis predicts the fraternal birth order effect (FBOE); thus, the former is a proximal explanation of the latter. Also, genes, necessarily at the beginning of all physiological events, including immunological and hormonal, are a proximal explanation for any determinant of the biodevelopment of same-sex sexual orientation. This is even true for effects described as non-genetic, such as the FBOE. The number of older brothers, or even the birth rank, is a non-heritable trait (you cannot select for a specific birth rank), and the FBOE is indeed a plastic, maternal effect. However, even if FBOE is purely a plastic trait, the ability to develop a FBOE could be genetic, in the same way as plasticity can have a genetic determination, be selected for and provide adaptive gains (e.g., Scheiner, 1993). In any case, proximal factors are generally less and less predictive of the final phenotype (here same-sex sexual orientation) when they are further down in the chain of causes. This is because they become increasingly loosely connected with the final phenotype, unless the causal chain is short and, at the end, only one or very few genes are concerned. The one-gene-one-phenotype configuration is very uncommon. In terms of same-sex sexual orientation, many genes have been found to be involved, each one with a small influence (Ganna et al., 2019), so that detailed knowledge of any one gene has a limited predictive power.

We also wish to stress that we concur with VanderLaan et al. (2022) when they advocate for more replications of empirical results, especially on brain anatomy differences between various classes of sexual orientation. Many of these studies have been based on a data mining strategy, where many anatomical traits have been measured on a (often small) sample of subjects with varying sexual orientation and differences are sought for all the measured traits. These procedures result in increased risk of false positives and any such results need to be reproduced in independent samples before they can be trusted. This problem is not limited to the study of human behavior of course (see Nakagawa & Parker, 2015 for ecology and evolution) and it is a well-recognized issue in the psychological literature (see Carpenter, 2012). As is evident from VanderLaan et al.'s (2022) Target Article, many of the results on brain anatomy have not been replicated. This is even more worrying if negative results have been less likely to be published. From the many effects discussed in this review, it seems that differences in brain anatomy are generally less likely to have been reproduced than other well-established effects (such as the FBOE), probably because they are much more challenging to study. This throws into

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question which of the many reported differences in brain structure between heterosexuals and homosexuals are real.

We need to mention, as another minor point, that VanderLaan et al. (2022) did not discuss recent research regarding the proximate mechanisms of same-sex attractions, namely, the role of epigenetic marks (Gavrilets et al., 2018; Ngun & Vilain, 2014; Rice et al., 2016). Most importantly, this mechanism also generates testable predictions (Rice et al., 2013), although this model does not explain why such epigenetic influence would only affect our species and not other primate species (see below).

In what follows, we aim to present some alternative conceptual frameworks to organize and discuss the mechanisms VanderLaan et al. (2022) present.

Everything Is Biological: Translating the Nature versus Nurture Distinction Into Clearly Defined Mechanisms of Variation

VanderLaan et al. (2022) explicitly narrow their review to the "biological development (hereto biodevelopment)" of same-sex sexual orientation, yet their review lacks a definition of "biological" or "biodevelopment." For biologists, studying the bases of a behavior *is* biology and all processes that influence the variations of this behavior are biological, including social interactions, learning, personal experiences, etc. VanderLaan et al.'s distinction between biological and "socialization" is probably akin to the "nature versus nurture" or "innate versus acquired" distinction that is at the root of many fields of thought working with human behavior. In the context of their Target Article, VanderLaan et al. seem to draw the line between "nature" and "nurture" at birth, and group all prenatal factors, genetic or non-genetic, as "nature" aka "biological." This surely makes sense, as it is reasonable to assume that prenatal processes (such as hormonal environment in utero) and postnatal experiences (including social interactions within the family group or early experiences as children or teenagers) rely on different mechanisms and pathways. Yet, all these processes are biological by nature. A large part of the genetic effects on mate choice in humans, including sexual orientation, might correlate with other traits of personality and be expressed during post-natal development.

For evolutionary biologists, what matters is less the timing of the processes (post-natal versus prenatal here) than how they are determined, which is expressed as the fundamental distinction between genetic and environmental factors (for a recent review, see Stamp & Hadfield, 2020), the latter including epigenetic effects. Genetic factors are based on differences between the gene themselves (the DNA sequence) and are transmitted from generations to generation unless the genetic variants are lost from the populations; they can be the target of natural selection and drive evolution. Epigenetic factors are based on differences in proteins that interact with the DNA chain and regulate its expression and are thus a type of environmental factor (Allis & Jenuwein, 2016); they typically cannot be transmitted over more than a few generations and are not the target of selection or the basis of evolution, although they can influence evolutionary processes. Non-epigenetic environmental factors affect the phenotype during development but these effects are not passed to the next generations. In biological terms, environmental effects (including epigenetic effects) are grouped under the term "phenotypic plasticity," which corresponds to all factors that produce different phenotypes for the exact same genotype. It is also essential to keep in mind that genetic and environmental factors interact to shape phenotype: genetic and environmental effects influence each other via genetic-environment interactions.

The "nature versus nurture" distinction of psychology is thus expressed as "genetic differences versus plasticity" in evolution and ecology, and we argue here that incorporating this distinction when trying to explain variations in human phenotypes, including behavior, provides a clearer framework for distinguishing among alternative hypotheses. Indeed, prenatal effects can be influenced by all three main types of factors (genetic, epigenetic, and environmental) while "non-biological" or "social" effects refer to only a fraction of the possible environmental effects. Some of the effects discussed by VanderLaan et al. (2022) are, by definition, non-genetic (for example, the FBO, which is an effect of the prenatal environment on the phenotype) while others (hormonal mechanisms, for example) can be genetic or plastic (i.e., environmental). The lack of clear distinction between these bases for variation in sexual orientation result in confusion in several places in VanderLaan et al.'s (2022) Target Article. Genetic differences can influence or determine hormonal or even immunological mechanisms. Hormonal differences are a mix of genetic (variation in receptors) and environmental (level of hormones exposure during pregnancy) factors. Only the immunological hypothesis is entirely environmental by nature, even though its outcome can be influenced by the fetus' genotype and the mother's genotype.

"How" Is Important, But So Is "Why"

Another fundamental concept in biology that is lacking in VanderLaan et al.'s (2022) Target Article is the separation between proximate and ultimate mechanisms. Although this terminology has sometimes been criticized (e.g., Haig, 2013), the distinction between how (proximate) and why (ultimate) a behavior varies remains both useful and necessary even in human behavioral sciences (Scott-Phillips et al., 2011). The list of proximal causes to sexual orientation

provided by VanderLaan et al. (2022) is impressive, and the general impression is that we are progressing toward a better understanding of the ontogeny of same-sex sexual orientation. However, proximal causes cannot address some basic question such as *why* same-sex sexual orientation exists in humans. Indeed, the analysis of genes, hormones or antibodies responsible for the biodevelopment of sexual orientation provides knowledge on the proximal causes of same-sex sexual orientation, but is of little help in understanding why same-sex sexual orientation exist at all in our species. For example, we might ask why infants are crying. A proximate answer could point to the separation from caregiver, cold, lack of food, or endogenous opioids. An ultimate explanation would suggest than crying elicits care and protection from mothers and other caregivers, which will increase the likelihood of survival and hence lead to inclusive fitness benefits, thereby explaining why this behavior has been selected to be expressed in some contexts (Bowlby, 1983; Scott-Phillips et al., 2011). Both answers are useful, but at different levels. Proximate studies are helpful to decipher physiological pathways, cellular interactions, and more generally to understand events linking genes and phenotypes at the individual level. These studies provide answer to "how" questions and, in humans, have obvious practical applications in medicine, health, and well-being. Evolutionary studies are helpful to answer "why" questions, and thus for understanding what sort of selection pressures operate, allowing us to sometimes make predictions about the evolution of the trait under scrutiny. Interestingly, proximate and evolutionary studies are not necessarily disconnected. On the contrary, there are clear interactions between them, for example most genes have pleiotropic effects, i.e., an influence on distinct traits. As a consequence, selection on one trait might trigger a concomitant change in another trait, with an uncertain overall outcome. Clearly, "how" and "why" questions are interconnected. VanderLaan et al. (2022) have presented the state of the art for the "how" aspects of same-sex sexual orientation in humans. How does their Target Article help to understand *why* there is variation in sexual orientation among humans?

This question is justified given that sexual orientation is heritable as mentioned by VanderLaan et al. (2022), and it is reproductively costly. This cost can be relatively high, possibly as high as around 70–100% for homosexual men in some contexts (Coome et al., 2020; Iemmola & Camperio-Ciani, 2009; Nila et al., 2018; Rieger et al., 2012; Vasey et al., 2007), and doubtlessly lower in societies with strong constraints on individual ways of life but probably never zero. As a costly and heritable trait cannot emerge in a population and be maintained at frequencies far above the mutation rate (e.g., Hedrick, 2010), a specific explanation should be sought.

A first step to understand this paradox is to determine which species or animal clades exhibit same-sex sexual orientation, as it helps to identify features that are associated with same-sex sexual orientation across the tree-of-life. There are numerous reports of homosexual behavior in many animal species in the wild, but there are no reports of exclusive same-sex sexual orientation (Bagemihl, 2000). It is, however, well-established that some domestic rams exhibit exclusive same-sex sexual orientation. However, information based on domestic rams cannot be used to make useful evolutionary inferences about the occurrence of same-sex sexual orientation in natural populations. Incidentally, studies which addressed the ontogeny of homosexuality for rams found that specific brain areas were involved, as well as hormonal correlates, and research had begun to decipher the pathways involved (Roselli & Stormshak, 2009; Roselli et al., 2011). There are other vertebrate species for which same-sex sexual preference can be induced experimentally, such as rats, mice, ferrets, zebra finches, and Japanese quail and, for those species, we are beginning to understand the neuroendocrinology of partner preference fairly well (Balthazart, 2020). For example, the early sex steroid environment has an organizational effect on later sexual partner preference in birds and mammals (Adkins-Regan, 2020; Henley et al., 2011). In any case, the phylogenetic uniqueness of exclusive same-sex sexual partner preference seems to be restricted to only one species, humans. This suggests that same-sex sexual orientation is probably not a mere constraint of mammalian developmental physiology, but rather, is the product of evolutionary processes generated by some idiosyncrasies of the human species.

The second step is to understand if same-sex attraction is an adaptation by itself, or if it represents the cost of a pleiotropic and otherwise advantageous trait. Homosexual preferences can emerge and be maintained under certain conditions if the associated decrease in reproduction is compensated by sufficient increases in reproduction among close relatives (reviewed in Gavrilets & Rice, 2006). This indirect reproduction may be increased behaviorally by kin selection, and it has been proposed that individuals displaying same-sex sexual preference could behave as "helpers," favoring reproduction by kin and thereby compensating for their direct reproductive cost (Trivers, 1974; Wilson, 1975). For homosexual men, this kin selection hypothesis is not supported in Western countries and Japan, although behavioral evidence (avuncular tendencies) has been obtained in Samoa, Mexico and Java (Gómez Jiménez & Vasey, 2022; Nila et al., 2018; Vasey & VanderLaan, 2009, 2010). However, a detailed study concluded that higher indirect reproduction did not fully compensate for homosexual men's direct reproductive cost, and the higher avuncular tendencies displayed by homosexual men marginally affected their number of nephews and nieces (Nila et al., 2018). Although this study should be repeated in an array of human societies, it nevertheless suggests that kin selection alone is insufficient to explain the emergence and maintenance of same-sex sexual orientation.

Another candidate for an adaptive explanation is reduced sibling competition, which could occur via a mother's plastic manipulation of the phenotype of male offspring, as displayed for example by the FBOE. The FBOE, a proximate explanation for same-sex sexual orientation in males, is indeed detected in several occidental and non-occidental societies (e.g., Blanchard, 2018; Raymond et al., 2022). It is not possible to evaluate whether FBOE is restricted or not to humans, as exclusive same-sex sexual preference is not found among non-domesticated animals, and research on homosexual rams have been stopped for non-scientific reasons (Ersly, 2013). However, based on current knowledge, effect of birth order on reproduction is only found in humans, and in no other primate species, including those that are close human relatives. This suggests that the FBOE is not a mere constraint of the gestation in primates, and thus the effect of male birth order on sexual orientation requires an evolutionary explanation. Nila et al. (2019) have proposed that the FBOE could decrease male sibling competition by later-born males. Such a mechanism could be selected for in patrilocal societies, but probably not in matrilocal ones, where males usually migrate, thus reducing local competition. To evaluate this prediction, it should be then interesting to compare FBOE between patrilocal and matrilocal societies.

Another line of hypotheses has proposed that the reproductive cost of homosexual individuals is compensated by increased fitness in close relatives. The idea is that genes responsible for same-sex sexual orientation have a pleiotropic effect, lowering reproduction in some individuals but increasing it in their relatives. The first candidate was a sex-antagonistic effect, such that a gene promoting same-sex sexual orientation in males, thus reducing male fertility, increases fertility in females. Samples of homosexual and heterosexual males were compared for their family composition, and it was found that female kin of homosexuals displayed a higher rate of fertility (Camperio-Ciani et al., 2004, 2009; Iemmola & Camperio-Ciani, 2009). However, this effect was subsequently found to be artefactual: in the presence of FBOE, homosexuals have a higher birth rank (due to their higher number of older brothers), thus mimicking a higher maternal fertility if birth rank is not controlled for (Blanchard et al., 2020; Raymond et al., 2022). A similar mechanism has also been proposed to explain female same-sex attraction, as female kin of homosexual women were found more fertile than corresponding families of heterosexuals (Camperio Ciani et al., 2018). However, due to a possible existence of an older sister effect (Ablaza et al., 2022; Blanchard & Lippa, 2007; for a discussion, see Raymond et al., 2022), a similar sampling bias (as described above for male homosexuals) could operate, as birth rank was not controlled for in this study. Finally, another type of pleiotropic gene has been proposed: instead of increasing fertility in female relatives, the advantage of the pleiotropic gene is to increase mating success in heterosexual male relatives (Zietsch et al., 2008, 2021). Further studies are required to confirm the presence of such an intra-sex antagonist effect.

Pleiotropic effects are very widespread in biology, for example they are abundant in common complex diseases and traits in humans (Sivakumaran et al., 2011). This high frequency is probably due to the fact that most genes are not restricted to only one phenotypic effect, with the consequence that a genetic change affects several phenotypes. If all phenotypic modifications have a negative effect on fitness, this change (deleterious mutation) is not retained. If only one modification has a positive effect, then the overall effect on fitness will determine the outcome of selection. Deleterious phenotypic traits could thus be maintained in populations if the genes responsible for these traits also induce other advantageous phenotypic traits, so that the resulting overall effect is a higher fitness. This could explain the relatively high frequency of same-sex attraction: this reproductively costly phenotype could have been selected via pleiotropic positive effects in relatives. One difficulty with this pleiotropic theory is the possibility that pleiotropy itself evolves, especially if one phenotype is costly: in this context, modifiers that would weaken or suppress the genetic correlation could be selected for. In such a case, same-sex sexual orientation would no longer be associated with a fitness advantage in relatives, and a decrease in same-sex sexual orientation would be expected. This outcome is not ineluctable, as there are examples of million-year-old pleiotropic genes displaying strong deleterious effects, but still present, due to their overall positive fitness (e.g., the t-haplotype in mice; Hammer, 1991). Whether or not this will be the case for same-sex sexual orientation is not known, but a detailed knowledge of the proximal causes, including genetic effects, could shade light on this question.

Conclusion

Evolutionary explanations for the selection and persistence of reproductively costly traits such as same-sex attraction in humans are not numerous. Much work in the previous decades has focused on kin selection, with the conclusion that this explanation alone seems insufficient and cannot explain the origin or the maintenance of same-sex sexual preference in our species. Pleiotropic effects are now scrutinized, although there is no consensus yet on the nature of the advantageous trait that would be selectively favored and linked to same-sex attraction. Immunological or epigenetic mechanisms also suggest that at least a fraction of the variation on sexual orientation is due to "side-effects" of physiological interactions between mothers and fetuses during pregnancy, which can be seen as a special case of pleiotropy. Other possible evolutionary explanations, that have not been empirically assessed as far as we know, include reduced sib-competition,

which could explain the maternal effect mediated by fraternal birth order that has been consistently found in multiple populations, and that also needs to be understood at an evolutionary level.

Clearly, the impressive amount of knowledge on genetic and environmental factors linked to same-sex sexual orientation that VanderLaan et al. (2022) describe will be invaluable in evaluate evolutionary explanations for the prevalence of same-sex sexual orientation in humans. We were especially intrigued by the possibility that distinct classes of homosexual individuals are present, as suggested by VanderLaan et al. (2022) in their Target Article, each associated with different proximate mechanisms, hence also suggesting the existence of different ultimate explanations. This situation could explain why it has been so difficult to reach a scientific consensus in explaining homosexual preference in humans.

While we are still far from understanding the proximate and ultimate bases of same-sex sexual attraction (the "how" and the "why"), we argue that much progress can stem from a better partitioning and prioritization of the sources of variations in human sexual preferences. Further studies should continue to decipher the relative influence of genetic and plastic effects (environmental, including epigenetic) or weight the relative importance of prenatal (e.g., physiological) versus post-natal (e.g., social) factors in shaping individual sexual preferences. These various effects doubtless interact and influence each other's, which should call for interdisciplinary approaches bringing different schools of research together. By providing an extensive review of our current state of knowledge on proximal mechanisms, VanderLaan et al. (2022) have made an important contribution to this goal.

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Declarations

Conflict of interest The authors have no conflicts of interest to declare.

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References

Ablaza, C., Kabátek, J., & Perales, F. (2022). Are sibship characteristics predictive of same sex marriage? An examination of fraternal birth order and female fecundity effects in population-level administrative data from the Netherlands. *Journal of Sex Research, 59*(6), 671–683. https://doi.org/10.1080/00224499.2021.1974330

Adkins-Regan, E. (2020). Sexual and pairing partner preference in birds and other animals. *Hormones and Behavior, 118*, 104646. https://doi.org/10.1016/j.yhbeh.2019.104646

Allis, C. D., & Jenuwein, T. (2016). The molecular hallmarks of epigenetic control. *Nature Reviews Genetics*, *17*, 487–500. https://doi.org/10.1038/nrg.2016.59

Bagemihl, B. (2000). *Biological exuberance: Animal homosexuality and natural diversity* (Reprint édition). Stonewall Inn Editions.

Balthazart, J. (2020). Sexual partner preference in animals and humans. *Neuroscience & Biobehavioral Reviews, 115*, 34–47. https://doi.org/10.1016/j.neubiorev.2020.03.024

Blanchard, R. (2018). Fraternal birth order, family size, and male homosexuality: Meta-analysis of studies spanning 25 years. *Archives of Sexual Behavior, 47*(1), 1–15. https://doi.org/10.1007/s10508-017-1007-4

Blanchard, R., & Lippa, R. A. (2007). Birth order, sibling sex ratio, handedness, and sexual orientation of male and female participants in a BBC internet research project. *Archives of Sexual Behavior, 36*(2), 163–176. https://doi.org/10.1007/s10508-006-9159-7

Blanchard, R., Krupp, J., VanderLaan, D. P., Vasey, P. L., & Zucker, K. J. (2020). A method yielding comparable estimates of the fraternal birth order and female fecundity effects in male homosexuality. *Proceedings of the Royal Society B: Biological Sciences, 287*(1923), 20192907. https://doi.org/10.1098/rspb.2019.2907

Bowlby, J. (1983). *Attachment*. Basic Books. https://www.basicbooks.com/titles/john-bowlby/attachment/9780465005437/

Camperio Ciani, A., Battaglia, U., Cesare, L., Camperio Ciani, G., & Capiluppi, C. (2018). Possible balancing selection in human female homosexuality. *Human Nature, 29*(1), 14–32. https://doi.org/10.1007/s12110-017-9309-8

Camperio-Ciani, A.-S., Corna, F., & Capiluppi, C. (2004). Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proceedings of the Royal Society London, B, 271*, 2217–2221.

Camperio-Ciani, A. C., Iemmola, F., & Blecher, S. R. (2009). Genetic factors increase fecundity in female maternal relatives of bisexual men as in homosexuals. *Journal of Sexual Medicine, 6*(2), 449–455. https://doi.org/10.1111/j.1743-6109.2008.00944.x

Carpenter, S. (2012). Psychology's bold initiative. *Science, 335*(6076), 1558–1561. https://doi.org/10.1126/science.335.6076.1558

Coome, L. A., Skorska, M. N., & VanderLaan, D. P. (2020). Direct reproduction and sexual orientation and gender diversity in Thailand. *Archives of Sexual Behavior, 49*(7), 2449–2460. https://doi.org/10.1007/s10508-020-01830-8

Ersly, W. (2013). The desideratum of discourse: Lessons learned from a gay sheep. In P. C. Hoy II., S. Donatelli, D. A. Forrest, G. Halliday, & E. Bobella (Eds.), *Mercer street: A collection of essays from the expository writing program* (pp. 47–55). New York University.

Ganna, A., Verweij, K. J. H., Nivard, M. G., Maier, R., Wedow, R., Busch, A. S., Abdellaoui, A., Guo, S., Sathirapongsasuti, J. F., Team16, 23andMe Research, Lichtenstein, P., Lundström, S., Långström, N., Auton, A., Harris, K. M., Beecham, G. W., Martin, E. R., Sanders, A. R., Perry, J. R. B., & Zietsch, B. P. (2019). Large-scale GWAS reveals insights into the genetic architecture of same-sex sexual behavior. *Science*, *365*. https://doi.org/10.1126/science.aat7693

Gavrilets, S., Friberg, U., & Rice, W. R. (2018). Understanding homosexuality: Moving on from patterns to mechanisms. *Archives of Sexual Behavior, 47*(1), 27–31. https://doi.org/10.1007/s10508-017-1092-4

Gavrilets, S., & Rice, W. R. (2006). Genetic models of homosexuality: Generating testable predictions. *Proceedings of the Royal Society London, B, 273*, 3031–3038.

Gómez Jiménez, F. R., & Vasey, P. L. (2022). Kin-directed altruism and the evolution of male androphilia among Istmo Zapotec Muxes. *Evolution and Human Behavior*, *43*, 224–233. https://doi.org/10.1016/j.evolhumbehav.2022.02.002

Haig, D. (2013). Proximate and ultimate causes: How come? And what for? *Biology & Philosophy, 28*(5), 781–786. https://doi.org/10.1007/s10539-013-9369-z

Hammer, M. F. (1991). Molecular and chromosomal studies on the origin of t haplotypes in mice. *The American Naturalist, 137*(3), 359–365. https://doi.org/10.1086/285169

Hedrick, P. W. (2010). *Genetics of populations* (4e édition). Jones and Bartlett Publishers, Inc.

Henley, C. L., Nunez, A. A., & Clemens, L. G. (2011). Hormones of choice: The neuroendocrinology of partner preference in animals. *Frontiers in Neuroendocrinology, 32*(2), 146–154. https://doi.org/10.1016/j.yfrne.2011.02.010

Iemmola, F., & Camperio-Ciani, A. C. (2009). New evidence of genetic factors Influencing sexual orientation in men: Female fecundity increase in the maternal line. *Archives of Sexual Behavior, 38*, 393–399.

Nakagawa, S., & Parker, T. H. (2015). Replicating research in ecology and evolution: Feasibility, incentives, and the cost-benefit conundrum. *BMC Biology, 13*(1), 88. https://doi.org/10.1186/s12915-015-0196-3

Ngun, T. C., & Vilain, E. (2014). The biological basis of human sexual orientation. *Advances in Genetics, 86*, 167–184. https://doi.org/10.1016/B978-0-12-800222-3.00008-5

Nila, S., Barthes, J., Crochet, P.-A., Suryobroto, B., & Raymond, M. (2018). Kin selection and male homosexual preference in Indonesia. *Archives of Sexual Behavior, 47*(8), 2455–2465. https://doi.org/10.1007/s10508-018-1202-y

Nila, S., Crochet, P.-A., Barthes, J., Rianti, P., Juliandi, B., Suryobroto, B., & Raymond, M. (2019). Male homosexual preference: Femininity and the older brother effect in Indonesia. *Evolutionary Psychology*. https://doi.org/10.1177/1474704919880701

Raymond, M., Turek, D., Durand, V., Nila, S., Suryobroto, B., Vadez, J., Barthes, J., Apostoulou, M., & Crochet, P.-A. (2022). Increased birth rank of homosexual males: Disentangling the older brother effect and sexual antagonism hypothesis. *BioRxiv*. https://doi.org/10.1101/2022.02.22.481477

Rice, W. R., Friberg, U., & Gavrilets, S. (2013). Homosexuality via canalized sexual development: A testing protocol for a new epigenetic model. *Bioessays*, *35*, 364–370.

Rice, W. R., Friberg, U., & Gavrilets, S. (2016). Sexually antagonistic epigenetic marks that canalize sexually dimorphic development. *Molecular Ecology, 25*(8), 1812–1822. https://doi.org/10.1111/mec.13490

Rieger, G., Blanchard, R., Schwartz, G., Bailey, J. M., & Sanders, A. R. (2012). Further data concerning Blanchard's (2011) "Fertility in the Mothers of Firstborn Homosexual and Heterosexual Men" [Letter to the Editor]. *Archives of Sexual Behavior*, *41*, 529–531.

Roselli, C. E., Reddy, R. C., & Kaufman, K. R. (2011). The development of male-oriented behavior in rams. *Frontiers in Neuroendocrinology, 32*(2), 164–169. https://doi.org/10.1016/j.yfrne.2010.12.007

Roselli, C. E., & Stormshak, F. (2009). Prenatal programming of sexual partner preference: The ram model. *Journal of Neuroendocrinology, 21*(4), 359–364. https://doi.org/10.1111/j.1365-2826.2009.01828.x

Scheiner, S. M. (1993). Genetics and evolution of phenotypic plasticity. *Annual Review of Ecology and Systematics, 24*, 35–68.

Scott-Phillips, T. C., Dickins, T. E., & West, S. A. (2011). Evolutionary theory and the ultimate–proximate distinction in the human behavioral sciences. *Perspectives on Psychological Science, 6*(1), 38–47. https://doi.org/10.1177/1745691610393528

Sivakumaran, S., Agakov, F., Theodoratou, E., Prendergast, J. G., Zgaga, L., Manolio, T., Rudan, I., McKeigue, P., Wilson, J. F., & Campbell, H. (2011). Abundant pleiotropy in human complex diseases and traits. *American Journal of Human Genetics, 89*(5), 607–618. https://doi.org/10.1016/j.ajhg.2011.10.004

Stamp, M. A., & Hadfield, J. D. (2020). The relative importance of plasticity versus genetic differentiation in explaining between population differences: A meta-analysis. *Ecology Letters, 23*(10), 1432–1441. https://doi.org/10.1111/ele.13565

Trivers, R. L. (1974). Parent-offspring conflict. *American Zoologist, 14*, 249–264.

VanderLaan, D. P., Skorska, M. N., Peragine, D. E., & Coome, L. A. (2022). Carving the biodevelopment of same-sex sexual orientation at its joints. *Archives of Sexual Behavior*. https://doi.org/10.1007/s10508-022-02360-1

Vasey, P. L., Pocock, D. S., & VanderLaan, D. P. (2007). Kin selection and male androphilia in Samoan fa'afafine. *Evolution and Human Behavior, 28*, 159–167.

Vasey, P. L., & VanderLaan, D. P. (2009). Kin selection and the evolution of male androphilia. *Achives of Sexual Behavior, 38*, 170–171.

Vasey, P. L., & VanderLaan, D. P. (2010). Monetary exchanges with nieces and nephews: A comparison of Samoan men, women, and fa'afafine. *Evolution and Human Behavior, 31*(5), 373–380. https://doi.org/10.1016/j.evolhumbehav.2010.04.001

Wilson, E. O. (1975). *Sociobiology: The new synthesis*. Harvard University Press. https://www.hup.harvard.edu/catalog.php?isbn=9780674002357

Zietsch, B. P., Morley, K. I., Shekar, S. N., Verweij, K. J. H., Keller, M. C., Macgregor, S., Wright, M. J., Bailey, J. M., & Martin, N. G. (2008). Genetic factors predisposing to homosexuality may increase mating success in heterosexuals. *Evolution and Human Behavior, 29*(6), 424–433. https://doi.org/10.1016/j.evolhumbehav.2008.07.002

Zietsch, B. P., Sidari, M. J., Abdellaoui, A., Maier, R., Långström, N., Guo, S., Beecham, G. W., Martin, E. R., Sanders, A. R., & Verweij, K. J. H. (2021). Genomic evidence consistent with antagonistic pleiotropy may help explain the evolutionary maintenance of same-sex sexual behaviour in humans. *Nature Human Behaviour, 5*, 1251–1258. https://doi.org/10.1038/s41562-021-01168-8