

Heterosexual Male Carriers Could Explain Persistence of Homosexuality in Men: Individual-Based Simulations of an X-Linked Inheritance Model

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Abstract Homosexuality has been documented throughout history and is found in almost all human cultures. Twin studies suggest that homosexuality is to some extent heritable. However, from an evolutionary perspective, this poses a problem: Male homosexuals tend to have on average five times fewer children than heterosexual males, so how can a phenomenon associated with low reproductive success be maintained at relatively stable frequencies? Recent findings of increased maternal fecundity of male homosexuals suggest that the genes responsible for homosexuality in males increase fecundity in the females who carry them. Can an increase in maternal fecundity compensate for the fecundity reduction in homosexual men and produce a stable polymorphism? In the current study, this problem was addressed with an individual-based modeling (IBM) approach. IBM suggests that male homosexuality can be maintained in a population at low and stable frequencies if roughly more than half of the females and half of the males are carriers of genes that predispose the male to homosexuality.

Keywords Homosexuality · Simulation · Population Genetics · X-linked · Sexual orientation

Introduction

Homosexuality has been documented throughout history and is found in almost all human cultures (Kirkpatrick, 2000). The oldest depictions of homosexuality come from Mesolithic rock art (Nash, 2001), although generally it is accepted that the prevalence of homosexuality in human societies is low (Diamond,

1993; Grulich, Visser, Smith, Rissel, & Richters, 2003; Sell, Wells, & Wypij, 1995). Homosexual males on average have less reproductive success than heterosexuals (Iemmola & Ciani, 2009; Rieger, Blanchard, Schwartz, Bailey, & Sanders, 2012; Van de Ven, Rodden, Crawford, & Kippax, 1997). Consequently, from an evolutionary perspective, homosexuality is a paradox: How can a phenomenon associated with low reproductive success be maintained at relatively stable frequencies (Hutchinson, 1959; Wilson, 1975)?

Research suggests that sexual orientation is influenced by genetic factors. Specifically, monozygotic (MZ) twins, who share all of their genes, are more likely to be concordant in their sexual orientation than are dizygotic (DZ) twins or simple siblings, who only share, on average, half of their genes (Kendler, Thornton, Gilman, & Kessler, 2000; Kirk, Bailey, Dunne, & Martin, 2000). In an effort to identify the gene or genes that influence sexual orientation, Hamer, Hu, Magnuson, Hu, and Pattatucci (1993) conducted a genetic study in a group of 40 families in which there were two gay brothers. Their data indicated a linkage between the Xq28 markers and sexual orientation. More recently, a genome-wide study replicated results showing linkage of homosexuality not only to Xq28, but also to elements on chromosome 8 (Sanders et al., 2014).

It is, however, also demonstrable that homosexual men, compared to heterosexual men, tend to come from larger families (Ciani, Corna, & Capiluppi, 2004; Ciani & Pellizzari, 2012; Iemmola & Ciani, 2009; King et al., 2005). Greater fecundity in relatives of homosexual men could potentially explain the counterintuitive persistence of homosexuality through sexually antagonistic selection—genes predisposing homosexuality in men increase fecundity in females who carry them (Ciani & Pellizzari, 2012; Iemmola & Ciani, 2009; Zietsch et al., 2008).

However, the positive fecundity increment accruing to mothers of homosexual men is not large: 1.16 times higher per Ciani et al. (2004), 1.31 times higher per Iemmola and Ciani (2009),

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and 1.38 times higher per Vasey and VanderLaan (2007), and it is not clear if this associated increased fecundity is sufficient to compensate for the reduced fecundity of homosexual men. Several studies attempted to address this question from a theoretical population genetics perspective. MacIntyre and Estep (1993) studied a model of overdominance, in which they suggest that homosexuality can persist in a population if heterozygous individuals have a reproductive advantage. Getz (1993) assumed that reduced mating success of homosexual men was compensated by increased rearing success of females, or increased joint fecundity and cooperation of couples. Later, Gavrillets and Rice (2006) developed a number of genetic models for overdominance, maternal effects, and sexual antagonism considering a single diallelic locus, either autosomal or X-linked. They showed by deterministic models that genes which predispose a carrier to homosexuality can persist in a polymorphic population across a wide range of conditions. Ciani, Cermelli, and Zanzotto (2008) expanded these models and tested them, along with two-locus genetic models; they concluded that only the two-locus genetic model, with at least one locus on the X chromosome, and incorporating sexually antagonistic gene expression, was able to explain the persistence of homosexuality. All other models yielded either extinction or fixation of alleles favoring homosexuality in the modeled populations.

Traditional mathematical models, which were used in the above listed studies, describe populations as an entire system, usually using differential equations. The basic principle of such genetic models is to find stable equilibrium between allele frequencies under which polymorphism can be maintained. Sex-linked locus equilibrium models are discussed in detail in Haldane and Jayakar (1964). An alternative, more recent approach uses individual-based modeling (IBM), wherein each individual is represented as an independent unit. IBMs allow for the possibility to include individual variation and to design more experiment-like scenarios (Borrill & Tesfatsion, 2011; Grimm & Railsback, 2013; Roughgarden, 2012). IBMs have not been used to address homosexuality, although they have been used to model sex chromosome dynamics in populations (Taylor & Jaenike, 2002, 2003). Published models (Ciani et al., 2008; Gavrillets & Rice, 2006; Getz, 1993) have thus provided significant contributions to theoretical genetic studies of homosexuality and laid the foundation for further research. However, they do not specifically treat separately homosexual and non-homosexual carriers of genes predisposing to homosexuality.

Not all MZ twin pairs who share the same genes are concordant for homosexuality. Population sample-based studies suggest that, on average, 20 % of twins are concordant for homosexuality: Bailey, Dunne, and Martin (2000): 23.1 %, Kendler et al. (2000): 31.6 %, Långström, Rahman, Carlström, and Lichtenstein (2010): 9.9 %. Older, self-selection studies suggest higher rates, about 50 % concordance: Kallmann (1952): 100 %, Heston and Shields (1968): 42.9 %, Bailey and Pillard (1991): 51.8 %, Whitam, Diamond, and Martin (1993): 64.7 %. In line with the underlying influence of

genetic factors, these reports also indicate the existence of carrier males. This means that, on average, 50–80 % of males who have genes predisposing them to homosexuality are heterosexual, and there are no data to suggest that they have reduced fecundity. Zietsch et al. (2008) showed that heterosexuals who have a homosexual twin have, on average, the same number of female sex partners as heterosexuals who have a heterosexual twin.

Here I report individual-based modeling results of single locus X-linked, sexually antagonistic model of homosexuality.

Method

Population Genetic Model

The model treats a single, X-linked locus with alleles A_1 and A_2 , with A_1 predisposing males to homosexuality and increasing female carrier's fecundity. This stochastic single population model is described in Taylor and Jaenike (2003). The script was written in general programming language PHP; data were stored in MySQL database.¹ Validation of the model was performed by comparing inputs, expected outputs, and simulation results.

Model Parameters

The first generation is generated with a population size $N = 250$. Generations are not overlapping. For simplicity, mating is monogamous (because there is no evidence that mothers of homosexual and heterosexual men have different numbers of mates). Gender is assigned randomly with ratio $N_m:N_f = 1:1$ (Table 1). If $N_f < N_m$ mating pairs number equals N_f ; If $N_m < N_f$ mating pairs number equals N_m . Other individuals are celibate and cannot produce children. Because the rate of allele fixation depends also on initial frequencies (Kimura, 1962), they were set equal. The first generation contains equal frequencies of A_1 and A_2 : 50 % of males with A_1 — and 50 % of males with A_2 —, 50 % of homozygous females with A_1A_1 and 50 % of females with A_2A_2 . All models are sexually antagonistic—females with A_1 allele accrue a reproductive advantage, and males with A_1 allele do not. Advantage is defined by increased average fecundity. If the father has A_1 allele and is homosexual, fecundity is reduced. If the father has A_1 allele and is heterosexual (carrier male), fecundity is the same as the population average (Table 2).

Female and male carrier fecundity parameters:

¹ PHP script and MySQL table instructions are available upon request by email.

$$\alpha = \frac{F_{A_1A_1}}{F_{A_2A_2}} = \frac{F_{A_1A_2}}{F_{A_2A_2}} > 1 \quad \text{and} \quad \beta = \frac{F_{A_1-}}{F_{A_2-}} \quad (1)$$

Empirical data from the literature are scarce and some variation for different populations is expected. To cover it, I included value of α and β below and above reported numbers: $\beta = 0.3$ (Rieger et al., 2012), $\alpha = 1.16$ (Ciani et al., 2004), $\alpha = 1.31$ (Iemmola & Ciani, 2009), and $\alpha = 1.38$ (Vasey & VanderLaan, 2007). In the current simulation, 25 combinations of α and β were tested for each model. α increasing from 1.1 to 1.6 with 0.1 interval; β increasing from 0.2 to 0.6 with 0.1 interval.

Simulations

For the first generation, the average number of children, r , is set to 2 (± 1 SD), assuming a Poisson distribution for r . Gender is randomly assigned to each new individual with equal probability being male or female. The male inherits one of the maternal X chromosomes. The female inherits an X chromosome from her father and one of her mother's X chromosomes, which is selected independently and randomly for each individual.

To ensure a stable population size after the second (and all following) generation, r is adjusted

$$r_{t+1} = r_t \frac{N_{\max}}{N_t} \quad (2)$$

where N_{\max} is maximum population size and t denotes generation number. Formula (2) is derived from Maynard-Smith and Slatkin's (1973) population logistic growth formula.

Table 1 Notations used in the stochastic model

Term	Description
α	Female carrier fecundity parameter ($\alpha > 1$)
β	Male carrier fecundity parameter ($\beta < 1$)
N	Total population size
N_e	Effective population size (Number of breeding pairs \times 2)
N_f	Number of females in the population
N_m	Number of males in the population
N_t	Population size of t generation
N_{\max}	Maximum population size
r	Average number of children per pair
$F_{A_1A_1}, F_{A_1A_2}$	Fecundity of females
F_{A_1-}, F_{A_2-}	Fecundity of males

Table 2 Male and female fitness parameters: $\alpha > 0$ and $\beta < 1$

	Female			Male		
	A_1A_1	A_1A_2	A_2A_2	A_1- (homosexual)	A_1- (Non-homosexual Carrier)	A_2- (Non-homosexual)
Model 1	α	α	1	β (100 % of A_1- males)	–	1
Model 2	α	α	1	β (50 % of A_1- males)	1 (50 % of A_1- males)	1
Model 3	α	α	1	β (20 % of A_1- males)	1 (80 % of A_1- males)	1

Model 1

All females with A_1A_1 and A_1A_2 genotypes have higher fecundity than females with A_2A_2 genotype. All males with A_1 allele are homosexual and have lower fecundity than males with A_2 allele (Table 2).

Model 2

All females with A_1A_1 and A_1A_2 genotypes have higher fecundity than females with A_2A_2 genotype. Fifty percent of males with the A_1 allele are homosexual and have lower fecundity than males with the A_2 allele. Fifty percent of males with the A_1 allele are carriers, having on average similar fecundity as males with the A_2 allele. The sexual orientation of a male with A_1 is defined at each birth; selection is random and independent for siblings (Table 2).

Model 3

All females with A_1A_1 and A_1A_2 genotypes have higher fecundity than females with A_2A_2 genotype. Twenty percent of males with A_1 allele are homosexual and have lower fecundity than males with A_2 allele. Eighty percent of males with A_1 allele are carriers, having on average similar fecundity as males with A_2 allele. The sexual orientation of males with A_1 is defined at each birth; selection is random and independent for siblings (Table 2).

Results

Non-homosexual male carriers increased persistence of a polymorphic population. Average number of generations until fixation was $0.84 N_e$ ($SD = 0.56$), $1.13 N_e$ ($SD = 0.80$), and $1.37 N_e$ ($SD = 0.98$), for Models 1, 2, and 3 accordingly (Fig. 1).

Model 1 did not produce a stable polymorphic population for any combination of α and β . Probability of extinction of allele A_1 was higher than for A_2 ($M = 1.31$, $SD = 0.25$) for all combinations of α and β (Fig. 2).

Model 2 predicted stable polymorphic populations for a range of combinations of α and β (Fig. 2); the model predicted the existence of homosexuals at low frequencies (4.9–35.9 %, Fig. 3), with the same probability of extinction for both alleles.

On average, 18.9 % (3.7–35.8 %) of males and 51.5 % (12.9–96.7 %) of females were carriers.

Model 3 produced stable polymorphic populations (Fig. 2) for a wide spectrum of values of α and β . Model 3 predicted low frequencies (5.9–17.9 %, Fig. 3) of homosexuals. The percentage of female carriers varied from 40 to 60 %. On average 52.7 % (23.5–67.7 %) of males and 79.8 % (40.2–95.2 %) of females were carriers.

Discussion

Population genetics IBMs presented in this study showed that persistence of homosexuality in a human population with low and stable frequencies can be explained by X-linked inheritance coupled with increased fecundity of females carrying an allele that predisposes males to homosexuality. Higher frequency of heterosexual carriers causes models to predict increased stability of a polymorphism in a population.

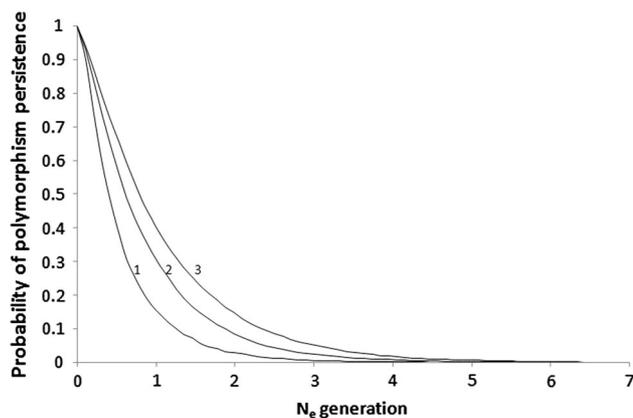


Fig. 1 Persistence of polymorphism in a population as a function of N_e generations. Average of 500 simulations. Simulations were run until one allele was lost due to genetic drift. Numbers on lines indicate model numbers

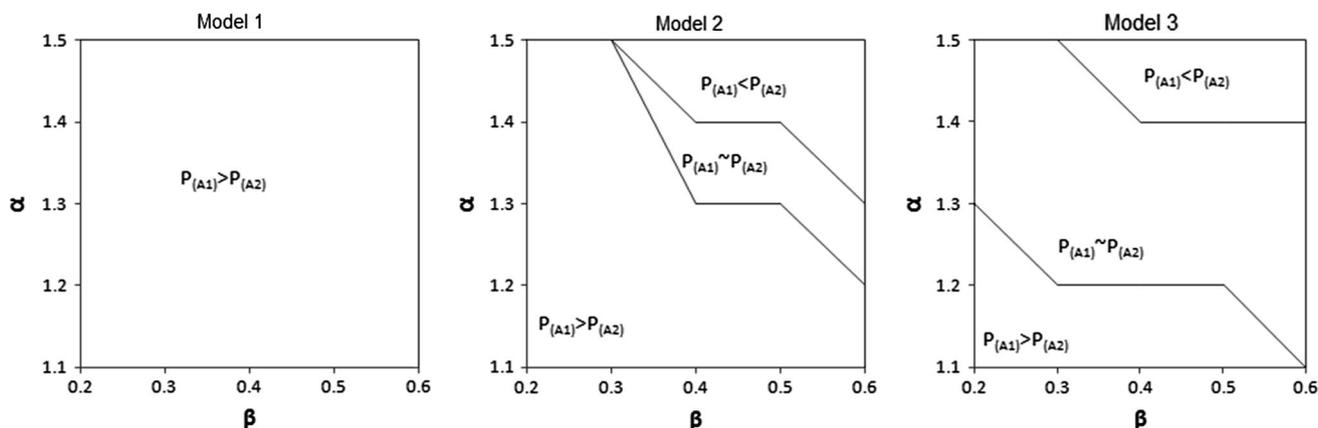


Fig. 2 Simulation results of a stochastic model: extinction probability (p) of alleles A_1 and A_2

Model 1 replicated results first reported by Ciani et al. (2008) that single locus X-linked model cannot explain persistence of homosexuality. For all combinations of α and β , A_1 has a higher probability of extinction than A_2 , without heterosexual male carriers. However, Models 2 and 3 predicted wide ranges of parameters where homosexuality can exist with low and stable frequencies in a polymorphic population.

Results of stochastic model simulations presented here represent mean values and have high fluctuations between generations (Fig. 4). Models predicted that stable polymorphic population can exist, with large percentage of non-homosexual carriers.

A large percentage of non-homosexual carriers was also expected from twin studies, because only 20–50 % of those who have genes predisposing to homosexuality will be homosexual. Sanders et al. (2014) emphasized that “genetic contributions are far from determinant but instead represent a part of the trait’s multifactorial causation, both genetic and environmental.” These current models indicate that trends apparent in empirical data— increase in maternal fecundity among relatives of homosexual men— can help explain persistence of homosexuality, if we also consider that those males who have such genes are not always homosexuals.

The possibility of significant numbers of heterosexual male carriers can also explain empirical data that estimates of men who report any lifetime same-sex sexual behavior and same-sex sexual attraction are much higher than estimates of those who identify as homosexual or bisexual. Ellis, Robb, and Burke (2005) surveyed U.S. and Canadian college students and found that only 3 % of men identified as homosexual or bisexual; however 8 % reported “1–10 % same-sex attraction.” Gates (2011) reported that only 2.8 % of adult males in the U.S. identify as gay or bisexual; however, 5.6 % report lifetime same-sex sexual contact. Vrangalova and Savin-Williams (2010) reported that 43 % of self-identified heterosexuals had some same-sex attraction, and 22 % had same-sex sexual fantasies. Sell et al. (1995) found that 8.7, 7.9, and 8.5 % of males in the U.S., the United Kingdom, and France, respectively, had some homosexual attraction but no homosexual

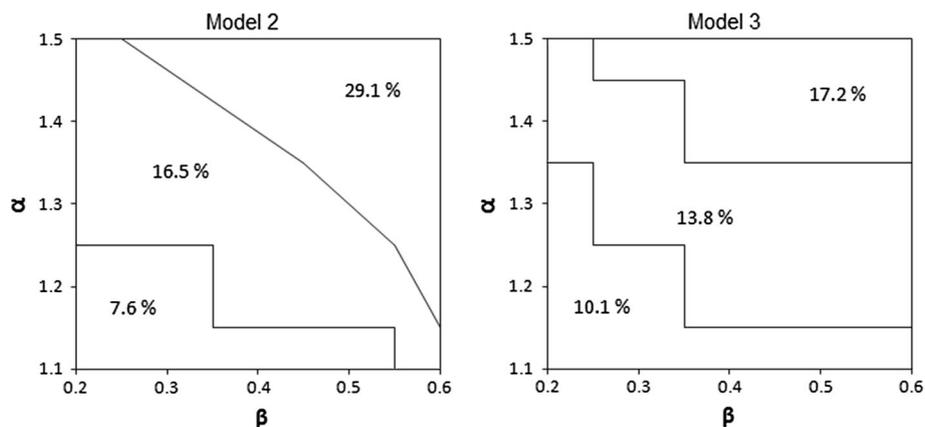


Fig. 3 Simulation results of a stochastic model: average prevalence of male homosexuality in a population

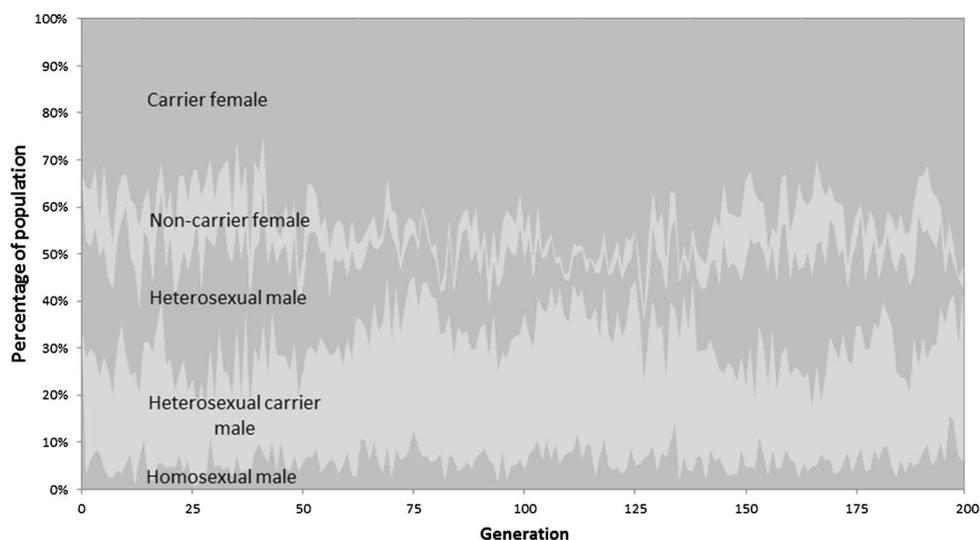


Fig. 4 Simulation results of stochastic Model 3. Parameter values: $\alpha = 1.3$; $\beta = 0.3$; $N = 100$

behavior after age 15. It could be that non-homosexual male carriers might sometimes manifest interest in homosexual behavior without having a homosexual identity.

The potentially large fraction of heterosexual males who could carry alleles predisposing to homosexuality has implications for genomic studies, because individuals included in a study as heterosexuals might have genes predisposing to homosexuality. Inclusion of individuals without homosexual relatives will reduce such influence but will not eliminate bias. This might be a reason for controversial results reported about linking male homosexuality to Xq28 region (Mustanski et al., 2005; Rice et al., 1999).

Despite being widely accepted, the higher concordance of sexual preferences in MZ twins might not necessarily represent heredity, but can be the result of similar rearing conditions. MZ

twins are raised more similarly than DZ twins: they are more likely to be dressed similarly, share the same room, and have common friends (Evans & Martin, 2000; Richardson & Norgate, 2005; Seligman, 2009). Joseph (2000) cites questionnaire studies of MZ and DZ pairs; MZ twins being “brought up as a unit” (72 vs. 19%) and being “inseparable as children” (73 vs. 19%). The influence of similar rearing conditions is best studied by twins reared apart, such as a study described by Eckert, Bouchard, Bohlen, and Heston (1986) based on data from the Minnesota Study of Twins Reared Apart on monozygotic twins. Conceptually, this study is very important; however, sampling was based on 6 MZ twins, from which 1 pair was concordant in sexual preferences, and concordance of the second pair was not clear: One set of twins had homosexual experience, but in the study rated himself as exclusively heterosexual, and has a wife

and four children. Eckert et al. stated: “That the twins are highly selected cannot be doubted; they are not representative of twins or homosexuals.” This study thus cannot be used as an argument to exclude postnatal factors influencing the development of sexual orientation in twins. Hopefully, the results of the present study will contribute to dialog about evolution and persistence of homosexuality in humans from computer modeling perspective.

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References

- Bailey, J. M., Dunne, M. P., & Martin, N. G. (2000). Genetic and environmental influences on sexual orientation and its correlates in an Australian twin sample. *Journal of Personality and Social Psychology*, *78*, 524–536.
- Bailey, J. M., & Pillard, R. C. (1991). A genetic study of male sexual orientation. *Archives of General Psychiatry*, *48*, 1089–1096.
- Borrill, P. L., & Tesfatsion, L. (2011). Agent-based modeling: The right mathematics for the social sciences? In J. B. Davis & D. W. Hands (Eds.), *Elgar companion to recent economic methodology* (pp. 228–259). Cheltenham, UK: Edward Elgar Publishing.
- Ciani, A. C., Cermelli, P., & Zanzotto, G. (2008). Sexually antagonistic selection in human male homosexuality. *PLoS One*, *3*(6), e2282. doi:10.1371/journal.pone.0002282.
- Ciani, A. C., Corna, F., & Capiluppi, C. (2004). Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. *Proceedings of the Royal Society of London B: Biological Sciences*, *271*, 2217–2221.
- Ciani, A. C., & Pellizzari, E. (2012). Fecundity of paternal and maternal non-parental female relatives of homosexual and heterosexual men. *PLoS One*, *7*(12), e51088. doi:10.1371/journal.pone.0051088.
- Diamond, M. (1993). Homosexuality and bisexuality in different populations. *Archives of Sexual Behavior*, *22*, 291–310.
- Eckert, E. D., Bouchard, T. J., Bohlen, J., & Heston, L. L. (1986). Homosexuality in monozygotic twins reared apart. *British Journal of Psychiatry*, *148*, 421–425.
- Ellis, L., Robb, B., & Burke, D. (2005). Sexual orientation in United States and Canadian college students. *Archives of Sexual Behavior*, *34*, 569–581.
- Evans, D. M., & Martin, N. G. (2000). The validity of twin studies. *GeneScreen*, *1*(2), 77–79.
- Gates, G. (2011). *How many people are lesbian, gay, bisexual and transgender?* Los Angeles: The Williams Institute of the University of California.
- Gavrilets, S., & Rice, W. R. (2006). Genetic models of homosexuality: Generating testable predictions. *Proceedings of the Royal Society of London B: Biological Sciences*, *273*, 3031–3038.
- Getz, W. M. (1993). Invasion and maintenance of alleles that influence mating and parental success. *Journal of Theoretical Biology*, *162*, 515–537.
- Grimm, V., & Railsback, S. F. (2013). *Individual-based modeling and ecology*. Princeton, NJ: Princeton University Press.
- Grulich, A. E., Visser, R. O., Smith, A., Rissel, C. E., & Richters, J. (2003). Sex in Australia: Homosexual experience and recent homosexual encounters. *Australian and New Zealand Journal of Public Health*, *27*, 155–163.
- Haldane, J. B. S., & Jayakar, S. D. (1964). Equilibria under natural selection at a sex-linked locus. *Journal of Genetics*, *59*, 29–36.
- Hamer, D. H., Hu, S., Magnuson, V. L., Hu, N., & Pattatucci, A. M. (1993). A linkage between DNA markers on the X chromosome and male sexual orientation. *Science*, *261*, 321–327.
- Heston, L. L., & Shields, J. (1968). Homosexuality in twins: A family study and a registry study. *Archives of General Psychiatry*, *18*, 149–160.
- Hutchinson, G. E. (1959). A speculative consideration of certain possible forms of sexual selection in man. *American Naturalist*, *93*, 81–91.
- Iemmola, F., & 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.
- Joseph, J. (2000). Not in their genes: A critical view of the genetics of attention-deficit hyperactivity disorder. *Developmental Review*, *20*, 539–567.
- Kallmann, F. J. (1952). Comparative twin study on the genetic aspects of male homosexuality. *Journal of Nervous and Mental Disease*, *115*, 283–298.
- Kendler, K. S., Thornton, L. M., Gilman, S. E., & Kessler, R. C. (2000). Sexual orientation in a US national sample of twin and nontwin sibling pairs. *American Journal of Psychiatry*, *157*, 1843–1846.
- Kimura, M. (1962). On the probability of fixation of mutant genes in a population. *Genetics*, *47*, 713.
- King, M., Green, J., Osborn, D. P., Arkell, J., Hetherington, J., & Pereira, E. (2005). Family size in White gay and heterosexual men. *Archives of Sexual Behavior*, *34*, 117–122.
- Kirk, K. M., Bailey, J. M., Dunne, M. P., & Martin, N. G. (2000). Measurement models for sexual orientation in a community twin sample. *Behavior Genetics*, *30*, 345–356.
- Kirkpatrick, R. C. (2000). The evolution of human homosexual behavior. *Current Anthropology*, *41*, 385–413.
- Långström, N., Rahman, Q., Carlström, E., & Lichtenstein, P. (2010). Genetic and environmental effects on same-sex sexual behavior: A population study of twins in Sweden. *Archives of Sexual Behavior*, *39*, 75–80.
- MacIntyre, F., & Estep, K. W. (1993). Sperm competition and the persistence of genes for male homosexuality. *Biosystems*, *31*, 223–233.
- Maynard-Smith, J. M., & Slatkin, M. (1973). The stability of predator-prey systems. *Ecology*, *54*, 384–391.
- Mustanski, B. S., DuPree, M. G., Nievergelt, C. M., Bocklandt, S., Schork, N. J., & Hamer, D. H. (2005). A genomewide scan of male sexual orientation. *Human Genetics*, *116*, 272–278.
- Nash, G. (2001). The subversive male: Homosexual and bestial images on European mesolithic rock art. In L. Bevan (Ed.), *Indecent exposure: Sexuality, society and the archaeological record* (pp. 43–55). Glasgow, UK: Cruithne Press.
- Rice, G., Anderson, C., Risch, N., & Ebers, G. (1999). Male homosexuality: Absence of linkage to microsatellite markers at Xq28. *Science*, *284*, 665–667.
- Richardson, K., & Norgate, S. (2005). The equal environments assumption of classical twin studies may not hold. *British Journal of Educational Psychology*, *75*, 339–350.
- 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.
- Roughgarden, J. (2012). *Individual based models in ecology: An evaluation, or how not to ruin a good thing*. Paper presented at the Philosophy of Science Association Biennial Meeting, San Diego, CA. Retrieved from http://philsci-archive.pitt.edu/9434/1/Roughgarden_PSA2012IBMLecture.pdf.
- Sanders, A. R., Martin, E. R., Beecham, G. W., Guo, S., Dawood, K., Rieger, G., ... Bailey, J. M. (2014). Genome-wide scan demonstrates significant linkage for male sexual orientation. *Psychological Medicine*, *45*, 1379–1388.
- Seligman, M. E. (2009). *What you can change and what you can’t: The complete guide to successful self-improvement*. New York: Vintage.

- Sell, R. L., Wells, J. A., & Wypij, D. (1995). The prevalence of homosexual behavior and attraction in the United States, the United Kingdom and France: Results of national population-based samples. *Archives of Sexual Behavior*, 24, 235–248.
- Taylor, J. E., & Jaenike, J. (2002). Sperm competition and the dynamics of X chromosome drive: Stability and extinction. *Genetics*, 160, 1721–1731.
- Taylor, J. E., & Jaenike, J. (2003). Sperm competition and the dynamics of X chromosome drive in finite and structured populations. *Annales Zoologici Fennici*, 40, 195–206.
- Van de Ven, P., Rodden, P., Crawford, J., & Kippax, S. (1997). A comparative demographic and sexual profile of older homosexually active men. *Journal of Sex Research*, 34, 349–360.
- Vasey, P. L., & VanderLaan, D. P. (2007). Birth order and male androphilia in Samoan fa'afafine. *Proceedings of the Royal Society of London B: Biological Sciences*, 274, 1437–1442.
- Vrangalova, Z., & Savin-Williams, R. C. (2010). Correlates of same-sex sexuality in heterosexually identified young adults. *Journal of Sex Research*, 47, 92–102.
- Whitam, F. L., Diamond, M., & Martin, J. (1993). Homosexual orientation in twins: A report on 61 pairs and three triplet sets. *Archives of Sexual Behavior*, 22, 187–206.
- Wilson, E. O. (1975). *Sociobiology: The new synthesis*. Cambridge, MA: Harvard University Press.
- Zietsch, B. P., Morley, K. I., Shekar, S. N., Verweij, K. J., Keller, M. C., Macgregor, S., ... Martin, N. G. (2008). Genetic factors predisposing to homosexuality may increase mating success in heterosexuals. *Evolution and Human Behavior*, 29, 424–433.