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

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Received: 23 March 2015 / Revised: 30 September 2015 / Accepted: 15 March 2016

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; Gruilich, 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),
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, Gavrilets 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; Gavrilets & 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 A1 and A2, with A1 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. 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 A1 and A2: 50% of males with A1- and 50% of males with A2-; 50% of homoygous females with A1A1 and 50% of females with A2A2. All models are sexually antagonistic—females with A1 allele accrue a reproductive advantage, and males with A1 allele do not. Advantage is defined by increased average fecundity. If the father has A1 allele and is homosexual, fecundity is reduced. If the father has A1 allele and is heterosexual (carrier male), fecundity is the same as the population average (Table 2).

Female and male carrier fecundity parameters:

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1. PHP script and MYSQL table instructions are available upon request by email.
\[ x = \frac{F_{A_1A_1}}{F_{A_2A_2}} = \frac{F_{A_1A_2}}{F_{A_1A_2}} > 1 \quad \text{and} \quad \beta = \frac{F_{A_1-}}{F_{A_2-}} \]  

(1)

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

\[ r_{t+1} = r_t \frac{N_{\text{max}}}{N_t} \]  

(2)

where \( N_{\text{max}} \) 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

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>( x )</td>
<td>Female carrier fecundity parameter (( x &gt; 1 ))</td>
</tr>
<tr>
<td>( \beta )</td>
<td>Male carrier fecundity parameter (( \beta &lt; 1 ))</td>
</tr>
<tr>
<td>( N )</td>
<td>Total population size</td>
</tr>
<tr>
<td>( N_e )</td>
<td>Effective population size (Number of breeding pairs ( \times 2 ))</td>
</tr>
<tr>
<td>( N_f )</td>
<td>Number of females in the population</td>
</tr>
<tr>
<td>( N_m )</td>
<td>Number of males in the population</td>
</tr>
<tr>
<td>( N_t )</td>
<td>Population size of ( t ) generation</td>
</tr>
<tr>
<td>( N_{\text{max}} )</td>
<td>Maximum population size</td>
</tr>
<tr>
<td>( r )</td>
<td>Average number of children per pair</td>
</tr>
<tr>
<td>( F_{A_1A_1} ), ( F_{A_1A_2} )</td>
<td>Fecundity of females</td>
</tr>
<tr>
<td>( F_{A_1-} ), ( F_{A_2-} )</td>
<td>Fecundity of males</td>
</tr>
</tbody>
</table>

Table 2 Male and female fitness parameters: \( x > 0 \) and \( \beta < 1 \)

<table>
<thead>
<tr>
<th>Model</th>
<th>Female</th>
<th>Male</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>( A_1A_1 )</td>
<td>( A_1A_2 )</td>
</tr>
<tr>
<td>Model 1</td>
<td>( x )</td>
<td>( x )</td>
</tr>
<tr>
<td>Model 2</td>
<td>( x )</td>
<td>( x )</td>
</tr>
<tr>
<td>Model 3</td>
<td>( x )</td>
<td>( x )</td>
</tr>
</tbody>
</table>

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 \( x \) and \( \beta \). Probability of extinction of allele \( A_1 \) was higher than for \( A_2 (M = 1.31, SD = 0.25) \) for all combinations of \( x \) and \( \beta \) (Fig. 2).

Model 2 predicted stable polymorphic populations for a range of combinations of \( x \) and \( \beta \) (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 $\alpha$ and $\beta$. 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.

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 $\alpha$ and $\beta$, $A_1$ 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...
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 implica-
tions for genomic studies, because individuals included in a
study as heterosexuals might have genes predisposing to homo-
sexuality. 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 & Nor-
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.

Acknowledgments I thank Lexo Gavashelishvili and David Tarkhnishvili for providing valuable suggestions on manuscript. I express my gratitude to Ryan Erickson and Cort Anderson for improving the use of English in the article. I also thank three anonymous reviewers and the Editor for their helpful comments.

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