

Genetic factors predisposing to homosexuality may increase mating success in heterosexuals[☆]

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Abstract

There is considerable evidence that human sexual orientation is genetically influenced, so it is not known how homosexuality, which tends to lower reproductive success, is maintained in the population at a relatively high frequency. One hypothesis proposes that while genes predisposing to homosexuality reduce homosexuals' reproductive success, they may confer some advantage in heterosexuals who carry them. However, it is not clear what such an advantage may be. To investigate this, we examine a data set where a large community-based twin sample ($N=4904$) anonymously completed a detailed questionnaire examining sexual behaviors and attitudes. We show that psychologically masculine females and feminine men are (a) more likely to be nonheterosexual but (b), when heterosexual, have more opposite-sex sexual partners. With statistical modelling of the twin data, we show that both these relationships are partly due to pleiotropic genetic influences common to each trait. We also find a trend for heterosexuals with a nonheterosexual twin to have more opposite-sex partners than do heterosexual twin pairs. Taken together, these results suggest that genes predisposing to homosexuality may confer a mating advantage in heterosexuals, which could help explain the evolution and maintenance of homosexuality in the population.

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1. Introduction

Estimates of the prevalence of homosexuality in modern Western populations vary greatly depending on how it is defined and measured and on how the samples are ascertained, but are generally in the approximate range of 1–10% and are usually lower for females than for males (Grulich, de Visser, Smith, Rissel, & Richters, 2003; Johnson, Wadsworth, Wellings, Bradshaw, & Field, 1992; Michael, Gagnon, Laumann, & Kolata, 1995). Research suggests that sexual orientation is influenced, in part, by

genetic factors. Homosexuality tends to run in families (Bailey & Bell, 1993; Pattatucci & Hamer, 1995); in particular, identical [monozygotic (MZ)] twins, who share all of their genes, are more likely to be concordant in their sexuality than are nonidentical [dizygotic (DZ)] twins, who only share, on average, half of their genes (Kendler, Thornton, Gilman, & Kessler, 2000; Kirk, Bailey, Dunne, & Martin, 2000).

In contemporary Western societies, homosexual individuals tend to have fewer children than heterosexual individuals (Bell & Weinberg, 1978; Bell, Weinberg, & Hammersmith, 1981; Pattatucci & Hamer, 1995; Van de Ven, Rodden, Crawford, & Kippax, 1997), and lowered reproductive fitness in homosexuals may have been the case in ancestral times as well (Pillard & Bailey, 1998). How, then, has homosexuality evolved, and how is it maintained in the population at a relatively high frequency? Numerous

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theoretical explanations have been proposed for this “Darwinian paradox,” many of which are critically reviewed in Rahman and Wilson (2003). Most of these theories are based on the idea that relatives of homosexuals are somehow at a reproductive advantage and thereby pass on their genes and balance the fitness cost of the homosexual phenotype. Indeed, recent evidence suggests that homosexual men, compared to heterosexual men, tend to come from larger families (Camperio-Ciani, Corna, & Capiluppi, 2004; King et al., 2005), which has been interpreted as greater fecundity in relatives of homosexual men. However, it could also be a corollary of the frequently observed “birth-order effect,” where males with a greater number of *older* brothers (Camperio-Ciani et al., 2004; Cantor, Blanchard, Paterson, & Bogaert, 2002) and sisters (Bogaert, 1998; King et al., 2005) are more likely to be homosexual; if homosexual men tend to be higher in birth order, it follows that they will tend to come from larger sibships and—if fecundity runs in families—from larger extended families as well.

If relatives of homosexuals *are* at a reproductive advantage, why might this be? The most frequently cited explanation is the kin selection or kin altruism model, where it is proposed that homosexuals provided resources and child care to family members. In doing this, homosexuals could have increased the reproductive fitness of their family members and hence increased their inclusive fitness, even while not having their own children. Although theoretically possible, this theory is generally not supported by empirical evidence (Bobrow & Bailey, 2001; Rahman & Hull, 2005).

A less discussed hypothesis is that genes predisposing to homosexuality are advantageous in heterosexuals who carry them. In this hypothesis, the genes should have the same effect on heterosexuals and homosexuals, and there should be a corresponding trait that is associated with homosexuality but confers some selective advantage in heterosexuals. The traits most reliably associated with homosexuality relate to masculinity–femininity; homosexual men tend to be more feminine than heterosexual men, and homosexual women tend to be more masculine than heterosexual women. Could this sex atypicality be advantageous when expressed in heterosexuals? Although perhaps counterintuitive, there is evidence that females are more attracted to males with certain feminine behavioral traits such as tenderness, considerateness, and kindness (Buss & Barnes, 1986; Howard, Blumstein, & Schwartz, 1987). They also prefer men with feminized faces (Boothroyd, Jones, Burt, & Perret, 2007; Penton-Voak et al., 1999; Perrett et al., 1998; Rhodes, Hickford, & Jeffery, 2000), although the preferred degree of feminization differs across the menstrual cycle and between short-term and long-term mating goals (Penton-Voak et al., 1999). There has been less research on what masculine traits in females may be attractive to men, but it should be noted that masculine traits such as competitiveness (Deaner, 2006) and unrestricted sociosexuality (Ostovich & Sabini, 2004) (willingness to engage in uncommitted sexual relations; Simpson & Gangestad, 1991) could contribute to a mating

advantage. Indeed, psychologically more masculine women have a greater number of opposite-sex sexual partners in their lifetime (Mikach & Bailey, 1999).

Our hypothesis is that a number of pleiotropic (more than one effect) genes predispose to homosexuality but also contribute to reproductive fitness in heterosexuals. In the case of males, there are a number of alleles that promote femininity; if only a few of these alleles are inherited, reproductive success is enhanced via increased levels of attractive but typically feminine traits such as kindness, sensitivity, empathy, and tenderness. However, if a large number of alleles are inherited, even the feminine characteristic of attraction to males is produced. In females, the converse explanation could be used—a low gene dose could lead to advantageous typically masculine characteristics such as sexual assertiveness or competitiveness, and a large dose could further lead to attraction to females. This hypothesis was proposed in detail by Miller (2000), but it has not been tested. Here we empirically test the hypothesis using questionnaire responses from a large ($N=4904$) community-based twin sample. Before doing so, though, we need to clarify how we conceptualize the major variables involved: sexual orientation, gender identity, and mating success.

1.1. *Sexual orientation*

Previous taxometric analyses of our data suggest that a dichotomous factor underlies the full range of sexual orientations in men and women, and that a much higher percentage (up to 15% for men and 10% for women) belong to the taxa associated with homosexuality than actually consider themselves homosexual (Gangestad, Bailey, & Martin, 2000). For the purposes of the following analyses, we operationally define those with any degree of sexual attraction to the same sex (using the 7-point Kinsey attraction scale) as nonheterosexuals, and the associated trait as nonheterosexuality. This gives us a rate of 11% for men and 13% for women, comparable with the taxa percentages given above. Note that we use sexual attraction as the indicator, rather than sexual behavior or sexual identity, because we believe that it is less affected by mate availability and social/cultural constraints and is, in this sense, more fundamental (Sell, Wells, & Wypij, 1995).

1.2. *(Continuous) gender identity*

We use the term “gender identity” to refer to masculine–feminine self-concept. Our conceptualization and scale imply that this trait is continuous (from no identification with the other sex to strong identification with it), whereas others often treat it as a dichotomous trait. Gender identity is not well understood (Bailey, 2003). Early approaches to identifying basic dimensions of personality were based on factor analyses of pools of personality adjectives, from which descriptors that differed between sexes were excluded (Schmitt & Buss, 2000). Presumably as a result, aspects of gender, including gender identity, have been relatively ignored in the personality literature. The most common

related measures are based on traditional masculine or feminine sex roles [e.g., Bem's (1974) Sex Role Inventory] that may have changed substantially since the design of the test. In the present study, we assess the degree to which the respondent identifies with the opposite gender (sex-atypical gender identity). Unless specified otherwise, we will use the terms 'masculine' and 'feminine' as shorthand for a more masculine gender identity or a more feminine gender identity, respectively.

1.3. Mating success

An obstacle to measuring reproductive fitness is that the most direct evolutionarily informative measure (i.e., number of viable offspring) is, in today's Western societies, confounded by family planning and contraception. People today generally *decide* whether to have children and how many to have, and the decision may be based on career choices, lifestyle factors, financial constraints, religious considerations, government policy, and so on. Whether a man or woman has children or not, and how many he or she has, can be independent of reproductive sex because of modern contraception methods and artificial insemination. Therefore, we will look at the lifetime number of opposite-sex sexual partners. Although certainly not an ideal indicator of reproductive fitness, we believe that the "free market" nature of mate acquisition may reflect the processes by which sexual behavior (and orientation) evolved. Although the number of sex partners may not directly reflect reproductive fitness for females, there is evidence that it is associated with attractiveness to certain a degree and may also reflect success at short-term mating strategies that are a normal part of the overall female mating strategy. Perusse (1993) also considered the problem of testing evolutionary hypotheses about mate choice in modern societies. He argued that differential partner acquisition among members of one sex reflects, to an extent, their attractiveness to the opposite sex and is therefore useful in studying sexual selection. Nevertheless, as we cannot be certain of evolutionary context and conditions, and how they have changed over time, there is a necessary degree of speculation behind our hypotheses, and the results should be treated with appropriate caution.

Using these variables (sexual orientation, gender identity, and number of opposite-sex partners), we test our general hypothesis described above by assessing more specific hypotheses:

Hypothesis 1. Sex-atypical gender identity is associated both with nonheterosexuality and, in heterosexuals, with an elevated number of opposite-sex partners.

Hypothesis 2. These associations are due, in part, to the same genetic factors influencing each trait. As further evidence that genes predisposing to nonheterosexuality are advantageous in heterosexuals, we predict that:

Hypothesis 3. Heterosexuals who have a nonheterosexual twin will have an elevated number of sex partners.

2. Methods

2.1. Participants

The community-based sample consisted of 4904 (1824 male and 3080 female) Australian twins reared together, ranging in age from 19 to 52 years. This included 1907 complete twin pairs (667 female MZ, 312 male MZ, 377 female DZ, 185 male DZ, and 366 opposite-sex DZ) and 1090 single twins. In 1991–1992, participants anonymously completed a mailed questionnaire about their sexual behavior and attitudes, as well as personality and demographic information. Further details of the sample, data collection, and zygosity determination are described elsewhere (Kirk et al., 2000).

2.2. Measures

2.2.1. Sexual orientation

We used a Kinsey-type sexual attraction measure, which asked "Which of the following best describes your sexual feelings at present?" There were seven response options ranging (for men, and *vice versa* for women) from 0 ("I am attracted to women only, never to men"), 1 ("I am almost always attracted to women but, on rare occasions, I am attracted to men"), through 6 ("I am attracted to men only, never to women").

2.2.2. Gender identity

Six dichotomous items were used to assess the degree to which participants' self-concept was masculine or feminine (e.g., "In many ways, I feel more similar to women [men] than to men [women]," "I don't feel very masculine," and "I pride myself on my femininity"). Items were summed to give a total score, with a high score indicating sex-atypical gender identity.

2.2.3. Number of opposite-sex partners

The number of opposite-sex partners was assessed by one item: "During your entire life, approximately how many women [men] have you had sexual contact with?" Response boxes were labelled *none*, *1 only*, *2*, *3–5*, *6–10*, *11–20*, *21–50*, and *over 50*, with these categories scored from 0 to 7. 'Sexual contact' was defined on the questionnaire as any activity that made the respondent sexually excited and in which one's genitals made contact with any part of the other person.

Each variable was converted into ordered categories for analysis because most people had very low scores on the first two measures, which therefore could not be analyzed as normal continuous variables. Sexual orientation was dichotomized into those scoring 0 (*exclusively heterosexual*) in one category and those scoring ≥ 1 (*homosexual attraction of any degree*) in the other category. Gender identity was divided into four categories, and the number of opposite-sex partners was reduced to six categories for the bivariate genetic threshold modelling, but left in its raw form (with normal distribution analyzed as a continuous variable) for univariate phenotypic tests.

Table 1
Twin correlations for each zygosity and each variable, with 95% confidence intervals

Zygosity	Twin correlations (95% confidence intervals)		
	Sex partners	Gender identity	Sexual orientation
MZ females	.61 (.56 to .66)	.25 (.15 to .35)	.47 (.30 to .61)
MZ males	.61 (.52 to .68)	.33 (.19 to .45)	.57 (.33 to .74)
DZ females	.35 (.25 to .44)	.18 (.04 to .31)	.37 (.15 to .56)
DZ males	.37 (.21 to .50)	.10 (−.07 to .27)	.20 (−.11 to .49)
DZ opposite sex	.14 (.03 to .25)	.07 (−.07 to .20)	−.01 (−.26 to .25)

2.3. Analyses

All analyses employed maximum-likelihood modelling procedures using the statistical package Mx (Neale, Boker, Xie, & Maes, 2006), which accounts for twin status. Analyses were performed on raw ordinal data, where it is assumed that thresholds delimiting the different categories overlay a normally distributed continuum of liability. In maximum-likelihood modelling, the goodness-of-fit of a model to the observed data is distributed as chi square (χ^2), and the number of unknown parameters (those to be estimated) is reflected by degrees of freedom (*df*). By testing the change in chi square ($\Delta\chi^2$) against the change in degrees of freedom (Δdf), we can test whether dropping model parameters or constraining them to be equal significantly worsens the model fit. In this way, we can test specific hypotheses regarding those parameters.

To test the hypothesis that sex-atypical gender identity is associated both with nonheterosexuality and, in heterosexuals, with an elevated number of opposite-sex partners, models with and without gender identity included as covariates were compared to test the significance of their relationship with (a) the number of sex partners and (b) sexual orientation, with the former model excluding nonheterosexuals. To test the hypothesis that the phenotypic relationships above are due to pleiotropic genes, we used genetic modelling of the twin data, as detailed below. To assess the hypothesis that heterosexuals with a nonheterosexual twin have an elevated number of sex partners, we tested whether the mean number of opposite-sex partners for heterosexuals with nonheterosexual twins could be equated with the mean for heterosexual pairs, without significant loss of model fit. In

the models described thus far, males and females were analyzed together, but separate parameters were fitted.

2.3.1. Genetic modelling

In the classical twin design, variance in traits (and covariance between traits) is partitioned into genetic (A) and environmental [shared within twin pairs (C) and unshared (E)] sources. This can be achieved because A, C, and E influences each predict different patterns of MZ and DZ twin-pair correlations. MZ twins share all their genes, while DZ twins share only half their genes on average. Thus, if A were the sole source of variance in a trait, we would expect a twin correlation of 1 for MZ pairs and .5 for DZ pairs. Twins reared together share many of their early environmental influences (C), which may include shared home environment, parental style, womb environment, and so on. If C were the sole source of variance in a trait, we would expect a twin correlation of 1 for both MZ and DZ pairs. Twins also have environmental influences that are unshared with their cotwin (E). If E were the sole source of variance in a trait, we would expect a twin correlation of 0 for both MZ and DZ pairs. In reality, observed MZ and DZ twin correlations generally reflect a combination of A, C, and E influences, and structural equation modelling determines the combination that best matches the observed data. Cross-twin cross-trait correlations allow us to partition covariance between traits into A, C, and E in the same way as we do for variance in a single trait. An assumption of the classical twin design is that trait-relevant environments are equally similar for MZ and DZ twin pairs; tests of this assumption for personality traits (Loehlin, 1992) and homosexuality (Kendler et al., 2000) suggest that it is valid. Further details of the classical twin design can be found elsewhere (Neale & Cardon, 1992; Posthuma et al., 2003).

Before modelling variance components, we tested for heterogeneity in thresholds and twin correlations across age, sex, and zygosity (using an α level of .01). The number of opposite-sex partners and gender identity showed significant sex and age effects on the thresholds, which were accounted for in subsequent modelling. For each variable, MZ twin correlations were higher than DZ twin correlations for both male and female pairs (Table 1); correlations were not significantly different for male versus female MZ pairs, nor

Table 2
Goodness-of-fit statistics for bivariate models of gender identity and sexual orientation

	Gender identity and homosexuality	vs.	$\Delta\chi^2$	Δdf	<i>p</i>
1	Sex-limited ACE Cholesky decomposition				
2	Fix genetic correlation between DZOS twins to .5	1	0.02	1	.90
3	Equate male and female genetic parameters	2	2.00	3	.57
4	Equate male and female environmental parameters	2	3.60	3	.31
5	Drop genetic effects ^a	5	14.131	3	.003
6	Drop genetic cross-path/covariance ^a	5	9.59	1	.002
7	Drop shared environmental effects	5	0.02	3	1
8	Drop unshared environmental cross-path/covariance	7	14.74	1	<.001

The best-fitting model is in bold.

^a Main hypothesis tests.

Table 3

Goodness-of-fit statistics for bivariate models of gender identity and opposite-sex sexual partners

	Gender identity and sex partners	vs.	$\Delta\chi^2$	Δdf	<i>p</i>
1	Sex-limited ACE Cholesky decomposition				
2	Fix genetic correlation between DZOS twins to .5	1	0.58	1	.44
3	Equate male and female genetic parameters	2	2.740	3	.43
4	Equate male and female environmental parameters	3	14.65	6	.02
5	Drop genetic effects ^a	3	159.32	3	<.001
6	Drop genetic cross-path/covariance ^a	3	11.68	1	<.001
7	Drop shared environmental effects	3	12.39	6	.05
8	Drop unshared environmental cross-path/covariance	7	.903	1	.64

The best-fitting model is in bold.

^a Main hypothesis tests.

for male versus female DZ pairs. Correlations were lower for opposite-sex than same-sex DZ pairs for each variable, suggesting different causalities in males and females, but the difference was significant only for the number of opposite-sex partners.

To test our hypothesis, we fitted two bivariate models: in the first model, we examined the covariance of gender identity and sexual orientation; in the second model, we examined the covariance of gender identity and the number of opposite-sex partners, with nonheterosexuals removed from the sample (for modelling steps, see Tables 2 and 3, respectively). Note that we could not include both sexual orientation and number of opposite-sex partners in a

single model because we use the full sample for the analysis of sexual orientation, but only the heterosexual subsample for the number of opposite-sex partners. To account for the low opposite-sex DZ twin correlations, we initially allowed for sex limitation in the genetic effects. However, genetic sex limitation could be removed in both models without significant loss of model fit and with negligible change in parameter estimates. At this stage, we tested our hypotheses using a conservative α level of .005 to account for multiple tests. A significant influence of genes was tested by dropping the genetic paths in each model and by comparing model fit. Significant overlap between the genetic influences on a pair of traits was

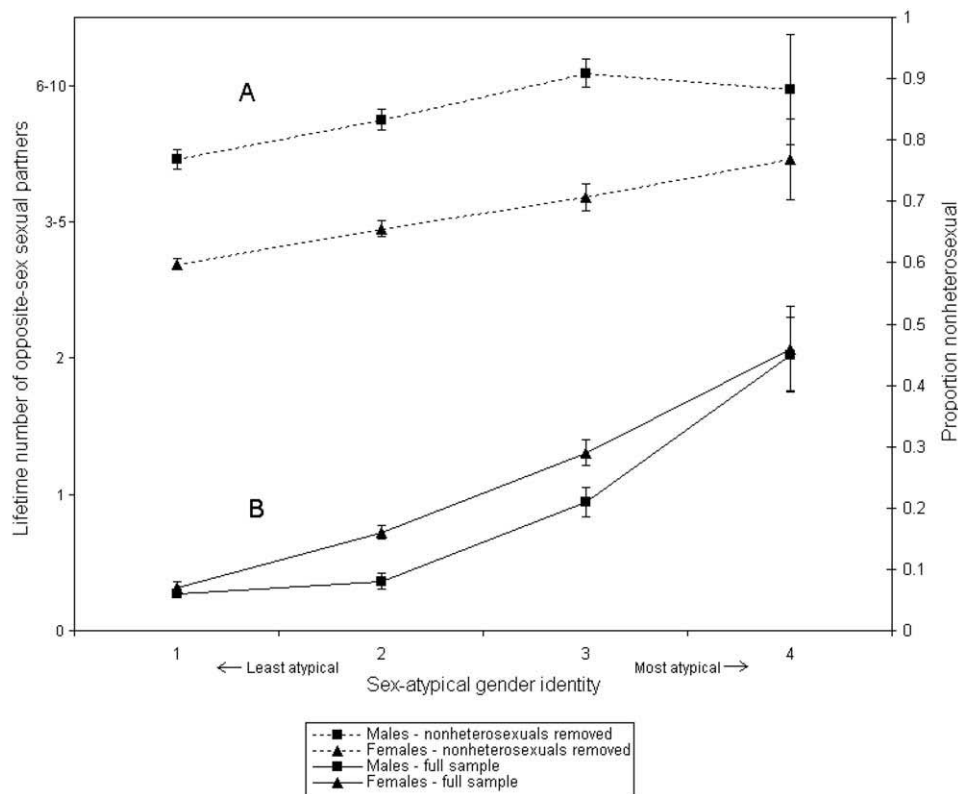


Fig. 1. Phenotypic relationship (A) between gender identity and nonheterosexuality (Kinsey 1–6) (right-hand axis), and (B) between gender identity and lifetime number of opposite-sex sexual partners (with nonheterosexuals removed from the sample) (left-hand axis). Means and standard errors for each gender identity category are displayed.

tested by dropping the genetic cross-path in each model and by comparing model fit. After the hypothesis testing, for simplicity of presentation and ease of interpretation, nonsignificant environmental parameters were dropped, and the best-fitting models were transformed from Cholesky forms into ‘correlated-factors’ models (Fig. 2), as suggested by Loehlin (1996).

3. Results

Fig. 1 shows that sex-atypical gender identity is associated both with nonheterosexuality and, in heterosexuals, with an elevated number of opposite-sex partners. Nonheterosexuals have a significantly more sex-atypical gender identity than heterosexuals in both men ($\chi^2=56.01$, $p<.001$) and women ($\chi^2=114.73$, $p<.001$). With nonheterosexuals removed from the sample, sex-atypical gender identity is significantly associated with a greater number of opposite-sex partners in both men ($\chi^2=51.15$, $p<.001$) and women ($\chi^2=70.92$, $p<.001$).

Genetic modelling results (see Tables 2 and 3) suggested that the relationships above are due to correlated genetic influences. The genetic influences could not be removed in either bivariate model without significant loss of fit, indicating that the traits are influenced by genetic factors. Also, in each model, genetic covariance between the two variables could not be removed, inferring that overlapping genetic factors are partly responsible for the relationship between gender identity and sexual orientation, and the relationship in heterosexuals between gender identity and number of opposite-sex partners. Displayed in Fig. 2 are estimates of the relative variance accounted for by genes (heritability; h^2) and environment, and estimates of genetic correlation and environmental correlation between traits. As can be seen, both genetic (A) and unshared environmental (E) factors influence each trait, but the genetic influences on the different traits are more correlated with each other than the environmental influences. Indeed, there is no significant environmental correlation between gender identity and number of sex partners.

Fig. 3 shows that heterosexuals who have a nonheterosexual twin tend to have a higher number of opposite-sex partners than heterosexuals who have a heterosexual twin, although the effect is significant only in women ($\chi^2=13.76$, $p<.001$; see Table 4 for means and standard deviations). This is consistent with our hypothesis that genes predisposing to nonheterosexuality in females increase mating success in their heterosexual twins. To see if the effect holds for exclusive homosexuality (only attracted to the same sex, never to the opposite sex), we made the same comparison but divided the sample differently—instead of dividing heterosexuals from nonheterosexuals, we divided homosexuals (exclusively attracted to the same sex) from nonhomosexuals (Table 5). As can be seen in Fig. 3B, the trend is in the same direction in this comparison and is larger in effect size.

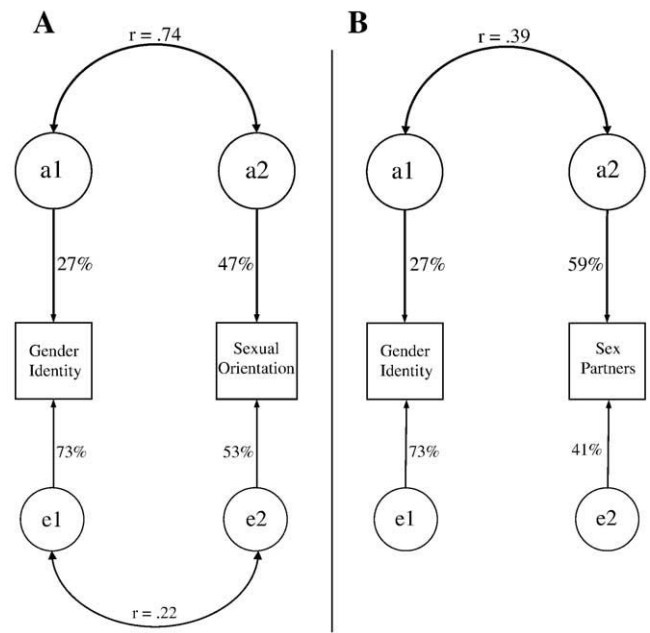


Fig. 2. Correlated-factors models showing the genetic and environmental correlations (double-headed arrows) (A) between gender identity and sexual orientation, and (B) between gender identity and number of opposite-sex partners (with nonheterosexuals removed from the sample). Percentages show the proportion of variance in each variable that is accounted for by additive genetic (a) and unshared environmental (e) influences. Correlations (r) indicate the degree to which the same genetic or environmental factors influence the different variables.

However, because of the very small sample sizes for this comparison, there is very low statistical power and, as such, standard errors are large and the effect is nonsignificant.

4. Discussion

With regard to our initial hypothesis, we have presented three lines of evidence: (a) sex-atypical gender identity is associated both with nonheterosexuality and, in heterosexuals, with an elevated number of opposite-sex partners; (b) these traits are genetically influenced, and the relationship of gender identity to both nonheterosexuality and number of opposite-sex partners is partly due to common genes; and (c) there is a trend for heterosexuals with a nonheterosexual twin to have more opposite-sex partners than twin pairs where both are heterosexual, and the trend is the same when the same comparison is made for exclusive homosexuality as opposed to nonheterosexuality. It should be noted that (c) reveals primarily nonsignificant trends in the direction hypothesized and so should be regarded only as tentative support for the main results (a) and (b).

Our evidence is consistent with a mechanism whereby some genetic variation underlying homosexuality could have been maintained over evolutionary time. The genes influencing homosexuality have two effects. First, and most obviously, these genes increase the risk for homosexuality,

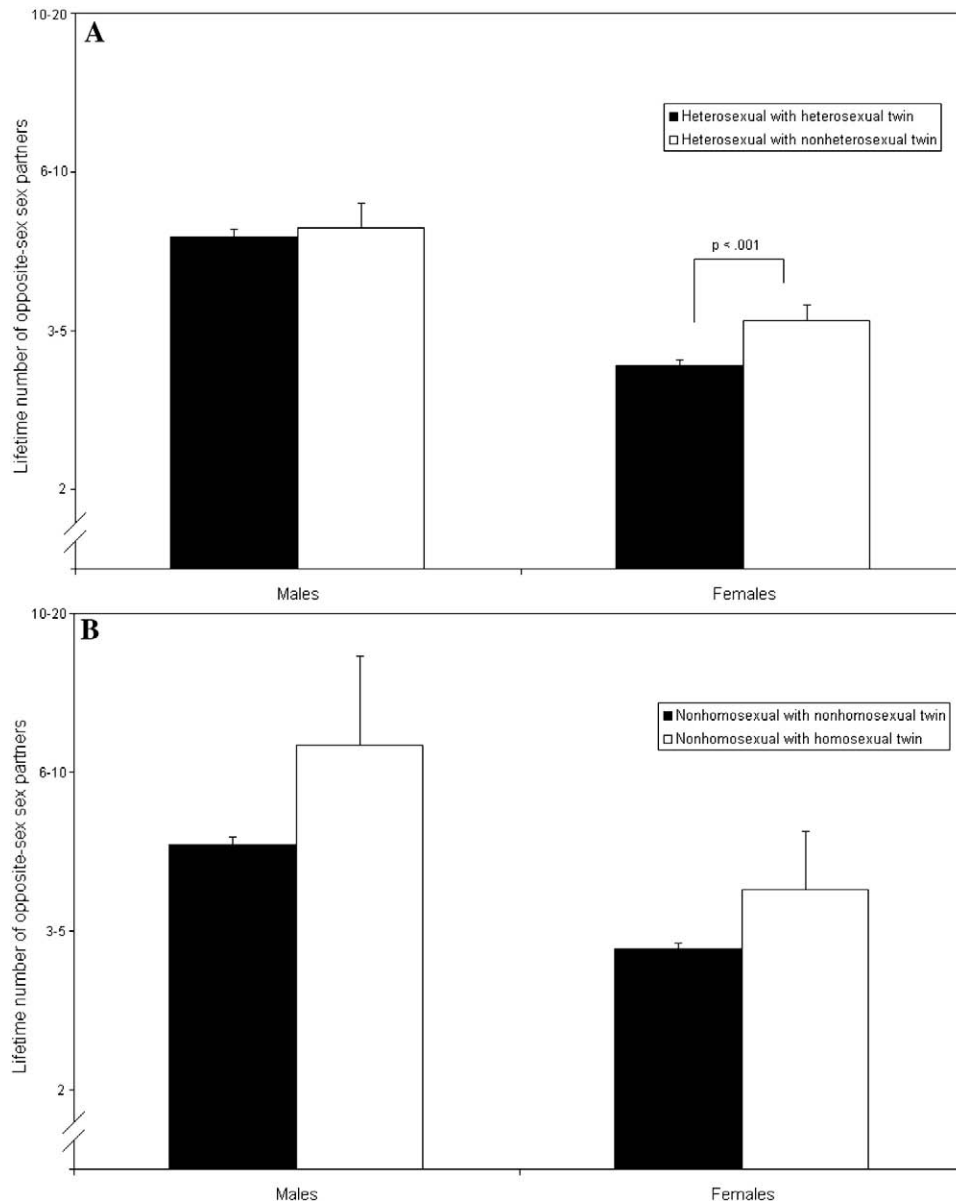


Fig. 3. (A) Mean lifetime number of opposite-sex partners of heterosexuals (Kinsey 0) with (■) a heterosexual twin and (□) a nonheterosexual (Kinsey 1–6) twin. Standard error bars are shown. (B) Mean lifetime number of opposite-sex partners of nonhomosexuals (Kinsey 0–5) with (■) a nonhomosexual twin and (□) a homosexual (Kinsey 6) twin. Standard error bars are shown.

which ostensibly has decreased Darwinian fitness. Countervailing this, however, these same genes appear to increase sex-atypical gender identity, which our results suggest may increase mating success in heterosexuals. This mechanism, called antagonistic pleiotropy, might maintain genes that increase the risk for homosexuality because they increase the number of sex partners in the relatives of homosexuals.

Gavrilets and Rice (2006) provide mathematical models whereby similar mechanisms (e.g., overdominance) could maintain genetic variation influencing sexual orientation, but others have cast doubt on the ability of antagonistic pleiotropy to maintain genetic variation indefinitely (Curtis, Service, & Prout, 1994; Hedrick, 1999). This is because natural selection should eventually drive

towards fixation at the genotype, which optimally balances the fitness costs and benefits of the different phenotypic effects. Nevertheless, antagonistic pleiotropy may considerably slow the progression towards genetic fixation, thereby maintaining genetic variation for a much longer time than would be predicted solely from the genes' fitness-lowering phenotype (homosexuality in this case). Given that we are observing only a snapshot of evolution, antagonistic pleiotropy may help to explain extant observations of genetic variation. It should be noted that linkage disequilibrium between separate genes influencing the different traits here cannot be distinguished from genuine pleiotropy in the current design. Furthermore, other mechanisms for maintaining genetic variation, such

Table 4

Mean (standard deviation) number of opposite-sex sexual partners^a for heterosexuals (Kinsey 0) with a heterosexual twin, as compared to heterosexuals with a nonheterosexual (Kinsey 1–6) twin

	With heterosexual twin	<i>n</i>	With nonheterosexual twin	<i>N</i>
Heterofemales with female twin	3.77 (1.51)	1574	4.01 (1.46)	173
Heterofemales with male twin	3.78 (1.51)	267	4.16 (1.44)	37
Heteromales with male twin	4.54 (1.72)	764	4.57 (1.66)	82
Heteromales with female twin	4.74 (1.78)	268	4.82 (1.72)	45
Total heterofemales	3.78 (1.51)	1841	4.05 (1.46)	210
Total heteromales	4.60 (1.74)	1032	4.64 (1.69)	127

n=number of individuals in each sample.

Analyses were performed in Mx to account for relatedness of twins.

^a Refers to the mean category value for the lifetime number of opposite-sex sexual partners as defined in Methods (0=0; 1=1; 2=2; 3=3–5; 4=6–10; 5=11–20; 6=21–50; 7=over 50).

as mutation–selection balance (Keller & Miller, 2006), are not mutually exclusive with the one proposed here, and a combination of mechanisms could be responsible.

Nongenetic alternative explanations cannot be ruled out. For example, a homosexual twin could create social pressure on the other twin to act in a more heterosexual way and to acquire or declare more partners than they would otherwise, or growing up with a homosexual twin could provide an individual with a privileged insight into the sexuality of the opposite sex, and hence a mating advantage. However, it is difficult to see how such effects would explain the genetic correlations between traits as revealed in our genetic modelling.

Other limitations result from limited statistical power; although our total sample is large, only a small proportion (13% of men, 11% of women) reported a nonheterosexual orientation, and a much smaller proportion (2.2% of men, 0.6% of women) reported an exclusively homosexual orientation. This afforded limited power to detect any sex differences that might have been expected in the genetic influence on sexual orientation and prevented reasonable statistical tests involving the exclusive homosexuality phenotype. A further limitation, and one that affects most research based on self-report questionnaires, is the potential for socially desirable responding. This could have been

particularly concerning here, given the sensitive nature of the questions, but the problem was minimized by ensuring that the responses were completely anonymous. Finally, we acknowledge again the absence of evidence relating the number of sexual partners and actual reproductive success, either in the present or in our evolutionary past.

Previous findings suggesting that male homosexuals tend to come from bigger families (Camperio-Ciani et al., 2004; King et al., 2005) are consistent with our results and interpretation. Camperio-Ciani et al.'s specific findings of greater fecundity in maternal but not paternal relatives would be inconsistent with our theory, but (King et al. 2005) found the effect in paternal relatives as well—if anything, there was a greater effect on the paternal line. The findings of those studies were compatible with either physiological or behavioral causes of elevated fecundity (e.g., lower abortion rate or increased ability to acquire mates, respectively; Camperio-Ciani et al., 2004) and could be explained by either genetic or environmental transmission of elevated fecundity. As explained earlier, the finding that homosexual men tend to come from bigger families could also be explained as a corollary of the birth-order effect. Our results address both male and female sexual orientations, and suggest a specific mechanism whereby pleiotropic genetic factors predispose

Table 5

Mean (standard deviations) number of opposite-sex sexual partners^a for nonhomosexuals (nonhom; anyone not exclusively homosexual; Kinsey 0–5) with a nonhomosexual twin, as compared to nonhomosexuals with an exclusively homosexual twin (Kinsey 6)

	With nonhomosexual twin	<i>n</i>	With homosexual twin	<i>n</i>
Nonhom females with female twin	3.89 (1.56)	1977	3.91 (1.38)	11
Nonhom females with male twin	3.90 (1.52)	346	4.75 (1.83)	8
Nonhom males with male twin	4.48 (1.76)	927	5.17 (1.95)	12
Nonhom males with female twin	4.69 (1.79)	347	–	0
Total nonhom females	3.89 (1.55)	2323	4.26 (1.59)	19
Total nonhom males	4.54 (1.77)	1274	5.17 (1.95)	12

n=number of individuals in each sample.

Analyses performed in SPSS do not account for relatedness of twins, as sample sizes for nonhomosexuals with homosexual twins are too small to get stable maximum-likelihood estimates in Mx. However, the means and standard deviations for Table 4 were virtually identical whether accounting for relatedness or not, so the estimates here should be reasonable.

^a Refers to the mean category value for the lifetime number of opposite-sex sexual partners as defined in Methods (0=0; 1=1; 2=2; 3=3–5; 4=6–10; 5=11–20; 6=21–50; 7=over 50).

to nonheterosexuality and increase mating success in heterosexuals via advantageous sex atypicality. While not explaining the birth-order effect, our results cannot simply be a by-product of it. In fact, Miller (2000) explains how the birth-order effect could itself be a by-product of a mechanism that shifts personalities more into the feminine direction in later-born sons, reducing the probability of these sons engaging in unproductive competition with each other.

There remain considerable gaps in our understanding that should provide avenues for future research, but the results of the present study contribute to dialogue on a prevailing conundrum—the evolution and maintenance of homosexuality in the human population.

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