ORIGINAL RESEARCH—LGBT

Common Genetic Factors among Sexual Orientation, Gender Nonconformity, and Number of Sex Partners in Female Twins: Implications for the Evolution of Homosexuality

Andrea Burri, PhD,*† Tim Spector, Professor,† and Qazi Rahman, PhD‡

*Department of Psychology, University of Zurich, Zurich, Switzerland; †Department of Twin Research and Genetic Epidemiology, King's College London, London, UK; ‡Department of Psychology, Institute of Psychiatry, King's College London, London, UK

DOI: 10.1111/jsm.12847

ABSTRACT

Introduction. Homosexuality is a stable population-level trait in humans that lowers direct fitness and yet is substantially heritable, resulting in a so-called Darwinian "paradox." Evolutionary models have proposed that polymorphic genes influencing homosexuality confer a reproductive benefit to heterosexual carriers, thus offsetting the fitness costs associated with persistent homosexuality. This benefit may consist of a "sex typicality" intermediate phenotype. However, there are few empirical tests of this hypothesis using genetically informative data in humans.
Aim. This study aimed to test the hypothesis that common genetic factors can explain the association between measures of sex typicality, mating success, and homosexuality in a Western (British) sample of female twins.
Methods. Here, we used data from 996 female twins (498 twin pairs) comprising 242 full dizygotic pairs and 256 full monozygotic pairs (mean age 56.8) and 1,555 individuals whose co-twin did not participate. Measures of sexual orientation, sex typicality (recalled childhood gender nonconformity), and mating success (number of lifetime sexual partners) were completed.
Main Outcome Measure. Variables were subject to multivariate variance component analysis.
Results. We found that masculine women are more likely to be nonheterosexual, report more sexual partners, and, when heterosexual, also report more sexual partners. Multivariate twin modeling showed that common genetic factors explained the relationship between sexual orientation, sex typicality, and mating success through a shared latent factor.
Conclusions. Our findings suggest that genetic factors responsible for nonheterosexuality are shared with genetic factors responsible for the number of lifetime sexual partners via a latent sex typicality phenotype in human females. These results may have implications for evolutionary models of homosexuality but are limited by potential mediating variables (such as personality traits) and measurement issues. **Burri A, Spector T, and Rahman Q. Common Genetic Factors among Sexual Orientation, Gender Nonconformity, and Number of Sex Partners in Female Twins: Implications for the Evolution of Homosexuality. J Sex Med 2015;12:1004–1011.**

Key Words. Evolution; Female Homosexuality; Twins; Genetics; Atypicality

Introduction

Human homosexuality is a key variant in the human sexual phenotype and of significant interest to evolutionary and behavioral biologists. Epidemiological research in Western samples suggests the trait is persistent and relatively stable, at a population level, with frequencies somewhat lower in women than men [1–3]. The rate of homosexuality as quantified via reported sexual identity labeling or any same-sex sexual contact is more stable for men than for women across age groupings within a cohort and from cohort-to-cohort in Western samples (e.g., [4]). Homosexuality in both

sexes appears persistent in spite of its reduced fitness differentials relative to heterosexuality in both Western samples and one non-Western one [5–8]. Male and female homosexuality is also modestly heritable from well-characterized and larger twin samples [9,10]. In addition, there is some evidence that male homosexuality is associated with elevated fecundity among relatives in Western and non-Western populations, although the matrilineal and/or patrilineal nature of these effects remains unresolved [11–13]. These data point to the presence of polymorphic alleles influencing homosexuality in both sexes. However, these data also pose a central "Darwinian paradox" within evolutionary biology in that selection should have eliminated alleles inducing homosexuality that reduce individual fecundity and fitness unless there was some compensatory mechanism. Resolving this paradox would be a significant advance not only in sex research but also in the broader biological sciences.

Theoretical and mathematical models have proposed two broad variants of balancing selection as putative compensatory mechanisms for human homosexuality: heterozygote advantage and sexually antagonistic selection [14–16]. Heterozygotic advantage mechanisms assume polygenic alleles predisposing towards homosexuality provide fitness benefits in heterozygous, heterosexual carriers. The fitness benefit may be mediated through a behavioral or physiological phenotype, e.g., success in attracting the opposite sex via behavioral feminine or masculine traits [16]. For example, a low dose of feminizing alleles may enhance fitness in heterosexual men via increased levels of attractive but typically feminine psychological traits in some Western cultures such as good parenting and empathy skills. However, a larger dose of these alleles, above a liability threshold, induces male homosexuality. In females, the converse explanation is proposed to hold [16]. Indeed, prospective and retrospective data show that homosexual men are, on average, more feminine in behavior, feelings, and interests during childhood compared with heterosexual men while homosexual women are more masculine in these respects relative to heterosexual women [17,18]. Evidence also suggests that women are attracted to feminine behavioral, personality, and physical traits (such as facial morphology) in men [19–22], although this depends in part on menstrual cycle changes, while masculinity in women is associated with increased lifetime number of sexual partners [23]. This behavioral feminization and masculinization is known as sex typicality and often operationalized as childhood gender nonconformity (or CGN).

Sexually antagonistic mechanisms propose that alleles inducing male homosexuality may increase female fitness but be detrimental (or indifferent) to male fecundity. This mechanism is supported by two mathematical models [14,24] and data showing that female maternal relatives (or both maternal and paternal line relatives) of homosexual men have increased fecundity compared with relatives of heterosexual men [11–13,25]. These models and behavioral data, such as fecundity rates, are only available for hypotheses regarding male homosexuality. Other behavioral data, e.g., on social preferences, which are relevant to alternative evolutionary models for the maintenance of homosexuality (such as kin selection, whereby gay relatives enhance the survival of their siblings' offspring through caregiving or resource provision), are also only available for men [26,27]. Thus, female populations are woefully under researched. Critically, empirical work using genetically informative data is lacking. One study that did use a genetically informative twin design in a Western sample (Australian) reported that sex atypicality in heterosexuals (more femininity in men and more masculinity in women) was associated with increased mating success and, expectedly, with homosexuality in both sexes [28]. These associations were found to be due to the same additive genetic factors influencing each trait in bivariate twin models. Although this study highlights the potential importance of balancing selection in maintaining homosexuality-related alleles, it did not perform comprehensive multivariate modeling of the link between sex atypicality, mating success, and sexual orientation in the twins. Here, we test the hypothesis that the common genetic factors can explain the association between measures of sex typicality, mating success, and homosexuality in a British sample of female twins. Using a more complete quantitative genetic analysis, we also test whether covariance between the traits is explained by a single underlying genetic factor or single random factor (independent pathway model) or whether a single, shared latent phenotype underlies the traits (common pathway model).

Methods

Participants

These were monozygotic (MZ) and dizygotic (DZ) volunteer female twins drawn from the "TwinsUK" registry at St. Thomas's Hospital [29].

Zygosity was established using standardized questions about physical similarity and confirmed by multiplex DNA genotyping in cases of uncertainty [30]. In 2002, twins were sent a survey asking about sexual behavior and sexual orientation (referring to "sexual attractions" with men and women in this study). Of the 8,418 questionnaires sent, 4,725 (56.1%) were returned. In a 2005 follow-up survey, an anonymous questionnaire assessing CGN (our core measure of sex typicality) was also sent to 6,934 female twins in the registry and returned by 4,850 (69.9%). The questionnaire was developed based on published scales but shortened for the purposes of practicality within a large twin register. Twins were not selected on the basis of variables being studied and were unaware of any hypothesis being tested.

In the end, questionnaire data relating to sexual orientation and CGN was available on a total of 4,426 female twin individuals. Of these, females who reported never having felt sexually attracted to anyone else (N = 44) were excluded from the analyses as were 228 (5.15%) females with missing values for any items, and 32 (0.72%) women with unknown zygosity. Of the resulting n = 4,322 individuals, final questionnaire data, where there was matching reports for sexual orientation (attractions), sex typicality (CGN), and our proxy for reproductive fitness—number of sex partners, were only available for a total of 996 individual twins comprising 242 full DZ pairs and 256 full MZ pairs (mean age 56.8) and 1,555 individuals whose co-twin did not participate. Thus, these numbers go forward into our statistical analysis reported next.

Measures and Procedure

The study was approved by the St. Thomas's Hospital research ethics committee. All study participants involved in this study provided informed written consent. Sexual orientation was measured with a Kinsey-type scale assessing sexual attraction with response options ranging from 1 ("only to/with males, never to/with females") to 5 ("only to/with females, never to/with males") with a supplementary option of "no sexual attraction." Scores of 1 were considered to be heterosexual whereas scores 2–5 were considered "non-heterosexual" in order to capture the less bimodal nature of female homosexuality [4,31]. Sexual attraction was used over sexual experience as the representative measure of sexual orientation as it is less affected by mate availability and social and cultural constraints.

Our measure of sex typicality, CGN, was retrospectively assessed with a four-item scale with response options ranging from "strongly agree" (1) to "strongly disagree" (7). Example items include "As a child I was called a tom boy by my peers" and "As a child I preferred playing with boys rather than girls." High average scores reflect feminine childhood behavior and interests.

The most direct measure of fitness is the number of direct offspring. However, estimation of this measure in today's Western societies is confounded by many socio-cultural and economic factors (e.g., availability of cheap contraception, family planning techniques, and the demographic transition in post-industrial societies). Other measures of reproductive performance have therefore been suggested, including age at first mating or number of partners [32]. Number of sexual partners in humans is closest in definitional terms to the variable understood in the biological and zoological sciences as "mating success" (i.e., the number of sexual partners over reproductive lifetime and/or the number and duration of copulation events) [33]. Accordingly, we chose lifetime number of sexual partners for our study here. The number of partners was assessed on the basis of a subject's response to the question: "Altogether, in your life so far, with how many different people (including your current partner) have you had sexual intercourse?" resulting in a continuous variable.

Statistical Analysis and Twin Modeling

The pattern of simple phenotypic associations between sexual orientation, CGN, and number of partners (for the whole sample and for heterosexual women only) was quantified using Pearson's correlations. For all analyses, a $P < 0.05$ was considered statistically significant. Descriptive and genetic analyses (twin modeling) were undertaken using STATA (StataCorp, College Station, TX, USA) and Mx software (Virginia Commonwealth University, Richmond, VA, USA). For the univariate and bivariate genetic analysis, sexual attractions, CGN, and number of partners were treated as continuous variables.

The classical twin design was used to investigate the relative contributions of genetic and environmental factors to variation and covariation in sexual attraction, CGN, and number of partners. For univariate heritability estimates, the total phenotypic variance is partitioned into additive (A), dominant (D), genetic, and shared familial (common; C) and unique to the individual (E) environmental components using a Cholesky decomposition; E incorpo-

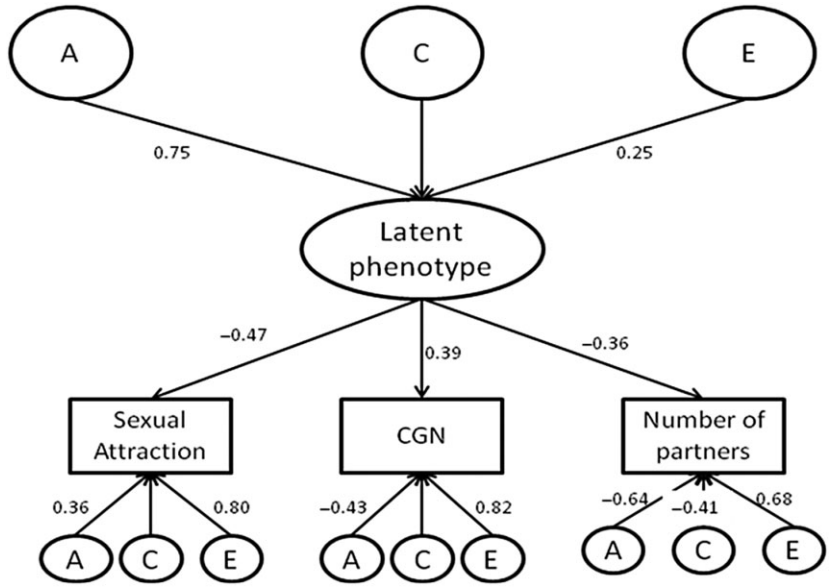

Figure 1 Path coefficients of the common pathway AE model selected as the most appropriate depiction of the data. The squares of the path coefficients provide an estimate of the variance explained by genetic and common and specific environmental components.

rates measurement error. Maximum likelihood modeling was conducted, which allows detailed modeling of the variance and covariance within MZ twin pairs compared with DZ twin pairs [34]. Univariate analyses can be extended to multivariate analyses. Three multivariate models were considered: a trivariate Cholesky decomposition (Figure 1), in which the covariance between the variables is partitioned in addition to the variance of each phenotype; the common pathway model, hypothesizing that a latent phenotypic factor intervenes between the three variables and the common genetic, shared environmental, and unique environmental factors so that the covariance among the three variables is determined by the phenotypic relationships of the three variables with the latent common factor; and the independent pathway model that posits that the common genetic and common environmental factors affect each variable directly and separately, having its own path to each variable. A goodness-of-fit test was implemented in each multivariate model to assess deterioration in model fit to the observed data using the Akaike's information criterion (AIC). We then compared the goodness-of-fit statistic values between the multivariate models and chose the best fitting model (i.e., the one that best explained the covariance structure).

Results

Phenotypic Relationships between Sexual Attractions, CGN, and Number of Sexual Partners

For the whole sample, masculine (low) scores on CGN were associated with greater nonheterosexual attractions and more sexual partners, *P*s < 0.001. When the analysis was restricted to heterosexual women, masculine scores on CGN were still associated with more sexual partners, *P* < 0.001 (Table 1).

Univariate and Multivariate Twin Modeling Analysis

Using the maximum likelihood raw data option in Mx [35,36], we tested differences in means and variances between the two zygosity groups and between twin 1 and twin 2 (numbering assigned arbitrarily upon registration and not related to birth order) for all variables. None of the variables showed a significant difference in the mean level or in variance between MZ and DZ twins or between twin 1 and twin 2 within zygosity, fulfilling assumptions for data analyses using twins.

Prior to the model-fitting analyses, the twin and cross-trait cross-twin correlations (CTCTs) for all variables were examined (Table 2). The CTCT is the correlation within a twin pair across two traits (e.g., correlation of twin 1's CGN score with twin

Table 1 Pearson's correlations between sexual attractions, CGN, and number of sexual partners in the overall sample of N = 2,551 individuals

	CGN	Number of sexual partners
Whole sample		
Sexual attractions	−0.19*	0.13*
CGN	—	−0.11*
Heterosexual women only		
CGN	—	−0.11*

*$P < .001$
CGN = childhood gender nonconformity.

Table 2 Twin and cross-twin cross-trait correlations

	Sexual attractions (n = 4,585)	CGN (n = 4,030)	Number of sexual partners (n = 4,212)	Heritability % (95% CI)
Sexual attractions	0.33/0.08	–0.04	0.10	30 (23–37)
CGN	–0.10	0.35/0.12	–0.08	32 (26–37)
Number of sexual partners	0.19	–0.15	0.52/0.17	50 (45–55)

Note. Twin correlations for MZs/DZs are presented on the diagonal. Cross-twin cross-trait correlations for MZs are presented below the diagonal. Cross-twin cross-trait correlations for DZs are presented above the diagonal.
CGN = childhood gender nonconformity; CI = confidence interval.

2's number of partner score). Twin and CTCT correlations were computed on the basis of complete twin pairs. The intraclass correlations were higher among MZ than DZ twins consistent with a genetic effect. Univariate analysis confirmed the presence of a genetic influence at each of the three variables considered separately. However, CTCT correlations in MZ twins were relatively low, which indicated a large amount of unique environmental influences on the covariation of the variables. The most appropriate model for the data contained only additive genetic and unique environmental components (the AE model). The heritabilities derived from the AE models are shown in Table 2.

Standard biometrical genetic model fitting methods suggested heritability (estimates of 30% for sexual orientation, 32% for CGN, and 50% for number of partners). The proportion of the total genetic variance between traits (genetic correlation), especially between CGN and number of partners, was high, ranging from $r_A = -0.28$ to $r_A = 0.39$. The bivariate heritability estimates further suggest that approximately 57% of the covariance between sexual attraction and CGN, 91% between CGN and number of partners, and 78% between sexual attraction and number of partners was due to additive genetic factors, with the remaining attributable to nonshared environmental factors (Table 3). In terms of multivariate analyses, a common pathway model was found to offer the most suitable explanation of the data with the lowest value of AIC at 2,340 (Table 4). The parameter estimates derived from the common pathway model are shown in Figure 1.

This common pathway model explains the variance at each site in terms of unique A and E contributions as well as a contribution from the "common phenotype" (P_c) with a heritability of 40% (0.63^2) that chiefly explains the correlation between sexual attraction, CGN, and number of partners. For sexual attraction, the common phenotype P_c accounted for 22% (0.47^2) of the variation, for CGN 15% (0.39^2), and for number of partners 13% (0.36^2). The heritability of the variation in the phenotype that was not accounted for by Pc was, respectively, 13% (0.36^2), 18% (0.43^2), and 41% (0.64^2).

Table 3 Phenotypic (R_p), genetic, and environmental correlations for sexual attraction, CGN, and number of sexual partners

	Sexual attraction—CGN	CGN—number of sexual partners	Sexual attraction—number of sexual partners
R_p	–0.19	–0.13	0.16
Proportion of R_p due to:			
A	0.57	0.91	0.78
D	0.00	0.00	0.00
E	0.43	0.09	0.22
Correlations:			
r_A	–0.37	–0.28	0.39
r_D	–0.87	–0.47	0.58
r_E	–0.11	–0.05	0.03

Note. N(MZ) = 256 pairs, N(DZ) = 242 pairs. The genetic and environmental correlations represent the correlation in additive (r_A) and dominant (r_D) genetic factors and unique environmental (r_E) factors, respectively.
CGN = childhood gender nonconformity.

Discussion

In this genetically informative design, we found that sex atypicality (measured via CGN) was associated with female homosexuality, that more masculine heterosexual women had greater numbers

Table 4 Results of multivariate modeling for the three measures using the full ADE model

	–2LL	df	AIC
Cholesky	13,700.303	5,675	2,350.303
Common pathway	*13,704.036*	*5,682*	*2,340.036*
Independent pathway	13,700.886	5,678	2,344.886

The common pathway model offered the most suitable explanation of the data. ADE is a genetic model which includes dominance genetic effects. –2LL represents the fit function of the data (–2× log likelihood). AIC represents the Akaike information criterion that describes the balance between goodness of fit and parsimony of each model: the lower the AIC the better the fit (best model is in italic typeface). df is the degrees of freedom.

of lifetime sexual partners, and that these relationships are influenced by common genetic factors via a single shared latent phenotype (with a heritability of 40%/0.63[2]). This is in line with one previous genetically informative study using similar measures [28].

Our results are consistent with the prediction from evolutionary theory that genetic variation underlying a fitness-reducing trait, female homosexuality, may be maintained over time via its potential reproductive benefits (here indexed via number of lifetime sexual partners) to heterosexual individuals [15,16]. They are consistent with Zietsch and colleagues suggestion that although these genetic factors increase the rate of homosexuality, they are buffered by also increasing sex atypical gendered behaviors and interests that may increase mating success in heterosexuals [28]. There are a range of such fluctuating selection mechanisms where the evolution of alleles for complex behavioral traits (e.g., personality, aptitudes, and other individual difference variables) acts in a "cost-benefit" fashion in order to maintain phenotypic diversity associated with differential success [37–39]. Our data cannot distinguish between all these fluctuating selection mechanisms (e.g., antagonistic pleiotropy, mutation–selection balance, overdominance) in part because they are cross-sectional. Our data do not shed light on what physiological, behavioral, and personality traits arise as a function of possessing degrees of sex atypicality that result in elevated numbers of sexual partners. Prior work in Western populations has suggested that masculinity in women might result in an increased ability to acquire mating partners but the mechanisms (e.g., higher sex drive, more aggression, or dominance in sexual encounters, or whether masculinized physical or behavioral traits in women are attractive to heterosexual men as feminine traits in men are to women) are unclear [23].

There are several limitations to our study so caution should be exercised in the interpretation of the results. Our sample in the twin analysis was small compared with the only other study on the evolution of homosexuality using twins [28]. Thus, our statistical power was quite low although is comparable with other studies using multivariate twin analyses [34]. The twin modeling software (Mx) uses structural equation modeling to estimate model parameters by minimizing a goodness-of-fit function between observed and predicted covariance matrices (e.g., by maximum likelihood methods) and is therefore less susceptible to low power caused by small twin samples. Thus, it is unclear how the relatively small sample could systematically bias our parameter estimates.

Using a retrospective measure of childhood, sex typicality may have been contaminated by recall biases. However, prospective studies confirm the predictive validity of such measures as do maternal reports of proband-recalled sex typicality and childhood home videos [18]. In addition, there was no evidence of a response difference in MZ or DZ twins, and the subjects were not aware of any specific hypotheses. The representativeness of our twin sample also diminishes any putative selection biases, as shown by a large comparative study demonstrating that our twin population is very similar to singletons on a wide range of common health and lifestyle factors [40]. Nonetheless, our results are constrained to a British population of twins answering questions that involve recalling internalized definitions of masculinity, femininity, and definitions of sexual activity that may be influenced by cultural expectations. Thus, our results should be interpreted appropriately for a Western population and not necessarily populations where definitions of masculinity and femininity (or types of sexual activity) are different. This does not negate the evolutionary context of our results because genetic predispositions may influence the internalization of cultural gender expectations and traits with high heritability do not imply that changes in culture or environment will have no phenotypic consequences.

Our statistical power may have been further limited due to small numbers of the nonheterosexual participants in our study. We attempted to increase power using a broader definition of "nonheterosexual" response scores but the skewed fundamental distribution of trait sexual orientation makes this a difficult design problem to overcome. In order to better quantify reproductive success, future studies must use numbers of actual offspring produced and validate the use of number of sexual partners with markers such as copulation events or motivations for reproductive sexual activity. Female homosexuality is consistent with a heritable basis and lower reproductive output in contemporary Western populations. However, it is not clear that lower reproductive output would have been characteristic of female homosexuality in ancestral environments or contemporary non-Western cultures. A further complication is sexual fluidity in women—if women switch from heterosexual to nonheterosexual sexual relationships, it is

not clear that this would be detrimental to fitness (more so given women have a lower reproductive ceiling than do men). The use of the phrase "sexual intercourse" in our measure of lifetime number of sexual partner may have resulted in underestimates of sexual partner number (e.g., more feminine women might have interpreted the item as strictly referencing penetrative sexual acts rather than other forms of genital sexual activity). It is also possible that a fourth variable explains the link between our measures of interest. For example, gender nonconformity may result in personality traits associated with sensation seeking and dominance that increase sexual behavior and mating success. In this case, personality traits might mediate the relationship between our three measures. Future work using twin modeling should attempt to quantify the effect of potential "mediating/moderating variables" (such as personality traits). Finally, our data only pertain to women, an under-researched population in sexual orientation sciences. Although the female focus here was a constraint of using this particular British twin register, future work must test larger numbers of male and female twin pairs to test for sex limitation in genetic effects given the putatively distinct (if somewhat overlapping) mechanisms underlying the development of male and female sexual orientation.

Our results suggest that some of the genetic factors responsible for female homosexuality may be the same as those which result in one, albeit imperfect, measure of mating success among female heterosexual carriers. Thus, they offer some support to balancing selection explanations for the maintenance of alleles promoting homosexuality. The results are also consistent with similar mechanisms demonstrated in mathematical models for male homosexuality [14,24]. However, they do not distinguish between different balancing selection explanations. Further work, using genetically informative designs, may test whether specific variants of these models apply differently to male and female homosexuality. In view of our findings and those of Zietsch et al., future investigations should also aim to identify the latent phenotype that was common to all our measures in significantly larger samples of heterosexual and nonheterosexual male and female twin sets [28]. In addition, such studies should be conducted in non-Western twin registers where these are available to test for possible cultural constraints on measures of sexual orientation, gender nonconformity, and sexual behavior.

Acknowledgment

None.

Corresponding Author: Qazi Rahman, PhD, Department of Psychology, Institute of Psychiatry, King's College London, London SE1 9RT, UK. Tel: +44 (0)20 7188 0183; Fax: +44 (0)20 7188 0184; E-mail: qazi.rahman@kcl.ac.uk

Conflict of Interest: The author(s) report no conflicts of interest.

Statement of Authorship

Category 1

(a) Conception and Design
Qazi Rahman; Andrea Burri

(b) Acquisition of Data
Andrea Burri; Tim Spector

(c) Analysis and Interpretation of Data
Andrea Burri; Qazi Rahman

Category 2

(a) Drafting the Article
Andrea Burri; Qazi Rahman; Tim Spector

(b) Revising It for Intellectual Content
Qazi Rahman; Andrea Burri; Tim Spector

Category 3

(a) Final Approval of the Completed Article
Andrea Burri; Tim Spector; Qazi Rahman

References

1 Johnson AM, Mercer CH, Erens B, Copas AJ, McManus S, Wellings K, Fenton KA, Korovessis C, Macdowall W, Nanchahal K, Purdon S, Field J. Sexual behaviour in Britain: Partnerships, practices, and HIV risk behaviours. Lancet 2001;358:1835–42.
2 Michael RT, Gagnon JH, Laumann EO, Kolata G. Sex in America: A definitive survey. Boston: Little, Brown, & Co; 1995.
3 Sell RL, Wells JA, Wypij D. The prevalence of homosexual behavior and attraction in the United States, the United Kingdom and France: Results of national population-based samples. Arch Sex Behav 1995;24:235–48.
4 Mercer CH, Tanton C, Prah P, Erens B, Sonnenberg P, Clifton S, Macdowall W, Lewis R, Field N, Datta J, Copas AJ, Phelps A, Wellings K, Johnson AM. Changes in sexual attitudes and lifestyles in Britain through the life course and over time: Findings from the National Surveys of Sexual Attitudes and Lifestyles (Natsal). Lancet 2013;382:1781–94.
5 Bell AP, Weinberg M, Hammersmith SK. Sexual preference: Its development in men and women. Bloomington, IN: Indiana University Press; 1981.
6 Bell AP, Weinberg M. Homosexualities: A study of diversity among men and women. Bloomington, IN: Indiana University Press; 1978.
7 Pattatucci AML, Hamer DH. Development and familiality of sexual orientation in females. Behav Genet 1995;25:407–20.

8 Vasey PL, Parker JL, VanderLaan DP. Comparative reproductive output of androphilic and gynephilic males in Samoa. Arch Sex Behav 2014;43:363–7.
9 Bailey JM, Dunne MP, Martin NG. Genetic and environmental influences on sexual orientation and its correlates in an Australian twin sample. J Pers Soc Psychol 2000;78:524–36.
10 Långström N, Rahman Q, Carlström E, Lichtenstein P. Genetic and environmental effects on same-sex sexual behavior: A population study of twins in Sweden. Arch Sex Behav 2010;39:75–80.
11 Camperio-Ciani A, Corna F, Capiluppi C. Evidence for maternally inherited factors favouring male homosexuality and promoting female fecundity. Proc Biol Sci 2004;271:2217–21.
12 Camperio-Ciani A, Pellizzari E. Fecundity of paternal and maternal non-parental female relatives of homosexual and heterosexual men. PLoS ONE 2012;7:e51088.
13 VanderLaan DP, Vasey PL. Male sexual orientation in independent Samoa: Evidence for fraternal birth order and maternal fecundity effects. Arch Sex Behav 2011;40:495–503.
14 Gavrilets S, Rice WR. Genetic models of homosexuality: Generating testable predictions. Proc Biol Sci 2006;273:3031–8.
15 McKnight J. Straight science? Homosexuality, evolution and adaptation. London: Routledge; 1997.
16 Miller EM. Homosexuality, birth order and evolution: Toward an equilibrium reproductive economics of homosexuality. Arch Sex Behav 2000;9:1–34.
17 Bailey JM, Zucker KJ. Childhood sex-typed behavior and sexual orientation: A conceptual analysis and quantitative review. Dev Psychol 1995;31:43–55.
18 Rieger G, Linsenmeier JAW, Gygax L, Bailey JM. Sexual orientation and childhood gender nonconformity: Evidence from home videos. Dev Psychol 2008;44:46–58.
19 Boothroyd LG, Jones BC, Burt DM, Perret DI. Partner characteristics associated with masculinity, health and maturity in male faces. Pers Individ Dif 2007;43:1161–73.
20 Buss DM, Barnes M. Preferences in human mate selection. J Pers Soc Psychol 1986;50:559–70.
21 Howard JA, Blumstein P, Schwartz P. Social or evolutionary theories? Some observations on preferences in human mate selection. J Pers Soc Psychol 1987;53:194–200.
22 Rhodes G, Hickford C, Jeffrey L. Sex-typicality and attractiveness: Are supermale and superfemale faces super-attractive? Br J Psychol 2000;91:125–40.
23 Mikach SM, Bailey JM. What distinguishes women with unusually high numbers of sex partners? Evol Hum Behav 1999;20:141–50.
24 Camperio-Ciani A, Cermelli P, Zanzotto G. Sexually antagonistic selection in human male homosexuality. PLoS ONE 2008;3:e2282.
25 King M, Green J, Osborn DPJ, Arkell J, Hetherton J, Pereira E. Family size in white gay and heterosexual men. Arch Sex Behav 2005;34:117–22.
26 Bobrow D, Bailey JM. Is male homosexuality maintained via kin selection? Evol Hum Behav 2001;22:361–8.
27 Vasey PL, VanderLaan DP. An adaptive cognitive dissociation between willingness to help kin and non-kin in Samoan Fa'afafine. Psychol Sci 2010;21:292–7.
28 Zietsch BP, Morley KI, Shekar SN, Verweij KJH, Keller MC, Macgregor S, Wright MJ, Bailey JM, Martin NG. Genetic factors predisposing to homosexuality may increase mating fitness in heterosexuals. Evol Hum Behav 2008;29:424–33.
29 Spector T, Williams F. The UK adult twin registry (TwinsUK). Twin Res Hum Genet 2006;9:899–906.
30 Ooki S, Yamada K, Asaka A, Hayakawa K. Zygosity diagnosis of twins by questionnaire. Acta Genet Med Gemellol 1999;39:109–15.
31 Baumeister RF. Gender differences in erotic plasticity: The female sex drive as socially flexible and responsive. Psychol Bull 2000;126:347–74.
32 Perusse D. Cultural and reproductive success in industrial societies: Testing the relationship at proximate and ultimate levels. Behav Brain Sci 1993;16:267–322.
33 Clutton-Brock TH. Reproductive success: Studies of individual variation in contrasting breeding systems. Chicago: University of Chicago Press; 1990.
34 Luciano M, Wright MJ, Geffen GM, Geffen LB, Smith GA, Martin NG. Multivariate genetic analysis of cognitive abilities in an adolescent twin sample. Aust J Psychol 2004;56:79–88.
35 Neale MC, Boker SM, Xie G, Maes HH. Mx: Statistical modeling. 7th edition. Richmond: Virginia Commonwealth University; 2006.
36 Neale MC, Cardon LR. Methodology for genetic studies of twins and families. Dordrecht, The Netherlands: Kluwer Academic; 1992.
37 Keller MC, Miller G. Resolving the paradox of common, harmful heritable mental disorders: Which evolutionary genetic models work best? Behav Brain Sci 2006;29:385–404.
38 Nettle D. Individual differences. In: Dunbar RIM, Barrett L, eds. Oxford handbook of evolutionary psychology. Oxford: Oxford University Press; 2007:479–90.
39 Nettle D. The evolution of personality variation in humans and other animals. Am Psychol 2006;61:622–31.
40 Andrews T, Hart DJ, Snieder H, de Lange M, Spector TD, Macgregor AJ. Are twins and singletons comparable? A study of disease related and lifestyle characteristics. Twin Res Hum Genet 2001;4:464–77.