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].