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Genetic and Environmental Effects on Same-sex Sexual Behavior: A Population Study of Twins in Sweden

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Abstract There is still uncertainty about the relative importance of genes and environments on human sexual orientation. One reason is that previous studies employed self-selected, opportunistic, or small population-based samples. We used data from a truly population-based 2005–2006 survey of all adult twins (20–47 years) in Sweden to conduct the largest twin study of same-sex sexual behavior attempted so far. We performed biometric modeling with data on any and total number of lifetime same-sex sexual partners, respectively. The analyses were conducted separately by sex. Twin resemblance was moderate for the 3,826 studied monozygotic and dizygotic same-sex twin pairs. Biometric modeling revealed that, in men, genetic effects explained .34–.39 of the variance, the shared environment .00, and the individual-specific environment .61–.66 of the variance. Corresponding estimates among women were .18–.19 for genetic factors, .16–.17 for shared environmental, and 64–.66 for unique environmental factors. Although wide confidence intervals suggest cautious interpretation, the results are consistent with moderate, primarily genetic, familial effects, and moderate to large effects of the nonshared environment (social and biological) on same-sex sexual behavior.

Keywords Sexual behavior · Sexual orientation · Population survey · Twin study

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Introduction

The prevalence of same-sex (or homosexual) sexual behavior varies over time and geographical region; population-based surveys suggest lifetime estimates of 3–20% for men and 2–9% in women (Caceres, Konda, Pecheny, Chatterjee, & Lyerla, 2006; Sell, Wells, & Wypij, 1995). The origin of same-sex sexual behavior is of obvious interest to theories regarding the development of sexual preferences (Rahman, 2005). However, the issue is also important for professionals concerned with sexual health in general and the psychological health of non-heterosexual populations. Same-sex behavior remains a substantial correlate of sexually transmitted infections in men and is also associated with increased risk of physical and psychiatric morbidity among both men and women in the general population (Cochran & Mays, 2000; Sandfort, Bakker, Schellevis, & Vanwesenbeeck, 2006; Sandfort, de Graaf, Bijl, & Schnabel, 2001).

Twin studies can be used to study the relative importance of genetic and environmental origins of same-sex behavior. Early studies reported significant familial aggregation—due to genetic and environmental factors—of behavioral (i.e., same-sex sexual experiences) and psychological (i.e., sexual attraction) components of sexual orientation (Bailey & Pillard, 1991; Bailey, Pillard, Neale, & Agyei, 1993). The inheritance pattern is still unclear. Family pedigree studies of male homosexual behavior have suggested that maternally inherited factors might be involved (Camperio-Ciani,

Corna, & Capiluppi, 2004; Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993; Hu et al., 1995; Rahman et al., 2007), but Bailey et al. (1999) failed to replicate maternal inheritance with data from a large pedigree study. Moreover, two linkage studies reported DNA marker loci for male homosexuality on the X chromosome (Hamer et al., 1993; Hu et al., 1995) whereas another study found no such linkage (Rice, Anderson, Risch, & Ebers, 1999; Sanders et al., 1998). Finally, one genome-wide scan reported several new autosomal markers for male sexual orientation (Mustanski et al., 2005).

Nonetheless, the conclusions of previous studies (twin, pedigree, and molecular genetic work) are limited by their reliance on self-selected volunteers (e.g., through advertisements in gay and lesbian publications) and associated ascertainment biases. To our knowledge, only two studies of same-sex behavior were population-based and both reported lower concordance rates than previously found in self-selected samples (Bailey, Dunne, & Martin, 2000; Kendler, Thornton, Gilman, & Kessler, 2000). One of these reports also suggested that the etiological pathways towards same-sex behavior are different for men and women (Bailey et al., 2000). However, these two studies require confirmation in larger population-level samples given low statistical power due to the skewed distribution of same-sex sexuality. Therefore, we undertook the largest ever population-based twin study to estimate the influence of genetic and environmental effects on same-sex sexual behavior.

Method

Participants

Data were from the Swedish Twin Registry, the largest population register of twin births in the world (Lichtenstein et al., 2006). All twin pairs born in Sweden 1959–1985 where both siblings were alive and living in the country were invited to participate in the Swedish Twin Study of Adults: Genes and Environments (STAGE); 21,481 men and 21,607 women were eligible for the web-based survey conducted in 2005–2006. Non-responders were approached with up to three reminders. They could also choose a telephone interview supplemented with a self-administered written questionnaire for sensitive topics such as traumatic events and sexuality. The STAGE screens for major psychiatric and physical morbidity, health-related experiences, and behaviors, including trauma, substance misuse, and sexuality. The overall response rate was 59.6%; 11,229 men (53.2%) and 14,096 women (65.9%) participated ($M = 33.7$ years, $SD = 7.7$, range = 20–47). By definition (being born in Sweden), no respondents were first generation immigrants. Sixty-four percent were married/cohabiting, 5% had a stable partner they were not living with, 27% were single, and 4% were separated/divorced, widowed or did not answer. With respect to the highest education completed (or currently attending), 5% responded elementary school, 41% high school, 12% vocational education, military college or other, and 42% college/university.

Measures

The STAGE survey included no direct question about self-defined sexual orientation. Actual partnered sexual behavior was assessed with two items: lifetime number of opposite-sex and same-sex individuals, respectively, that the respondent had ever "been sexually together with." We deliberately attempted to use a more gender- and sexual orientation-neutral definition rather than "sexual intercourse." Responses for lifetime number of same-sex individuals were obtained from 7,231 men (64.4% of survey respondents) and 10,676 women (75.7%). From this, we constructed the two variables *any lifetime same-sex partner* and *total number of lifetime same-sex partners* (divided into seven categories to minimize the impact of outliers; 0, 1, 2, 3–5, 6–10, 11–20, and 21 or more partners) based on prior work (Kirk, Bailey, Dunne, & Martin, 2000).

Zygosity was established using standard physical similarity questions previously validated through genotyping (98% correct classification; Lichtenstein et al., 2006). For 7,335 individuals, their twin siblings did not participate and 121 twin pairs were not possible to classify regarding zygosity. After we had excluded 1,339 opposite-sex dizygotic (DZ) twin pairs from further analysis, the final sample comprised 7,652 individuals with known zygosity where both twins had responded to sexuality items; 2,320 monozygotic (MZ) pairs (807 male and 1,513 female) and 1,506 DZ same-sex pairs (517 male and 989 female). The Regional Ethics Committee in Stockholm, Sweden provided ethical approval.

Procedure

Twin resemblance was assessed using probandwise concordance (i.e., the proportion of twins reporting same-sex sexual behavior given the same behavior in the co-twin) and tetra- or polychoric correlations for the liability to express same-sex sexual behavior. Liability is assumed to be a continuous and normally distributed latent tendency for a certain phenotype resulting from numerous etiological components. Individuals who exceed a theoretical threshold on this latent distribution would exhibit same-sex sexual behavior.

To study the sources of individual differences, we used univariate twin modeling based on contingency tables for same-sex sexual behavior between twins in MZ and DZ pairs. We tested a model where observed phenotypic variance was assumed to result from the sum of additive genetic

effects, shared or familial environmental effects, and unique environmental effects. The model was fitted with maximum likelihood estimation using the Mx structural modeling program (Neale, 1999). Since same-sex behavior may have different etiology in men and women (Bailey et al., 2000), we included only same-sex twin pairs in the model-fitting analyses.

Results

Fewer men (407/7,231; 5.6%) than women (835/10,676; 7.8%) reported any lifetime same-sex sexual partner, $\chi^2(1) = 32.11$, $p < .001$. The average number of same-sex sexual partners among those reporting any such partner was 12.86 in men and 3.53 in women, Wilcoxon rank sum test = 8.75, $p < .001$. A weak negative correlation between any lifetime same-sex partner and age was found in women, rho = −.05, $p < .001$, but not in men, rho = .01, $p = .24$.

Table 1 shows twin resemblance and estimates of genetic and environmental influences on same-sex behavior for both men and women. Twin resemblance for the two measures was moderate overall, but higher upon direct comparison in monozygotic than in dizygotic twins for both sexes (Table 1). In men, the full twin model suggested heritability estimates of 39% for any lifetime same-sex partner (95% CI: 00–59%) and 34% for total number of same-sex partners (95% CI: 00–53%) whereas unique environmental factors accounted for 61% (95% CI: 41–85%) and 66% (95% CI: 47–87%), respectively (Table 1). No shared environmental effects were found among men. For women, 18–19% of same-sex sexual behaviors were explained by genetic factors and 64–66% by unique environmental factors. Shared environmental effects were weak at 16–17%.

Discussion

In the largest twin sample studied so far, we found familial clustering of same-sex sexual behavior in both men and women. Our results support the notion that same-sex behavior arises not only from heritable but also from individual-specific environmental sources. Further, although not statistically significant, hereditary effects appeared weaker in women and of the same magnitude as those of the shared environment.

This is the first study assessing same-sex behavior in a truly population-based Scandinavian register of twins, thus avoiding the problems of volunteer self-selection, known to introduce bias towards monozygotic twin pairs concordant for specific traits (Bailey et al., 2000; Kendler & Eaves, 1989). This study also had greater statistical power and high response rates relative to prior research. However, it was inevitably limited by the fact that same-sex behavior was relatively rare. With the unexpectedly low familial effects, the twin method has restricted power to detect statistically significant influences. For example, with 4% prevalence and a sample size of 2,000 twin pairs, there was 25% power to detect a heritability of 20% at the 5% level (assuming that shared environmental effects explain 15% and unique environmental effects 65%). To achieve 80% power to detect significant genetic effects with these assumptions,

Table 1 Behaviorally measured same-sex sexual orientation in 20- to 47-year-old Swedish twins separated by sex

Twin group		Twin resemblance					Model fitting parameter estimate (95% confidence interval)		
		Number of pairs	Number of concordant pairs	Number of discordant pairs	Probandwise concordance	Correlation in liability[a]	Genetic	Shared environment	Unique environment
Any lifetime same-sex partner[b]									
Males	Monozygotic	807	7	64	.18	.39	.39 (.00–.59)	.00 (.00–.46)	.61 (.41–.85)
	Dizygotic	517	3	50	.11	.19			
Females	Monozygotic	1,513	26	188	.22	.36	.19 (.00–.49)	.17 (.00–.42)	.64 (.51–.78)
	Dizygotic	989	13	127	.17	.27			
Total number of same-sex partners[c]									
Males	Monozygotic	807	2	69	.05	.40	.34 (.00–.53)	.00 (.00–.39)	.66 (.47–.87)
	Dizygotic	517	0	53	.00	.17			
Females	Monozygotic	1,513	12	202	.11	.38	.18 (.11–.45)	.16 (.00–.39)	.66 (.55–.78)
	Dizygotic	989	5	135	.07	.25			

[a] Tetrachoric correlation for Any lifetime same-sex partner, polychoric correlation for Total number of same-sex partners

[b] Prevalences were 4.8% for monozygotic male twins, 5.4% for dizygotic male twins, 7.9% for monozygotic female twins, and 7.7% for dizygotic female twins, $\chi^2(3) = 21.48$, $p < .001$

[c] Divided into seven ordinal categories; 0, 1, 2, 3–5, 6–10, 11–20, and 21+ partners

Table 2 Demographic and methodological characteristics and major findings of three population-based twin studies of sexual orientation

Variable	Study		
	Bailey et al. (2000)	Kendler et al. (2000)	Långström et al. (present study)
Country of origin	Australia	USA	Sweden
Type of sample	Volunteer register of twins	National household sample Relatives or twins provided co-twin contact information	National population register
Definition used for sexual orientation	Mean of sexual attraction and fantasies, both rated on 7-point Kinsey scales, and divided into three ordinal categories	Self-reported sexual orientation with response options heterosexual, homosexual or bisexual attraction	Any lifetime same-sex sexual partner Lifetime number of same-sex partners
Year(s) of data collection	1992	1995–1996	2005–2006
Overall response rate (%)	53.8	60.0	59.6
Age range of overall sample (years)	17–50	25–74	20–47
Overall sample size (individuals in same-sex pairs with complete data)[a]	3,076	1,128	7,652
No. of male twins in same-sex pairs with complete data	988	Not specified	2,648
No. of female twins in same-sex pairs with complete data	2,088	Not specified	5,004
Summary of parameter estimates			
Genetic	.45 for men (95% CI: .00–.71) .08 for women (95% CI: .00–.67)	.62 with no information on 95% CIs or sex differences	.34–.39 for men (95% CIs: .00–.59) .18–.19 for women (95% CIs: .00–.49) considering both measures
Shared environment	.00 for men (95% CI: .00–.41) .41 for women (95% CI: .00–.64)	.05 with no information on 95% CIs or sex differences	.00 for men (95% CIs: .00–.46) .16–.17 for women (95% CIs: .00–.42) considering both measures
Unique environment	.55 for men (95% CI: .18–.85) .50 for women (95% CI: .30–.69)	.33 with no information on 95% CIs or sex differences	.61–.66 for men (95% CIs: .41–.87) .64–.66 for women (95% CIs: .51–.78) considering both measures

[a] Reflects the total number of individuals included in model fitting analyses, although sample sizes varied somewhat within each study depending on the analysis performed

one would need to increase the sample size 5-fold (i.e., include approximately 10,000 monozygotic and dizygotic same-sex twin pairs with complete data). Consequently, though familial effects certainly are important for same-sex behavior, the exact magnitude of genetic and environmental contributions to these effects should be interpreted cautiously. Moreover, the sensitivity of the topic should be borne in mind although this study was conducted in a sexually liberal Scandinavian country. Even if very large twin samples are approached, the number of pairs where both twins choose to reveal same-sex behavior will remain limited. Furthermore, while this study focused on same-sex sexual behavior, assessment of sexual attraction or fantasies and even romantic attractions would more fully capture the complexity of sexual orientation. However, both behavioral measures of sexual orientation (any same-sex sexual partner and total number of same-sex partners) correlated strongly with self-reported same-sex sexual attraction ($r = .70–.75$) upon cross-validation in a contemporary referred sample ($n = 555$) of age-matched adult men in Toronto, Canada (data available upon request).

There are only two other published population based twin studies of same-sex behavior, from Australia (Bailey et al., 2000; Kirk et al., 2000) and the U.S. (Kendler et al., 2000). Table 2 provides a comparison of demographic and methodological characteristics and major findings of these two studies and the present one. Our results of genetic and unique environmental effects largely agreed with the estimates found by Bailey et al. (2000), but less so with those reported by Kendler et al. (2000). Unfortunately, the latter study did not report sex-separated analyses. The concordance rates found by us were lower than in studies using opportunistic or otherwise non-representative samples of non-heterosexual individuals (Bailey & Pillard, 1991; Bailey et al., 1993). Although our results are not inconsistent with relatively large heritabilities, this suggests cautious interpretation of data from non-representative sampling of individuals from sexual minorities.

Our data indicated that genetic influences on any lifetime same-sex partner and total number of same-sex partners were weaker in women than in men. This disagrees with the greater genetic influences on number of same-sex partners in women found by Kirk et al. (2000), using different complex genetic models. In contrast, our finding does concur with the results obtained when the same group conducted less complex univariate modeling using essentially the same data (Bailey et al., 2000, summarized in Table 2). We did not perform sex-limitation tests in our model fitting because these would not be statistically powerful even in our large sample. Also, the large confidence intervals warrant careful interpretation. Nevertheless, our findings for women (suggestive of weaker but equal genetic and shared environmental effects and higher levels of same-sex behavior compared to men) are congruent with observations that female sexual attraction and behavior are more flexible in response to shared environmental factors contrasted to that of men (Baumeister, 2000; Lippa, 2006; Rahman, 2005).

It has been suggested that individual differences in heterosexual and homosexual behavior result from unique environmental factors such as prenatal exposure to sex hormones, progressive maternal immunization to sex-specific proteins, or neurodevelopmental instability (Rahman, 2005). Although the unique environmental variance component also includes measurement error, the present results support the notion that the individual-specific environment does indeed influence sexual preference.

In conclusion, although confidence intervals were wide, we believe this study provides the most unbiased estimates presented so far of genetic and non-genetic contributions to same-sex sexual behavior. The results should inform further research on this complex trait.

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