

Brief report

Potential for homosexual response is prevalent and genetic

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Abstract

We investigated the potential to engage in homosexual behavior in 6001 female and 3152 male twins and their siblings finding that 32.8% of the men and 65.4% of the women reported such potential ($p < 0.001$). 91.5% of these men and 98.3% of these women reported no overt homosexual behavior during the preceding 12 months. The potential to engage in homosexual behavior was influenced by genetic effects for both men (37.4%) and women (46.4%) and these overlapped only partly with those for overt homosexual behavior.

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1. Introduction

Reported rates of homosexuality are generally low. In a recent U.S. study, for example, 2.6% of men and 4.7% of women reported homosexual behavior during the last 12 months (Villaruel et al., 2006). Compared to heterosexuality, homosexuality has been considered atypical, implying that the genetic and psychological factors leading to it should only affect a small minority of individuals. Individual differences in overt homosexual behavior have been found to be affected by genes in twin studies (Hyde, 2005). Kirk et al. (2000b) analyzed twins from an Australian twin registry and found genetic influences accounted for approximately 42–60% of a range of psychological and behavioral measures of sexual orientation for male twins. Here again, the results suggested that nonadditive genetic effects were likely to affect feelings and fantasies directed towards same-sex partners. In yet another study, Kirk et al. (2000a) showed using multiple measures of sexual orientation that genetic influences accounted for 50–60% of the variation for females and approximately 30% of the variation for males.

Molecular genetic studies constitute another strand of evidence for genetic effects on homosexual orientation. Studies showing a preponderance of gay male relatives on the mother's

side of the families of gay men have led to the hypothesis that male homosexuality may involve an X-chromosome linked inheritance. The Xq28 region of the X-chromosome in male homosexuality has received particular attention with a meta-analysis of four studies showing a significant effect (Hamer, 2002). However, the evidence provided by molecular genetic studies for the role of genes in the development of sexual orientation is not particularly strong.

Interestingly, informal observations suggest that potential for homosexual response (PHR) may be more prevalent than usually thought in empirical research into its antecedents. Situational homosexual behavior by heterosexual men has been reported in exclusively male circumstances such as prisons and the military, and for financial profit in prostitution and the gay porn industry (Escoffier, 2003). Also, a population-based survey found that almost 10% of men who identified themselves as heterosexual reported having had sex with another man during the previous year (Pathela et al., 2006). In fact, of the men engaging in homosexual behavior, more identified themselves as heterosexual than homosexual. These findings suggest that at least many men have the potential for homosexual response. Less direct research evidence concerning situational homosexuality in women is available. However, women have been found to report more retrospective change in their sexual orientation compared to men (Kinnish et al., 2005).

The aims of the present study were to investigate (1) the prevalence of PHR in both men and women, (2) the overlap between PHR and overt homosexual behavior, (3) whether PHR

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and overt homosexual behavior are influenced by genetic effects, and (4) whether the genetic effects on PHR are the same as the genetic effects on overt homosexual behavior.

2. Methods

2.1. Participants

The analyses presented in this study were based on a population-based sample of 9153 (female $n = 6001$; $M = 26.1$ years, $S.D. = 5.1$; male $n = 3152$; $M = 26.2$ years, $S.D. = 4.8$) Finnish twins and their siblings. All participants were included in all phenotypic analyses, even if they did not form part of a full sibling pair, whereas only participants belonging to same-sex sibling pairs were included in model fitting analyses.

Participants were a subset from the second data collection of the Genetics of Sex and Aggression (GSA) sample. This data collection was carried out in 2006 and targeted 18–33-year-old twins and their over 18-year-old siblings. The participants' addresses were obtained from the Finnish population registry. The overall response rate was of 45%. The response rate represents an underestimate due to address changes resulting in some potential participants not receiving the request to participate. According to Statistics Finland (<http://www.statistics.fi>) approximately 15% of Finns move each year. Considering that the data collection lasted over half a year, the real response rate was approximately 50%.

Zygosity was determined using questionnaire items completed by the twins (Sarna et al., 1978). Previous studies have shown that this kind of method of zygosity determination is 95% accurate when compared with blood typing analyses (Eisen et al., 1989). The research plan was approved by the Ethics Committee of the Åbo Akademi University.

2.2. Instruments

2.2.1. Self assessment of sexual orientation (SASO, Sell, 1996)

One item from the SASO instrument was used to assess the existence of same-sex sexual behavior among the respondents: during the past year, on average, how often did you have sexual contact with a man (woman for female participants)? The response alternatives were: never, less than one time per month, one to three times per month, one time per week, two to three times per week, four to six times per week, Daily. The respondents were given numerical scores so that a response of "None"/"Never" gave a score of 0 and a response of "100 or more"/"Daily" gave a score of 7. The resulting variable was additionally subjected to a logarithmic transformation in order to render its distribution more appropriate for the correlative and model fitting analyses (with a change in skewness from 2.77 to 0.95). It was also regressed for age prior to the model fitting analyses. To estimate the frequency of any homosexual contact during the preceding year, the variable was dichotomised with 0 coded as 0 and all other values as 1.

2.2.2. Potential for homosexual response

The participants were asked to respond to the following question: "If a, in your opinion, handsome man [to male participants]/beautiful woman [to female participants], whom you like, suggested sexual interaction with you, how likely would you be able to do it (if you could define the nature of the interaction and nobody else would know about it)?" The participants indicated their response on a Likert-type scale with the following response options: quite impossible, very unlikely, quite unlikely, not likely, not unlikely, relatively likely, very likely. For correlative and model fitting analyses the responses were coded so that quite impossible given the value of 0 and very likely to value of 5. The resulting variable was additionally subjected to a logarithmic transformation in order to render its distribution more appropriate for the correlative and model fitting analyses (with a change in skewness from 1.08 to 0.49). It was also regressed for age prior to the model fitting analyses. To estimate the prevalence of any potential, the variable was dichotomised with 0 coded as 0 and all other values as 1.

2.3. Data analysis

2.3.1. Phenotypic analyses

Phenotypic analyses were conducted with the SPSS for Windows (Version 14) Complex Samples module. This module takes into account the dependence between the members of the same families.

2.3.2. Genetic analyses

Polychoric twin correlations were computed with the program POLYCORR Classic (Uebersax, 2006). In some instances, it was necessary to regroup the categories prior to running polychoric correlations. Next, model fitting analyses were undertaken. The first aim was to estimate the extent to which the total variance was due to genetic and environmental influences. Additive genetic (A), shared environmental (C) and nonshared environmental (E) influences on a phenotype of interest are estimated in this model. Detailed descriptions of twin modeling analyses can be found in Posthuma et al. (2003).

The second aim was to examine the extent to which genetic and environmental influences on the two phenotypes overlapped. In this analysis, we used a bivariate genetic model in which both within-trait and cross-trait genetic and environmental influences were modeled. With this type of model, it is possible to examine the genetic and environmental correlations. The genetic correlation refers to the correlation between genetic influences on one measure, x and genetic influences on a second measure y . A genetic correlation of unity between x and y would indicate that genetic influences contributing to variance in x and y were identical. Correlations between nonshared environmental influences were also calculated.

Models were estimated by full-information maximum likelihood (FIML) estimation using the program Mx (Neale et al., 2002). We used scripts for continuous variables as the variables were close to normal distribution following the logarithmic transformation and age regression. Attempts at fitting univariate and bivariate extended-family scripts for categorical data were not successful, probably due to problems with thresholds.

3. Results

The 12-month incidence of overt homosexual behavior was 3.1% for men and 1.2% for women ($p < 0.001$). The percentages of men and women who reported any potential

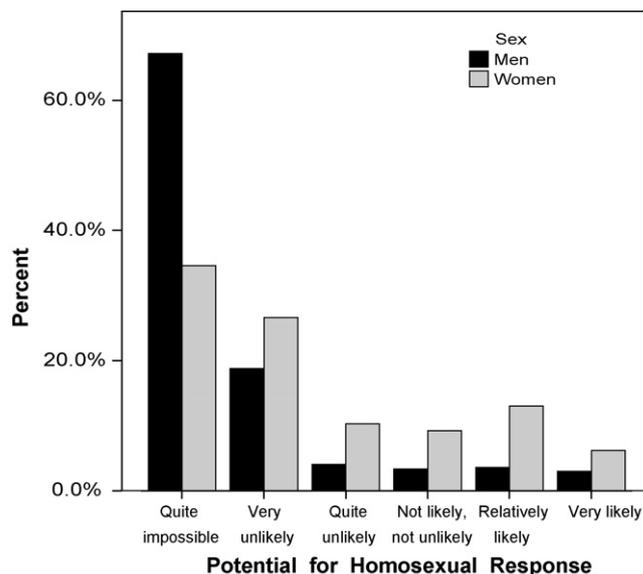


Fig. 1. Potential for homosexual behavior. The distribution of responses of the participants to the question "If a, in your opinion, handsome man/beautiful woman, whom you like, suggested sexual interaction with you, how likely would you be able to do it (if you could define the nature of the interaction and nobody else would know about it)?".

Table 1
Polychoric twin correlations (standard error) for potential for homosexual response and overt homosexual behavior (last 12 months)

	Potential for homosexual response	Overt homosexual behavior (last 12 months)
Cross twin within-trait		
MZM	0.534 (0.080)	0.380 (0.195)
DZM	0.234 (0.107)	0.000 (0.010)
MZF	0.526 (0.035)	0.594 (0.172)
DZF	0.264 (0.054)	0.000 (0.008)
Cross twin cross-trait		
MZM		0.355 (0.121)
DZM		0.157 (0.160)
MZF		0.472 (0.100)
DZF		0.222 (0.127)

Note: MZM, monozygotic male pairs; DZM, dizygotic male pairs; MZF, monozygotic female pairs; DZF, dizygotic female pairs.

for homosexual response were 32.8 and 65.4%, respectively ($p < 0.001$) (for the full distribution of responses see Fig. 1). Age was somewhat related to the PHR for both men, $r_p = -0.04$, $p < 0.029$, and women, $r_p = -0.12$, $p < 0.001$, with older respondents being less likely to report PHR. The association was stronger for women ($p < 0.001$). PHR correlated moderately with overt homosexual behavior during the last 12 months (men $r_p = 0.285$, women $r_p = 0.217$, both $ps < 0.001$). Even so, the majority (91.5% of men and 98.3% of women) of those reporting PHR had not engaged in any overt homosexual behavior during the last 12 months.

The polychoric twin correlations are shown in Table 1. These correlations suggest the presence of genetic effects on both PHR and overt homosexual behavior. Also, any phenotypic correlation between the two traits would seem to be partly explainable by common genetic effects. Results from model fitting analyses confirmed the pattern observed in the polychoric correlations and showed that variations in PHR were genetic for both men (37.4%) and women (46.4%) with no shared environmental effects (Table 2). The

variations in overt homosexual behavior during the last 12 months were also influenced by genetic effects for both men (27.0%) and women (16.2%) again with no shared environmental effects. Removing the genetic components resulted in statistically significant decreases in model fit in each instance.

We also estimated the extent to which the genetic and environmental effects were the same for PHR and homosexual behavior from bivariate AE models separately for males and females. For males, the genetic ($r_g = 0.468$ [0.238, 0.673]) and the nonshared environmental ($r_e = 0.273$ [0.167, 0.372]) effects underlying the two phenotypes were moderately correlated. The removal of both the genetic ($\Delta\chi^2 = 11.842$, $p < 0.001$) and the nonshared environmental correlation ($\Delta\chi^2 = 24.429$, $p < 0.001$) resulted in a significant decrease in model fit. Likewise for females, the genetic ($r_g = 0.531$ [0.356, 0.746]) and the nonshared environmental ($r_e = 0.123$ [0.054, 0.190]) effects were moderately correlated and the removal of both the genetic ($\Delta\chi^2 = 31.654$, $p < 0.001$) and the nonshared environmental correlation ($\Delta\chi^2 = 12.346$, $p < 0.001$) decreased the fit of the models.

Table 2
Estimates (95% confidence intervals) from ACE models for potential for homosexual behavior and overt homosexual behavior (last 12 months) separately for males and females from full-information maximum likelihood (FIML) estimation using the program Mx

	A	C	E	-2*LL	AIC	$\Delta\chi^2$	ACE vs. CE	ACE vs. AE	ACE vs. E
Males									
Potential for homosexual response	0.374 (0.123, 0.467)	0.000 (0.000, 0.191)	0.626 (0.533, 0.733)	-217.515	-3263.515	7.810**	0.000		45.224***
Overt homosexual behavior (last 12 months)	0.270 (0.027, 0.380)	0.000 (0.000, 0.177)	0.730 (0.620, 0.850)	-4107.803	-7153.803	4.554*	0.000		19.336***
Females									
Potential for homosexual response	0.464 (0.323, 0.516)	0.000 (0.000, 0.108)	0.536 (0.484, 0.595)	606.909	-5743.091	35.125***	0.000		214.367***
Overt homosexual behavior (last 12 months)	0.162 (0.083, 0.236)	0.000 (0.000, 0.036)	0.839 (0.764, 0.913)	-6683.461	-13033.461	10.856***	0.000		18.515***

Note: A, additive genetic influences; C, shared environmental influences; E, nonshared environmental influences; AIC, Akaike information criterion.

* $p < 0.05$.

** $p < 0.01$.

*** $p < 0.001$.

4. Discussion

The results show, for the first time, prevalent potential for homosexual response in both men and women, even among individuals who do not report any overt homosexual behavior. Women reported more potential which is in accordance with previous findings showing that their sexual orientation may be more changeable (Kinnish et al., 2005). These results suggest that sexual identity, behavior and potential may often be quite disentangled as suggested by both Escoffier (2003) and Pathela et al. (2006). The phrasing of the question does not suggest that the homosexual sex would take place in the absence of possibilities for heterosexual sex, indicating that the responses refer to homosexual behavior by choice. Therefore, the concept of PHR should not be mixed with situational homosexuality occurring in all-male or all-female environments such as prisons.

PHR was also influenced by genes. These genetic effects overlapped only partly with overt homosexual behavior. Any alleles underlying the genetic effect on PHR should be relatively common and, therefore, also likely to have served some evolutionary purpose, such as limiting the aggressiveness of males and thereby making them more attractive to females (Miller, 2000). PHR was more prevalent among women, a result that is not in line with suggestions that the alleles influencing homosexuality would be predominantly linked to the X-chromosome (Hamer, 2002). It is also of interest to note that there was no evidence of any shared environmental effects on either phenotype. Such results suggest no role for neither intrauterine effects, arguing against hormonal theories of sexual orientation, nor for familial effects shared by all siblings to the same degree, arguing against simple parental personality or parenting style effects.

5. Conclusion

The results imply that we should rethink how the phenotype of homosexuality is defined. Finally, previous psychological and genetic correlates of homosexual orientation may actually have more to do with why some people engage in homosexual behavior as opposed to being correlates of a potential to do so.

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