Arch Sex Behav (2010) 39:81–92
DOI 10.1007/s10508-008-9457-3

ORIGINAL PAPER

Common Genetic Effects of Gender Atypical Behavior in Childhood and Sexual Orientation in Adulthood: A Study of Finnish Twins

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Received: 12 July 2007 / Revised: 16 June 2008 / Accepted: 20 September 2008 / Published online: 27 January 2009

Abstract The existence of genetic effects on gender atypical behavior in childhood and sexual orientation in adulthood and the overlap between these effects were studied in a population-based sample of 3,261 Finnish twins aged 33–43 years. The participants completed items on recalled childhood behavior and on same-sex sexual interest and behavior, which were combined into a childhood gender atypical behavior and a sexual orientation variable, respectively. The phenotypic association between the two variables was stronger for men than for women. Quantitative genetic analyses showed that variation in both childhood gender atypical behavior and adult sexual orientation was partly due to genetics, with the rest being explained by nonshared environmental effects. Bivariate analyses suggested that substantial common genetic and modest common nonshared environmental correlations underlie the co-occurrence of the two variables. The results were discussed in light of previous research and possible implications for theories of gender role development and sexual orientation.

Keywords Gender identity · Gender role behavior · Sexual orientation · Sex differences · Behavior genetics

Introduction

A number of studies have found that gender atypical behavior (GAB) in childhood, i.e., a consistent pattern of activities and preferences that are statistically more prevalent for the other biological sex, is related to sexual orientation in adulthood (e.g., Dunne, Bailey, Kirk, & Martin, 2000; Green, 1987). Bailey and Zucker (1995) conducted a meta-analysis of 41 studies that analyzed the link between childhood GAB and adult sexual orientation. Gay men and lesbians recalled significantly more childhood GAB than heterosexual men and women. Similar findings have been reported across different cultural groups (Lippa & Tan, 2001; Whitam & Mathy, 1991). Bailey and Zucker (1995) further found that GAB was more predictive of an adult homosexual orientation for boys than for girls.

Several behavior genetic studies have been conducted in order to look into the balance of genetic and environmental causes for GAB. These studies have found evidence for both genetic and shared environmental influences (i.e., nongenetic influences contributing to resemblance in family members). Knafo, Iervolino, and Plomin (2005) found in an extremes analysis of GAB, in a UK sample of 3- to 4-year-old twins, moderate genetic effects in boys (37%) in contrast to larger genetic effects in girls (82%). Shared environmental influences accounted for 42% and 0% of the variance for boys and girls, respectively. van Beijsterveld, Hudziak, and Boomsma (2006) studied genetic contributions to GAB in a Dutch sample of 7- and 10-year-old twins. They found that genetic influences accounted for 70% of the variance in GAB for both boys and girls. Bailey, Dunne, and Martin (2000) studied sexual orientation and its correlates with childhood gender nonconformity (i.e., GAB) and gender identity in an Australian twin registry based sample. Significant genetic effects could only be found for childhood gender nonconformity, with an estimate of 50% for men and 37% for women. Familial effects for all three variables were found, although it was not possible to distinguish between genetic and common environmental factors for the other two variables. Gender

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nonconformity was associated with adult sexual orientation, strongly for men ($r = .57$) and moderately for women ($r = .33$). Multivariate analysis suggested the existence of a common familial factor that contributed to the covariance among the three observed variables.

Evidence for genetic effects on homosexuality has been found in several studies. Besides studies that have shown that homosexuality runs in families (Bailey & Bell, 1993; Dawood, Pillard, Horvath, Revelle, & Bailey, 2000; Pillard & Weinrich, 1986), there is evidence for higher concordance rates for homosexuality among monozygotic (MZ) compared to dizygotic (DZ) twins, both among men and women (Bailey & Pillard, 1991; Bailey, Pillard, Neale, & Agyei, 1993; King & McDonald, 1992; Whitam, Diamond, & Martin, 1993). Similar findings have been reported by behavior genetic studies: Kirk, Bailey, Dunne, and Martin (2000) found that genetic influences accounted for 50–60% of the variation in sexual orientation for women and approximately 30% of the variation for men. Bailey et al. (2000) found genetic estimates of 45% for male and 8% for female homosexuality. However, Hershberger (1997) found heritability estimates of 49% for same-sex sexual attraction and 45% for number of same-sex sexual encounters for women, with no significant genetic effects for men. Furthermore, preliminary findings from molecular genetic studies suggest some evidence for the role of specific loci for male sexual orientation. In a meta-analysis, Hamer (2002) combined results from existing studies and concluded that the Xq28 region may play some role, for some men, in the development of sexual orientation.

In an attempt to consolidate biological findings with those concerning psychosexual development, Bem (1996, 2000) has proposed a theory linking childhood GAB and sexual orientation. In the exotic becomes erotic (EBE) theory, Bem argued that the developmental pathway starts from biological variables, such as genes or prenatal hormonal exposure that affect a child's temperament in a sex atypical direction. The child becomes alienated from same-sex peers and a physiological arousal occurs in the presence of them. Further on, the physiological arousal is mixed with sexual arousal and same-sex peers are interpreted as erotic. Bem (2000) found evidence for the EBE theory by conducting path analyses in which the correlation between genetic similarity of twins and sexual orientation disappeared when GAB was entered as a mediating variable in the analysis, suggesting that the genetic effects affecting sexual orientation are the same as those affecting GAB. However, the theory has been criticized for providing a poor explanation for female homosexuality (Peplau, Garnets, Spalding, Conley, & Veniegas, 1998).

The aims of the present study were to explore the phenotypic association between childhood GAB and adult sexual orientation. The association was assumed to be stronger for male compared to female participants based on results from earlier studies (Bailey & Zucker, 1995). We also wanted to identify the extent of genetic effects on childhood GAB and sexual orientation in a population-based sample of both male and female twins. The hypothesis based on previous literature was that genetic effects would be found for both men and women. Additionally, we wanted to explore whether there would be genetic or environmental correlations between GAB and sexual orientation. The hypothesis was that there would be genetic covariation between the two variables.

Method

Participants

The present sample was created from data obtained through the Central Population Registry of Finland and consisted of Finnish twins between 33 and 43 years of age, born and currently residing in Finland. The mean age for male participants was 37.4 years ($SD = 2.95$) and for female participants 37.54 years ($SD = 2.91$), with no significant sex difference in age. The addresses of 5,000 twin pairs (2,000 male same-sex pairs, 2,000 female same-sex pairs, and 1,000 opposite-sex pairs) were obtained from the Registry by sampling all twin pairs born in 1971 or earlier.

The questionnaire, along with a cover letter that explained that participation was voluntary and anonymous, was mailed with a stamped return envelope. A repeat mailing was sent to nonresponders after 8 weeks. A total of 3,604 participants returned the questionnaire, resulting in an overall response rate of 36%. The response rate was lower for male (27%) when compared to female (45%) participants. The response rate was comparable to other sex-related studies with population based samples in Finland (e.g., 40% in Ojanlatva, Helenius, Rautava, Ahvenainen, & Koskenvuo, 2003). Haavio-Mannila, Kontula, and Kuusi (2001) noted lower response rates for Finnish male compared to Finnish female participant as well as a trend with diminishing response rates to questionnaires on sex-related topics in Finland, with a reduction in response rate from 91% in 1971, 76% in 1991, and 64% in 1999. They suggested that the diminishing response rates were due to differing methods of data collection, with mailed surveys achieving the lowest response rates. The response rate in the present study can be seen to follow the same trend. In a study of young adult Finnish twins with a mean age of 24.4 years ($SD = 0.84$), Mustanski, Viken, Kaprio, Winter, and Rose (2007) found that the mean age at first sexual intercourse was 17.63 for male and 17.33 for female participants. In the present study, the corresponding figures were 18.72 for male and 17.81 for female participants. This result suggests that the present sample was comparable to another representative sample of the Finnish population with respect to an important sexuality related characteristic.

The questionnaire covered multiple aspects of sexuality, including sensitive topics such as number of sexual contacts, cross-dressing, and sexual interest in children. Therefore, it was decided that the questionnaire should not contain any questions about the identity of the participants in order to make responding more probable. To pair the twins in our sample, we used their sex, age, status as first- or second-born twin, number of siblings besides the twin brother or sister, and three specially created questions (what were the two-first letters of their mother's name, what were the two last letters of their father's name, and in which month they were born). Zygosity was determined using questionnaire items completed by the twins (Sarna, Kaprio, Sistonen, & Koskenvuo, 1978). Previous studies have shown that this method of zygosity determination is 95% accurate when compared with blood typing analyses (Eisen, Neuman, Goldberg, Rice, & True, 1989).

Our final sample consisted of 91 male MZ twin pairs, 247 female MZ twin pairs, 110 male DZ twin pairs, 270 female DZ twin pairs, and 203 opposite-sex DZ twin pairs. In addition, there were 207 single male MZ twins, 199 single female MZ twins, 329 single male DZ twins from same-sex pairs, 395 single female DZ twins from same-sex pairs, and 289 (95 male and 194 female) single twins from opposite-sex pairs. For 131 (29 male and 102 female) pairs and 35 (19 male and 16 female) single twins, zygosity could not be determined. These pairs were not included in the model-fitting analyses. The phenotypic analyses were conducted with 3,593 individuals and the genetic analyses with 3,261 individuals.

The research plan was approved by the Ethics Committee of the Department of Psychology at Åbo Akademi University.

Measures

A shortened version of the Recalled Childhood Gender Identity/Gender Role Questionnaire (RCGIGR) by Zucker et al. (2006) was used to assess the gender typical behavior of the participants before the age of 12 years. The original 23-item questionnaire had two factors, one measuring gender identity and gender role and the other measuring parental identification and closeness. In the present study, only items that had a factor loading of at least .60 on the first factor in the original study were included. This resulted in a scale with 13 items. The scoring ranged from 1 to 5, with a lower score implying more gender atypical behavior. Some of the questions contained a response option, "I did not engage in such an activity." These responses were not included in the analysis and treated as missing values.

We conducted exploratory factor analyses on the 13 items of the RCGIGR, separately for male and female participants. One person per family was randomly selected, in order to avoid dependence between observations. Normality of the distributions was assessed through visual inspection of histograms. Data were positively skewed. However, logarithmic transformations did not alter the results, and, consequently raw data were used. First, a principal components analysis was used to test whether the data were suitable for factor analysis. A *KMO* value above 0.80 and a significant Bartlett's test of sphericity, in addition to values over .40 in the anti-image correlation matrix, implied that the data were factorable.

In the principal components analysis, three factors had eigenvalues over 1. A three factor solution might have been considered; however, visual inspection of the scree plot suggested the presence of only one factor. A one factor solution was also theoretically most appropriate and was, therefore, pursued. Next, a generalized least squares (GLS) factor analysis was conducted. The GLS method was chosen as it is a robust method and the data need not be completely normally distributed. The factor loadings are presented in Table 1. The factor explained 33% and 38% of the variations for men and women, respectively.

A majority of the items reached satisfactory or good factor loadings as can be seen in Table 1. On only two instances did the loadings not reach .30. However, we decided to include all items, so that we could keep the same questionnaire items for both male and female participants. Next, composite variables were formed, so that a high value indicated a more conventional pattern of gender role behavior. If a participant indicated that a specific item did not apply, it did not add or reduce the summary score.

The second measure used was the Sell Assessment of Sexual Orientation (SASO; Sell, 1996). Four items from the SASO instrument were used to assess the existence of same-sex sexual interest and behavior among the participants:

Item 1: During the past year, on average, how often were you sexually attracted to a man (woman for female participants)? The response alternatives were: never, less than 1 time per month, 1–3 times per month, 1 time per week, 2–3 times per week, 4–6 times per week, daily. *Item 2*: During the past year, on average, how often did you have sexual contact with a man (woman for female participants)? The response alternatives were the same as for Item 1 above. *Item 3*: How many different men (women for female participants) have you had sexual contact with during the past year? *Item 4*: During the past year, on average, how many different men (women for female participants) have you felt sexually attracted to? The response alternatives to Items 3 and 4 were: none, 1, 2, 3–5, 6–10, 11–49, 50–99, 100≥. The participants 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.

In order to augment the reliability of the measure, we conducted a GLS factor analysis. A KMO value of 0.75 for males and 0.56 for females and a significant Bartlett's test of

Table 1 Factor loadings and Cronbach's α for the items of the Recalled Childhood Gender Identity/Gender Role Questionnaire

Item number	RCGIGR	Factor loadings	
		Men	Women
3	Favorite toys and games (masculine versus feminine)	.56	.63
5	Cosmetics/jewelry	.59	.42
6	Sex of admired/imitated TV character	.30	.38
7	Enjoying sports such as basketball, hockey, etc.	.42	.23
8	Male/female fantasy role	.55	.66
9	Dress up play	.59	.65
10	Felt masculinity–femininity	.71	.81
11	Compared to others, felt feminine/masculine	.23	.65
14	Masculinity/femininity of appearance	.58	.67
15	Enjoyment of feminine clothing	.62	.61
18	Reputation as sissy/tomboy	.73	.74
19	Content with one's sex	.68	.62
20	Wish to be of opposite sex	.70	.70
	Cronbach's α	.82	.88

sphericity for both male and female participants suggested adequate factorability of the items. A one factor solution was found to best fit the data and explained 79% and 55% of the variance, for male and female participants, respectively. Cronbach's α was .93 for male and .79 for female participants. Next, a composite variable for sexual orientation was created by summing the items and dividing by four. A higher score on the variable indicated a more homosexual orientation.

We explored the effects of the general level of sexual desire on the sexual orientation variable because of a possible confounding effect between desire and sexual orientation due to the formulation of the SASO items. A question that addressed the general level of sexual desire was used for this purpose, "How often did you feel sexually aroused during the preceding four weeks?" The response options were: "Several times during an hour," "several times during a day," "approximately once a day," "a couple of times during a week," and "seldom or never." Significant correlations between the composite variable for sexual orientation and this sexual desire variable were found (men: $r_p = .09, p < .001$, women $r_p = .14, p < .001$). This suggests that there was a confounding effect of the general level of sexual desire on the measurement of sexual orientation. Male participants also reported a higher level of sexual desire compared to female participants (male $M = 0.13, SE = 0.02$, female $M = 0.06$, $SE = 0.01$), $F(1, 2505) = 836.15$ $p < .01$, $R^2 = .21$. The possible effects of gender were further explored in a regression analysis with an interaction term between gender and sexual desire, $F(1, 2492) < 1$, $R^2 = .016$, but none was found. Age was not associated with the general level of sexual desire for male $r_p = .004$, $F(1, 2505) < 1$, $R^2 = .00$ or for female participants, $r_p = .002$, $F(1, 2505) < 1$, $R^2 = .00$.

In order to extract the effects of sexual desire from the measure of sexual orientation, sexual desire was regressed from the sexual orientation variable, separately for male, $F(1, 2492) = 8.91, p < .005, R^2 = .01$, and female participants, $F(1, 2492) = 20.90, p < .001, R^2 = .02$. The residuals from this analysis were, thereafter, logarithmically transformed in order to correct for skewness which diminished from 7.35 to 4.62. Visual inspection of histograms revealed a distribution resembling a normal distribution. This variable was used in all further analyses as the measure of sexual orientation.

Statistical Analyses

Phenotypic Analyses

Phenotypic analyses comparing groups as well as regression analyses were conducted with the General Linear Model of the SPSS for Windows (version 14) Complex Samples module. This module takes into account the dependence between the members of the same families. The R^2 effect size estimates from these analyses are reported. Correlations between variables were computed with Pearson correlations. Analyses concerning means, variances, and twin correlations were conducted with the Mx statistical package (Neale, Boker, Xie, & Maes, 2002).

Genetic Analyses

The standard quantitative genetic model for twin data rests on the assumption that the observed (phenotypical) variance (*Vp*) in a trait is a linear function of additive genetic influences (*A*), nonadditive genetic influences (*D*) or common environmental influences (*C*), and nonshared environmental

influences (E) (i.e., $Vp = A + D + C + E$). Additive genetic influence refers to the total effects of multiple alleles on the phenotype. Nonadditive genetic influence refers to the interactive effect among multiple alleles that occupy the same loci on different chromosomes (i.e., dominance) and multiple genes (i.e., gene–gene interaction) on the phenotype. Shared and nonshared environmental influences refer to nongenetic influences that contribute to familial resemblance among relatives and nongenetic influences that uniquely influence individuals, respectively. When estimating these components, measurement error is subsumed under the nonshared environmental source of variance.

Genetic and environmental influences can be separated in the twin design because genetic resemblance varies as a function of zygosity, whereas familial resemblance due to shared environmental influences does not. Specifically, MZ twins are genetically identical, whereas DZ twins share, on average, 50% of their segregating genes. Environmental influences that contribute to familial resemblance are assumed to affect MZ and DZ twins equally. Nonshared environmental (E) factors, by definition, make twins different from one another. Detailed descriptions of twin modeling analyses can be found in Posthuma et al. (2003). The assumptions of twin modeling analyses are detailed in full in Plomin, DeFries, McClearn, and McGuffin (2001).

Quantitative gender differences imply that the magnitude of genetic and environmental influences differ between the biological sexes. A way to test for quantitative sex differences is by allowing the magnitude of genetic and environmental parameters to vary across the sexes, compared to a model where the parameters are fixed to be equal (Mustanski et al., 2007). Qualitative gender differences indicate that different genetic or environmental factors operate in men and women. To test these, we fixed additive or dominant genetic correlations between men and women (i.e., in DZO twin pairs) to 0.5 or 0.25, respectively. A large discrepancy between same-sex and opposite-sex DZ twin correlations is one implication for sex differences. If the DZO twin correlation is less than that of same-sex DZ twins, a qualitative gender difference can be suspected (McEwen et al., 2007).

As noted above, we can examine nonadditive genetic effects (D) and common environmental effects (C) with the twin design. However, a twin model that includes additive genetic influences, nonadditive genetic influences, shared and nonshared environmental influences simultaneously would not be statistically identified. In the present study, both *ACE* and *ADE* models were estimated for comparative purposes.

One objective of the present study was to examine the extent to which genetic and environmental influences on sexual orientation overlap with genetic and shared environmental influences on GAB. In this analysis, we used a bivariate genetic model in which both within-trait and cross-trait genetic and environmental influences were modeled (see Posthuma et al., 2003). With this type of model, it is possible not only to examine genetic and environmental contributions to the *variance* within each measure, but also the genetic and environmental contributions to the *covariance* between measures. One way of expressing the genetic and environmental covariance between measures is in terms of 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, whereas a genetic correlation of zero would indicate that different gene loci or effects influence the two measures.

The relative contributions of A, C/D, and E effects for each measure were estimated using a series of structural equation model-fitting analyses. Models were estimated by full-information maximum likelihood (FIML) estimation, using the program *Mx* (Neale et al., 2002). The goal of this process was to minimize twice the negative log-likelihood ($-2LL$), which is essentially an index of the discrepancy between the data and the model. A $-2LL$ estimate is estimated for each individual, and the individual $-2LL$ estimates are summed over the entire sample to estimate the overall $-2LL$. Comparisons between models were made using the likelihood comparison of the $-2LL$ estimate for the models, which is distributed as a chi-square statistic. A nonsignificant decrease in the $-2LL$ indicates that the model with fewer parameter provides a reliable but more parsimonious fit to the data compared with the full model. We also compared models using Akaike's Information Criterion (Akaike, 1987). Models having lower *AIC* values are preferred. We made use of the raw data in our analyses in order to minimize any bias resulting from missing data.

Results

Prevalence and Effects of Gender and Age on GAB and Homosexual Orientation Variables

Men reported overall significantly more gender *typical* behavior ($M = 4.47, SE = 0.01$) than women ($M = 3.67, SE = 0.01, F(1, 2489) = 2529.9, p < .001, R^2 = .39$), as indicated by their higher values on the RCGIGR scale. Age was associated with gender *typical* behavior for men, $r_p = .06$, $F(1, 2497) = 4.32, p < .038, R^2 = .003$, with older men reporting more gender typical behavior, but not for women, $r_p = .01$, $F(1, 2497) < 1, R^2 = .00$. However, a regression analysis that included an interactive term between age and gender showed that the association was not significantly stronger for men than for women, $F(1, 2497) < 1, R^2 = .38$.

The proportions of participants reporting a homosexual orientation were 6.1% for men and 6.6% for women. A

significant gender difference was found for the sexual orientation variable, with men reporting higher scores for homosexual orientation (men: $M = 0.13$, $SE = 0.02$; women $M = 0.06$, $SE = 0.01$), $F(1, 2492) = 11.36$ $p < .01$, $R^2 = .005$). Age was not associated with sexual orientation for men $r_p = -.02$, $F(1, 2492) = 1.18$ $p = .28$, $R^2 = .00$ or for women, $r_p = -.023$, $F(1, 2492) = 1.66$ $p = .20$, $R^2 = .001$.

Association Between GAB and Sexual Orientation

For both men and women, childhood GAB was associated with adult sexual orientation. Childhood gender *typical* behavior was negatively related to homosexual orientation for both men, $r_p = -.42$, $F(1, 2490) = 51.99$, $p < .001$, $R^2 = .18$, and women, $r_p = -.19$, $F(1, 2490) = 24.43$, $p < .001$, $R^2 = .048$. An interactive term between gender and gender typical behavior in regression analyses predicting sexual orientation showed that the strength of the effects of gender typical behavior on sexual orientation were significantly different for men and women, $F(1, 2490) = 34.06$, $p < .001$, $R^2 = .13$.

Phenotypic Analyses

Next, we tested for equality of means and variances of GAB and sexual orientation among MZ twins, DZ twins, and DZO twins. In these models, means and variances were equated across groups in consecutive steps, separately for men and women. The results are shown in Table 2. There was a decrease in model fit for men when constraining the means or the variances to be equal among MZ twins, DZM twins, and DZOM twins for GAB, but not for sexual orientation. For women, there was no decrease in model fit when constraining the means to be equal among MZ, DZF, and DZOF twins for neither variable. When constraining the variances between the twin groups to be equal for the sexual orientation variable, a decrease in model fit was found. However, when both the means and the variances were restricted to be equal for all identified twin groups, a significant reduction in model fit was found only for the GAB variable. This suggested that the opposite-sex DZ twins could be included in genetic analyses for the sexual orientation variable, but not GAB or for the multivariate analyses, which included both variables.

Twin Intra-Class Correlations

The scale for the RCGIGR was reversed for these analyses by subtracting the actual GAB score from the maximum score of 5.00 for the men, leaving us with a scale measuring "girlishness," that is, higher values indicating more girlish behavior for both men and women. This new scoring was then used for the rest of the analyses.

Table 2 Differences in means and variances between MZ, DZ and opposite-sex DZ twins for gender atypical behavior (GAB) and sexual orientation (SO)

Test	$\Delta\chi^2$ GAB	Δdf GAB	p GAB	$\Delta\chi^2$ SO	Δdf SO	p SO
Means						
MZM versus DZM	0.22	1	ns	0.02	1	ns
MZM versus DZOM	237.07	1	.00	2.21	1	ns
DZM versus DZOM	239.07	1	.00	2.39	1	ns
MZM versus DZM versus DZOM	241.85	2	.00	3.15	2	ns
MZF versus DZF	0.21	1	ns	0.45	1	ns
MZF versus DZOF	0.93	1	ns	0.01	1	ns
DZF versus DZOF	0.78	1	ns	0.37	1	ns
MZF versus DZF versus DZOF	1.0	2	ns	0.64	2	ns
Variances						
MZM versus DZM	13.78	1	.00	0.98	1	ns
MZM versus DZOM	516.69	1	.00	0.00	1	ns
DZM versus DZOM	870.20	1	.00	1.34	1	ns
MZM versus DZM versus DZOM	2637.33	2	.00	1.61	2	ns
MZF versus DZF	0.70	1	ns	1.36	1	ns
MZF versus DZOF	1.17	1	ns	2.44	1	ns
DZF versus DZOF	2.12	1	ns	5.62	1	.02
MZF versus DZF versus DZOF	2.46	2	ns	5.99	2	.05
All identified twins restricted to be equal for *M* and *SD*	1640.01	8	.00	11.41	8	ns

MZM monozygotic male pairs, *DZM* dizygotic male pairs, *MZF* monozygotic female pairs, *DZF*, dizygotic female pairs, *DZO* opposite sex dizygotic pairs

Table 3 Twin intra-class correlations (95% confidence intervals) for gender atypical behavior and sexual orientation

	MZM	DZM	MZF	DZF	DZO
GAB	0.56 (0.39, 0.68)	0.27 (0.00, 0.47)	0.53 (0.43, 0.60)	0.07 (−0.04, 0.18)	[a]–
Sexual orientation	0.50 (0.31, 0.63)	0.25 (−0.04, 0.46)	0.47 (0.37, 0.55)	−0.01 (−0.12, 0.09)	−0.01 (−0.13, 0.12)

MZM monozygotic male pairs, *DZM* dizygotic male pairs, *MZF* monozygotic female pairs, *DZF*, dizygotic female pairs, *DZO* opposite sex dizygotic pairs

[a] Not calculated due to the departures from assumptions of equal means and variances for the different groups of men

Table 3 shows the intra-class correlations for the studied variables, presented separately for MZ twins, same-sex DZ twins, and opposite-sex DZ twins. When inspecting the correlations, it was apparent that the MZ correlations were consistently higher than DZ correlations, for both studied variables. This indicates that genetic influences contributed to the variance in these measures. Genetic factors did not account completely for the observed variance, because the MZ twin correlations were not unity. This denotes that nonshared environmental influences and measurement error were also important. The correlations for both GAB and sexual orientation for male MZ twins were about twice the correlations for male DZ twins, suggesting the presence of genetic effects which are not likely to be dominant in nature. For female MZ twins, twin correlations were at least twice the correlations for same-sex DZ twins, implying models including a dominant genetic component (*ADE*). For comparative purposes, both models were fitted to the data, separately for men and women.

Univariate Genetic Analyses

Next, univariate *ACE* and *ADE* models were fitted to the data. This was done in one analysis for men and women for the GAB variable, excluding the DZO twins. For the sexual orientation variable, the analyses were conducted separately for men and women but with the DZO twins included. The results are shown in Table 4.

When fitting *ACE* models to the data, additive genetic effects were observed for both GAB (nonsignificant) and sexual orientation (nearly significant, $p = .08$) for men (49% and 29%, respectively) and significant effects for women (38% and 46%, respectively). The estimates for GAB for men indicate that approximately half of the variance can be explained by additive genetic effects and half by common environmental effects. The confidence intervals for the additive genetic effects for both GAB and sexual orientation overlapped for men and women, suggesting that the genetic effects were not significantly different for men and women. No significant shared environmental effects were observed.

When *ADE* models were fitted to the data, significant nonadditive genetic effects were found for women, for both GAB and sexual orientation (45% and 51%, respectively). For men, no nonadditive genetic effects were found. Confidence intervals for men and women overlapped neither for GAB nor for sexual orientation.

While testing for different submodels, when both additive and dominant genetic effects were restricted to zero, model fit worsened significantly for both men and women. This indicates that genetic effects were operative for each variable but that the statistical power was not adequate in order to separate between additive and dominant genetic effects. However, results for both GAB and sexual orientation indicated that an *AE* model more adequately fitted the data for men whereas an *ADE* or *DE* model was found to more adequately fit the data for women.

Quantitative gender differences were further tested by modeling the reduction in model fit, by a difference in χ^2, when restricting the *A*, *C/D*, and *E* components to be equal for men and women. As shown in Table 4, significant differences were found for some of the components, when testing for different models. In the analyses with the *ACE* model, quantitative gender differences were not found for sexual orientation. For GAB, a significant gender difference in the magnitude of genetic effects was found for the *A* component, as well as for the combined familial *AC* component. When analyses were conducted with the *ADE* model, quantitative gender differences were found for the sexual orientation variable, for the *A*, *D*, and *AD* components. For GAB, significant gender differences were found for the *D* and *AD* components.

Qualitative gender differences were also assessed for the sexual orientation variable, in analyses including only DZO twins. For GAB, qualitative gender differences could not be tested as the DZO twin pairs differed significantly with regard to the means and variances, as shown in Table 2. The qualitative gender differences for sexual orientation were tested with both *ACE* and *ADE* models. This was done in consecutive steps, by restricting the additive genetic correlation across sexes to be .5 and the nonadditive genetic correlation to be .25 and testing whether this resulted in a significant reduction of model fit. No qualitative gender differences were analyzed for shared environment, as the *C* component for women was nonexisting with a value of zero.

In the analyses with the *ACE* model, a tendency toward a reduction in model fit when the additive genetic correlation between DZO male and female twin pairs was fixed at .5 (observed $r_g = .016$, $\Delta\chi^2 = 2.84$, $p = .09$), indicating that

Table 4 Quantitative gender difference estimates (95% confidence intervals) from full ACE and ADE models for gender atypical behavior (RCGIGR) and sexual orientation

	A	*C*/*D*	*E*	−2*LL	AIC	$\Delta\chi^2$			Quantitative difference *A* $\Delta\chi^2$	Quantitative difference *C*/*D* $\Delta\chi^2$	Quantitative difference *AC*/*AD* $\Delta\chi^2$
						ACE versus CE ADE versus DE	ACE versus AE ADE versus AE	ACE versus E ADE versus E			
ACE											
Sexual orientation MEN	0.49 (0.00, 0.62)	0.01 (0.00, 0.45)	0.51 (0.37, 0.69)	−5975.64	−12439.64	3.00†	0.00	22.07***			
Sexual orientation WOMEN	0.38 (0.28, 0.47)	0.00 (0.00, 0.05)	0.62 (0.53, 0.72)	−5975.64	−12439.64	24.28***	0.00	51.66***			
									1.38	0.00	0.33
GAB MEN	0.29 (0.00, 0.61)	0.21 (0.00, 0.54)	0.50 (0.38, 0.67)	3206.79	−1891.21	1.26	0.57	31.77***			
GAB WOMEN	0.46 (0.36, 0.55)	0.00 (0.00, 0.07)	0.54 (0.46, 0.63)	3206.79	−1891.21	26.92***	0.00	77.53***			
									13.99***	0.57	25.80***
ADE											
Sexual orientation MEN	0.50 (0.32, 0.62)	0.00 (0.00, 0.09)	0.50 (0.38, 0.68)	−5988.28	−12452.28	22.04***	0.01	22.07***			
Sexual orientation WOMEN	0.00 (0.00, 0.15)	0.45 (0.27, 0.53)	0.55 (0.47, 0.65)	−5988.28	−12452.28	0.00	12.64***	64.30***			
									11.18***	4.80*	18.12***
GAB MEN	0.51 (0.00, 0.62)	0.00 (0.00, 0.59)	0.49 (0.38, 0.65)	3199.14	−1898.86	1.78	0.00	31.19***			
GAB WOMEN	0.00 (0.00, 0.29)	0.51 (0.20, 0.58)	0.49 (0.42, 0.58)	3199.14	−1898.86	0.00	8.22**	85.75***			
									1.59	6.01*	33.42***

A Additive genetic influences, *C* common environmental influences, *D* nonadditive genetic effects, *E*, nonshared environmental influences, *AIC* Akaike Information Criterion. For analyses with the GAB variable DZO twins were excluded, whereas for analyses with the sexual orientation variable, DZO twins were included

* $p < .05$, ** $p < .01$, *** $p < .001$, † $p \leq .10$

different additive genetic factors might influence sexual orientation of men and women.

For the *ADE* model there was no significant reduction in model fit when either the additive genetic correlation (r_g = .34, $\Delta\chi^2$ = .00, ns) or the dominant genetic correlation (r_g = −.25, $\Delta\chi^2$ = .00, ns) was fixed at .5 and .25, respectively, for the DZO male female twin pairs.

The qualitative gender differences that were found thus concerned the additive genetic component, but as different models more adequately described the data for men (*AE*) and women (*DE*) definite conclusions are difficult to draw.

Multivariate Genetic Analyses

Next, bivariate Cholesky models were used to evaluate how much of the phenotypic correlations between the measured traits resulted from shared genetic and environmental effects. The results are shown in Table 5. Analyses were conducted separately for men and women, excluding DZO twins, and separately for *ACE*, *ADE*, *AE* and *DE* models. The parameters of interest were the genetic and nonshared environmental correlations which were each removed in sequence from the bivariate models.

For men, the additive genetic correlation between GAB and sexual orientation, in the best fitting *AE* model, was .73 whereas the correlation for common unique environmental effects did not reach the level of statistical significance. Neither in the *ACE*, nor the *ADE* model, did genetic or environmental correlations reach the level of statistical significance. The high genetic correlation implies that the shared genetic covariation between GAB and sexual orientation was substantial.

For women, significant correlations for GAB and sexual orientation were found, −.28 for additive genetic effects and .30 for dominant genetic effects, in the most adequately fitting *DE* model. The correlation due to shared unique environmental effects was low, −.12, but significant. Significant correlations were found in the *ACE* model, −.28 for additive genetic effects and −.15 for unique environment, as well as for the *ADE* model, −.30 for dominant genetic effects and −.12 for unique environment. The negative correlations imply that highly girlish behavior does not genetically covary with a homosexual sexual orientation.

Table 5 Genetic and environmental correlations between GAB and sexual orientation due to *A*, *C* or *D* and *E*

	r_a	r_d	r_c	r_e
Men				
ACE	.61	–	1.00	.17
ADE	.80	1.00	–	.16
AE	.73***	–	–	.15
Women				
ACE	−.28**	–	−.57	−.15**
ADE	1.00	−.30*	–	−.12*
DE	–	−.30***	–	−.12*

r_a, Correlation between additive genetic effects; r_d, correlation between nonadditive genetic effects; r_c, correlation between shared environmental effects; r_e, correlation between nonshared environmental effects

* $p < .05$, ** $p < .01$, *** $p < .001$

Discussion

Our first aim was to study the phenotypic correlations between childhood GAB and adult sexual orientation. Significant correlations of moderate sizes were found, indicating that the two phenomena were related. The strength of the phenotypic association was higher for male participants, implying that childhood GAB was a stronger predictor of adult sexual orientation for men. This is in line with previous findings (Bailey & Zucker, 1995). An interesting parallel is the result from a Finnish study that assessed parental attitudes toward gender atypical boys and girls (Sandnabba & Ahlberg, 1999). When predicting future sexual orientation, gender atypical boys were deemed to have a greater likelihood of becoming gay than gender atypical girls of becoming lesbian. This lay theory of the etiology of homosexuality may have influenced the observation made in the same study, namely, that GAB was much less accepted for boys than for girls. Gender atypical girls were expected to have grown out of masculine characteristics by adulthood. It is possible that since GAB is more frequent in girls than in boys, the gender atypical boys will to a greater extent stick out from other boys as peculiar and much more effort will be made to correct the behavior of gender atypical boys than girls.

In light of the assumption that GAB in boys is regarded rather negatively, it is interesting that the phenotypic association between GAB and sexual orientation was stronger for men. If GAB was socially more accepted for boys, the strength of the association might diminish, as also those who would become heterosexual adults could explore different behaviors during childhood. The association might also reflect a tendency for boys to hide their GAB or that there is a differing biological determinism behind the etiology of the association for boys/men than for girls/women (Bailey et al., 2000). If the shared family environment was rejecting of GAB, a common environmental effect should have been found in the present study. However, no such effect was found, which could be due to the fact that gender norms are transmitted on a social level. In the present study, this might have been reflected by significant unique environmental effects. Cross-cultural studies might be informative for comparing shared environmental and unique (or social) environmental effects.

Significant genetic effects were found for women and men for both GAB and sexual orientation, as was our second hypothesis. The heritability estimates for childhood GAB were 51% and 29%, and for sexual orientation 45% and 50%, for women and men, respectively. In the model-fitting analyses, different models were found to have best fit for men and women for both GAB and sexual orientation (*DE* models for women, *AE* models for men). Gender differences in genetic effects have also been found in other studies (Bailey et al., 2000; Hershberger, 1997; Kirk et al., 2000; Knafo et al., 2005). The findings in the present study were in line with previous studies regarding GAB, as well as the female estimates for sexual orientation. However, the genetic estimates for male sexual orientation were somewhat higher in the present study than what has been reported previously (Bailey et al., 2000; Hershberger, 1997; Kirk et al., 2000). One needs to bear in mind that it was not possible to clearly distinguish between additive and nonadditive genetic influences for men, just that genetic factors were important for both GAB and sexual orientation.

The fact that different models best fitted the data for men and women might be explained in various ways. The nonadditive nature of genetic effects that in the present study were found for women for both GAB and sexual orientation would suggest that the genetic effects might be fitness related (Merilä & Sheldon, 1999). The nonadditive effects found in the present study could indicate that recessive alleles are influencing sexual orientation. Recessive alleles have a higher likelihood of remaining in the population even if they are associated with reduced fitness, which may be the case for homosexual orientation. Although expected to gradually vanish from the gene pool (Buss, 1999), such alleles might still survive as a result of, for example, mutation or balanced polymorphism (for details, see Miller, 2000). Alternatively, selective pressure during human evolution might have caused different genetic architecture resulting in gender differences (Neale & Cardon, 1992). For instance, a situation where balanced polymorphism maintains the alleles in the gene pool might have come up as some feminine characteristics have led to reproductive benefits for males who possess them (Miller, 2000).

Our third aim was to study the common underlying covariation between GAB and sexual orientation. We found shared genetic correlations between GAB and sexual orientation for both men and women. The large genetic correlation, especially for men, suggests that there are shared genes for GAB and sexual orientation and substantial covariation between the traits. The additive genetic correlation was stronger for men than for women. However, confidence intervals did overlap with the female estimates, indicating that the effect might not be significantly stronger. The genetic correlation should be regarded as statistical pleiotropism, that is, when allelic effects on one trait predict allelic effects on another trait (Carey, 1988). Future studies could focus on genetic polymorphism, that is, when more than one allele exists in the population, and different etiologies should be expected for men and women, as suggested by the present study as well as other studies (e.g., Bailey et al., 2000).

Significant nonshared environmental correlations were found for women, suggesting that the same nonshared environment can contribute to both traits. The nonshared environment includes measurement error, meaning that it can also imply that measurement errors were alike for both variables. Bailey et al. (2000) suggest that actual behavior might be more environmentally restricted than interest. This may be especially true for same-sex sexual behavior due to the restricted social opportunities for getting to know potential partners, at least in less urbanized areas, etc.

We also studied whether the genetic and shared environmental influences affecting sexual orientation were the same for men and women. We found a tendency toward qualitative gender differences for the genetic component for sexual orientation, which, however, did not reach statistical significance. As different models best fit the data for men (*AE*) and women (*DE*), it is difficult to interpret the results. However, it can be assumed that, with a larger sample size more prominent differences could be found, and that different genes do, in fact, affect the sexual orientation of men and women. A large discrepancy between the DZO and the same-sex DZ twin correlations further supports the assumption that different genes affect the expression of GAB and sexual orientation in men and women.

Different forms of connections between GAB and sexual orientation have been suggested. Bailey et al. (2000) examined whether the observed association between homosexual orientation and GAB was explained by shared genetic or environmental influences. GAB and sexual orientation were influenced by a latent phenotypic component that differed markedly between the sexes. They hypothesized that the latent factor reflected early hormonal exposure. The EBE theory by Bem (1996, 2000) suggests that GAB mediates the development from biological factors, such as genes or hormones, to sexual orientation. Dawood et al. (2000) proposed that homosexuality that has been preceded by GAB has different causes than homosexuality that was preceded by a gender typical childhood. There might, in other words, be different genotypes for different kinds of homosexuality. It might also be possible that the relative importance of shared environment and genetic influences vary during development. It is plausible that parents influence their children directly only as long as they live at home (Knafo et al., 2005; Plomin et al., 2001). Bailey et al. (2000) found that GAB predicted about 30% of the variance in men's sexual orientation. As neither the phenotypic nor the genetic correlations were unity in the present sample, GAB preceded a homosexual orientation for some participants, whereas gender

typicality preceded a homosexual orientation for other participants.

Heritability is not a fixed parameter, but may change during life. Further, the heritability estimates obtained from twin studies are population measures (i.e., explain the reasons for variation at a group-level) and cannot be applied to the individual (Plomin et al., 2001). Also, because heritability is population-specific, it is probably not surprising that different studies, based on different populations, have yielded diverse heritability estimates. One limitation of the study is the generalizability of findings. However, as more researchers replicate studies and findings, results become more generalizable.

Retrospective measures of childhood GAB have been criticized for being vulnerable to biased recall. However, in several studies, this method has been proved to be valid for the kind of study in hand (Bailey et al., 1993; Zucker et al., 2006). A measure of aversion for gender typical activities could be included in further studies of GAB (Bem, 2000), as it could better distinguish between "partly and totally" gender atypical children (Knafo et al., 2005).

The relatively low response rate raises questions regarding the representativeness of the sample. A discussion of the representativeness of the present sample can be found elsewhere (Varjonen et al., 2007). The question of sufficient power in statistical analyses is also present. At least 200 pairs are required for twin analyses for highly heritable traits, whereas for low or moderate heritable traits much larger sample sizes are required (Rijsdik & Sham, 2002). In our analyses, the sample size did exceed 200, and allowed us to detect heritability for the variables studied, but failed to suffice for low heritable traits. Further, overlapping confidence intervals, when comparing men and women, should lend some caution to the interpretations, as results are not clear-cut.

In sum, we found genetic effects on GAB and sexual orientation for both men and women. The genetic correlation between GAB and same-sex sexual orientation was substantial for male and moderate for female participants. The findings indicate a shared genetic influence for the traits.

Acknowledgments This research was financed by Grant No. 210298 from the Academy of Finland and a Centre of Excellence Grant from the Stiftelsen för Åbo Akademi Foundation and personal grants to the first author from the Signe and Ane Gyllenberg Foundation, the Waldemar von Frenkell Foundation, and the Stiftelsen för Åbo Akademi Foundation.

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