Journal of Personality and Social Psychology

Volume 78(3) March 2000 p 524–536

Genetic and Environmental Influences on Sexual Orientation and Its Correlates in an Australian Twin Sample

[Personality Processes and Individual Differences]

Bailey, J. Michael[1,5]; Dunne, Michael P.[2]; Martin, Nicholas G.[3,4]

[1]Department of Psychology, Northwestern University
[2]School of Public Health, Queensland University of Technology, Brisbane, Queensland, Australia
[3]Epidemiology Unit, Queensland Institute of Medical Research, Brisbane, Queensland, Australia
[4]Joint Genetics Program, University of Queensland, Brisbane, Queensland, Australia.
[5]Correspondence concerning this article should be addressed to J. Michael Bailey, Department of Psychology, Northwestern University, 2029 Sheridan Road, Evanston, Illinois 60208-2710. Electronic mail may be sent to jm-bailey@nwu.edu.

We thank Joan Linsenmeier for her comments on a previous version of this article.
Received Date: January 7, 1998; Revised Date: July 22, 1999; Accepted Date: July 30, 1999

Outline

Graphics

Abstract

We recruited twins systematically from the Australian Twin Registry and assessed their sexual orientation and 2 related traits: childhood gender nonconformity and continuous gender identity. Men and women differed in their distributions of sexual orientation, with women more likely to have slight-to-moderate degrees of homosexual attraction, and men more likely to have high degrees of homosexual attraction. Twin concordances for nonheterosexual orientation were lower than in prior studies. Univariate analyses showed that familial factors were important for all traits, but were less successful in distinguishing genetic from shared environmental influences. Only childhood gender nonconformity was significantly heritable for both men and women. Multivariate analyses suggested that the causal architecture differed between men and women, and, for women, provided significant evidence for the importance of genetic factors to the traits' covariation.

The causes of sexual orientation have provoked intense scientific interest, inspiring both empirical work and theory (Bell, Weinberg, & Hammersmith, 1981; Bem, 1996a; Ellis & Ames, 1987; Hamer & Copeland, 1994; LeVay, 1996). This interest stems, in part, from the mostly mistaken belief that different etiological accounts of sexual orientation have different social and ethical implications (Bem, 1996b; Greenberg & Bailey, 1993; Schmalz, 1993; Stein, 1994). But there are also legitimate and important scientific reasons for interest in the issue. Sexual orientation is a fundamental aspect of human sexuality, guaranteeing that for the large majority, men mate with women. Furthermore, sexual orientation is empirically closely linked to some aspects of gender roles, including childhood play behavior and gender identity (Bailey & Zucker, 1995; Bell et al., 1981; Green, 1987; Zuger, 1988) and aspects of adult sex-typed behavior as well, particularly occupational and recreational interests (Bailey, Finkel, Blackwelder, & Bailey, 1996; Lippa, 1998). Thus, illuminating the origins of sexual orientation could also shed light on the development of other important sex differences.

Empirical research about the origins of sexual orientation has been organized, generally, around the nature–nurture dichotomy, motivated by two different theoretical approaches. The first, sometimes called the “neurohormonal” or “neuroendocrine” theory (Ellis & Ames, 1987; Meyer-Bahlburg, 1987), examines the possibility

that homosexual people have been subject to atypical levels of hormones in development, thus causing sex-atypical neural diffentiation. LeVay's finding that for one hypothalamic nucleus, gay men are more similar to heterosexual women than to heterosexual men is perhaps the most important finding motivated by this perspective (LeVay, 1991). The second approach, behavioral genetics, has focused on whether sexual orientation is familial, and if so, whether familial aggregation is attributable to genetic or shared environmental factors. It is important to emphasize that the two approaches are not competing theories but represent different levels of analysis. The present study is primarily an example of the second approach, although some of the variables considered (e.g., childhood gender nonconformity) are also highly relevant to the neurohormonal approach.

Distribution of Sexual Orientation

In recent years a great deal of attention has been given to the prevalence of homosexuality (e.g., Diamond, 1993; Laumann, Gagnon, Michael, & Michaels, 1994). Media accounts have focused on the prevalence of homosexual behavior per se. Most current researchers, including us, define sexual orientation psychologically rather than behaviorally (e.g., Money, 1988; LeVay, 1996, p. 45; Zucker & Bradley, 1995). Sexual orientation is one's degree of sexual attraction to men or women. Of course, sexual orientation should be closely related to sexual experience with one sex or the other, though many factors, especially social ones, could cause sexual orientation and sexual behavior to correlate less than perfectly.

Kinsey's original surveys of sexual orientation suggested a rather marked sex difference (see LeVay, 1996, pp. 47–49). For men, sexual orientation appeared to be somewhat bimodal, but for women, it tapered gradually from strictly heterosexual to strictly homosexual orientation, with no elevation at the latter end of the sexual orientation scale. If this difference is true, it would suggest that sexual orientation development and phenomenology differ between the sexes. Unfortunately, Kinsey's assessment of sexual orientation confounded behavioral and psychological measures. Furthermore, his sampling scheme was notoriously haphazard (Laumann et al., 1994, pp. 44–45).

Familiality and Genetics of Sexual Orientation

Both male and female homosexuality appear to run in families (Bailey & Bell, 1993; Bailey & Benishay, 1993; Pattatucci & Hamer, 1995; Pillard, 1990; Pillard & Weinrich, 1986). Studies of unseparated twins have suggested that this is primarily due to genetic rather than familial environmental influences (Bailey & Pillard, 1991; Bailey, Pillard, Neale, & Agyei, 1993). Furthermore, there is some evidence that male sexual orientation is influenced by a gene on the X chromosome (Hamer, Hu, Magnuson, Hu, & Pattatucci, 1993; Hu et al., 1995), though some other studies have found contradictory evidence (Bailey et al., 1999; Rice, Anderson, & Ebers, 1995).

Although prior twin studies have been generally consistent in indicating a genetic contribution to male and female sexual orientation, they have also been rather consistent in their methodological limitations. Most importantly, all sizable twin studies of sexual orientation recruited probands by means of advertisements in homophile publications or by word of mouth (Bailey & Pillard, 1995). Such sampling is likely to result in volunteer bias that affects twin concordances and heritability analyses (Kendler & Eaves, 1989), though it is difficult to estimate the direction or magnitude of the bias from available information. Furthermore, respondents with exclusively homosexual orientations may be overrepresented, and those with modest levels of homosexual attraction, underrepresented, obscuring the potentially continuous nature of sexual orientation.

Correlates of Sexual Orientation

Another limitation of prior twin studies of sexual orientation has been their frequent neglect of its correlates. Specifically, a great deal of evidence supports an association between childhood gender nonconformity and sexual orientation (Bailey & Zucker, 1995). That is, gay men tend to recall having been feminine boys, and lesbians, masculine girls. Prospective studies have validated this association for men (Green, 1987; Zuger, 1988); prospective studies of masculine girls have not yet been conducted. The association between childhood gender nonconformity and adult sexual orientation has been so well-documented and is so strong that Bem (1996a) remarked: "It is difficult to think of other individual differences (besides IQ or sex itself) that so reliably and so strongly predict socially significant outcomes across the life span, and for both sexes, too. Surely it must be true" (p. 323). Bem, and others, have recognized that developmental and etiological theories of sexual orientation must try to account for the association between the two traits, and Bem has proposed a theory intending to do precisely that. The theory, "exotic becomes erotic," specifies that biological factors may cause childhood gender nonconformity and that gender-nonconforming children tend to feel different from other children of their sex, and as a result (through a rather complicated pathway), eroticize them. Bem's theory hypothesizes that childhood gender nonconformity is influenced by biological factors, perhaps including genetic ones. This hypothesis has been examined using a neurohormonal approach (Berenbaum & Hines, 1992), but it has not been tested through behavior genetics. Although some prior twin studies assessed (retrospective) childhood gender nonconformity (Bailey & Pillard, 1991; Bailey, Pillard, et al., 1993), their sampling of homosexual probands (as opposed to sampling from a population of unspecified sexual orientation) prevented either univariate or multivariate genetic analysis of traits other than sexual orientation.

There have been fewer studies examining whether sexual orientation has analogous associations with adult behavior, but available evidence is generally consistent with this possibility (Bailey et al., 1996; Finn, 1987; Lippa, 1998; Pillard, 1991). Occupational and recreational interests appear to be correlated with sexual orientation (Bailey & Oberschneider, 1997; Lippa, 1998), as does gender identity when considered as a continuous trait (Finn, 1987). That is, homosexual people feel somewhat like the opposite sex, compared with heterosexual people, although they recognize and accept their biological sex. One study examined the genetic and environmental influences on sex-typed occupational interests and yielded a moderate heritability estimate for both males and females (Lippa & Hershberger, 1999). No study has examined the heritability or environmentality of gender identity, in either its dichotomous or continuous sense.

The desirability of exploring the heritability of correlates of sexual orientation, as well as sexual orientation itself, is consistent with Gottesman's recommendation to study *endophenotypes* (Gottesman, 1998; Gottesman & Shields, 1972). Endophenotypes are characteristics related to the phenotype of primary interest (in this case, sexual orientation) that are hypothesized to "mediate the impact of gene products on emergent systems" (Gottesman, 1998, p. 1522). That is, endophenotypes may in some sense be "closer to the genes" than the primary phenotype. Thus, Bem's hypothesis that childhood gender nonconformity is inherited rather than being due to sexual orientation per se is equivalent to the position that childhood gender nonconformity is an important psychometric (as distinct from biological) endophenotype of homosexuality. A wider focus, including correlates of sexual orientation such as childhood gender nonconformity and relevant adult traits, is also desirable because such correlates are likely to be more variable than sexual orientation. If so, then genetic linkage studies aimed at elucidating the influences on sexual orientation could focus on sibling pairs either concordant or discordant for extreme scores on these correlates, rather than for homosexuality per se. One could set appropriate cutoffs (e.g., one standard deviation above or below the mean) so that a higher percentage of sibling pairs qualified for a relevant study than they would if sexual orientation were the only qualifying variable. Finally, it is generally much more personally and politically sensitive to assess sexual orientation than its correlates. To the extent that sexual orientation can be studied indirectly through its correlates, relevant research can be performed more easily.

The Present Study

In the present study we examined the distributions, correlates, and causes of male and female sexual orientation using a large sample of Australian twins (N = 4,901), who represent the largest carefully ascertained twin sample ever assembled for such a study. More specifically, we addressed four main issues: (a) the comparison of male and female distributions of sexual orientation; (b) the construct validity of sexual orientation self-ratings (Kinsey scores) indicating low but positive levels of homosexual interest; (c) the relative role of genes, shared environment, and nonshared individual-specific environment in the development of sexual orientation, childhood gender nonconformity (with special attention to the validity of this retrospectively measured variable), and adult masculinity–femininity (using the Continuous Gender Identity [CGI] measure); and (d) the genetic architecture of their covariation.

Method

Participants

Participants were drawn from the Australian National Health and Medical Research Council Twin Register (ATR). The ATR is a volunteer register begun in 1978 and has about 25,000 twin pairs of all zygosity types and all ages enrolled and in various stages of active contact. Participants for this study were recruited from two phases of a large twin-family study of alcohol use and abuse (Heath et al., 1994). The twins were resident in all eight states and territories of Australia. There is a disproportionate number of young women and people with higher than average levels of education (Baker, Treloar, Reynolds, Heath, & Martin, 1996). In relation to psychological factors, comparisons with normative data from the 1980s indicated that participants were generally representative of the Australian population in terms of personality and depression (Jardine, Martin, & Henderson, 1984) and alcohol consumption (Jardine & Martin, 1984). Diversity within the sample in terms of religious affiliation and social attitudes has been documented elsewhere (Dunne, Martin, Pangan, & Heath, 1997; Martin et al., 1986).

During 1992 we asked all ATR twins between ages 17 and 50 years who had completed a postal health and lifestyle survey (HLQ) between 1988 and 1990 (N = 9,112) about their willingness to receive a questionnaire regarding sex. Specifically, they were asked, “We have applied for funding to carry out an *anonymous* study of sexual behaviour and attitudes. Would you be willing to receive a questionnaire with explicit questions on these topics?” All those who said “Yes” were mailed the sex questionnaire. When participants received the sex questionnaire, they were asked to complete a consent form with their name, date of birth, and signature and to return this separately to indicate whether they had consented to complete the sex questionnaire. Anonymity was assured, and we asked co-twins to privately choose the same 10-digit identification number so that we could match them with their questionnaires. Approximately 2 weeks after the initial mailing of the sex questionnaire, all twins were sent a reminder letter. Consent forms were logged as they were returned, and subsequently all twins who had not returned a consent form were followed up once by telephone. Because we received many queries from twins asking whether they should complete the questionnaire if their co-twin had decided not to participate, we sent a further letter urging such “singles” to cooperate.

Twenty-eight percent explicitly refused to participate, and 54% (4,901) completed questionnaires. The remainder (18%) initially agreed to participate but did not respond when contacted (following one letter or one phone call). Our response rate was not substantially lower than that of other recent large-scale mail sex surveys, which have typically achieved responses between 55 and 65% (Biggar & Melbye, 1992; Johnson et al., 1989; Sundet, Magnus, Kvalem, Samuelsen, & Bakketeig, 1992). Similar to other volunteer samples, ours overrepresented women and monozygotic (MZ) twins (Lykken, McGue, & Tellegen, 1987). In a recent analysis, we compared the individuals who returned the sex survey consent form with those who did not on a range of psychological and behavioral characteristics derived from data collected in other, thematically unrelated research interviews carried out with these twins between 1988 and 1994 (Dunne, Martin, Bailey, et al., 1997).

There was some indication of a modest participation bias. People who returned consent forms and those who initially agreed to participate but could not subsequently be contacted generally had more liberal sexual attitudes, more novelty-seeking and less harm-avoidant personalities, an earlier age of first sexual intercourse, and a greater likelihood of childhood sexual abuse than people who explicitly refused to participate in the sex survey. However, the effect sizes were small, suggesting that the behavioral data in the mailed sex survey probably did not seriously misrepresent sexual activity and attitudes.

With respect to sample sizes of twin pairs, there were 312 male MZ, 182 male dizygotic (DZ), 668 female MZ, 376 female DZ, and 353 opposite-sex DZ complete pairs. However, sample sizes varied somewhat in different analyses because of missing data. The median age for both men and women was 29 years.

Measures

Zygosity.

Zygosity of twins was established when twins completed the HLQ, from their response to standard items about physical similarity and being mistaken for each other. Subsequently, we indicated this zygosity on the anonymous questionnaires that we sent to participants. Such items have been shown to be at least 95% accurate when judged against genotyping results (e.g., Kasriel & Eaves, 1976; Martin & Martin, 1975). We further improved on this by selecting for further investigation any pair whose answers were not completely consistent, within or between co-twins, with either mono- or dizygosity. These pairs were telephoned to detect the source of any confusion, and about 80% were readily decided on the phone. Those still equivocal were asked to send us photographs at several stages of their lives and most were then assigned with little hesitation by the project staff, leaving but a few genuinely doubtful cases. Where possible, blood was subsequently obtained for genotyping these few uncertain pairs. We recently genotyped 329 same-sex pairs whose zygosity had been assigned using the above procedures, with 11 independent highly polymorphic markers. Of 131 pairs who reported themselves to be DZ, 5 (3.8%; 4 female, 1 male) were concordant at all loci, with a probability of monozygosity of over 0.9999. Of the 198 pairs who reported themselves to be MZ, none was found to be DZ. The accuracy overall was 98.5% (Duffy, 1994).

Sexual orientation.

Sexual orientation was measured using the Kinsey Scale (Kinsey, Pomeroy, & Martin, 1948), which ranges from 0 (*exclusively heterosexual*) to 6 (*exclusively homosexual*). The questionnaire included separate Kinsey Scale items to assess sexual fantasies (i.e., the degree to which participants fantasize about their own or the opposite sex), sexual attraction (i.e., the degree to which they are attracted to their own or the opposite sex), and sexual behavior (i.e., the relative frequency of same-sex or opposite-sex partners). In this article, we focus on psychological rather than behavioral sexual orientation because we suspect that the former is more stable than the latter and, furthermore, because psychological sexual orientation can typically be measured for individuals who have not had sex. Thus, the main dependent variable of this article was calculated as the average of scores from the Kinsey fantasy items and Kinsey attraction items. For men, these two items correlated .92, and for women, .67. To be consistent with the sexual orientation literature, which typically uses 7-point Kinsey scores, we have rounded the averaged Kinsey scores down (to be conservative) to the nearest integer.

Childhood gender nonconformity.

The measures of male and female childhood gender nonconformity (CGN) included items retrospectively

assessing childhood sex-typed behavior (i.e., participation in sex-stereotypic games and activities) and gender identity (i.e., internal feelings of maleness or femaleness). “Childhood” was defined as before the age of 12.

Our CGN measures were adapted from several published scales, by taking relevant items (e.g., those related to childhood rather than adulthood) and, in some cases, rewriting the items so that they were appropriate for Australian participants (e.g., “cricket” rather than “baseball”). For males, items were taken from the Gender Identity Scale for Males (Freund, Langevin, Satterberg, & Steiner, 1977), the Childhood Play Activities Checklist (Grellert, Newcomb, & Bentler, 1982), the Recalled Childhood Gender Behaviors Questionnaire (Mitchell & Zucker, 1992), and the Physical Aggressiveness Scale (Blanchard, McConkey, Roper, & Steiner, 1983). For females, items were taken from the Childhood Play Activities Checklist, the Recalled Childhood Gender Behaviors Questionnaire, and the Masculine Gender Identity Scale (Blanchard & Freund, 1983). All of these scales have been shown to differ reliably between homosexual and same-sex heterosexual individuals, as indeed have all scales of similar content (Bailey & Zucker, 1995). Although retrospective measures raise validity concerns, including recall biases and forgetting, a study of gay men and their mothers found moderate to high agreement between the two sets of informants regarding the men's childhood gender nonconformity (Bailey, Nothnagel, & Wolfe, 1995).

Both questionnaires (one for men and one for women) contained 24 items, but the items differed between the two versions, and so male and female scores were not comparable. Items varied in their response format and included both dichotomously rated items and rating scales. Scree tests of the principal components suggested that for each sex, one general factor primarily accounted for the item intercorrelations. Items were standardized within sexes and summed to yield a total CGN score. Coefficient alpha was .79 for both male and female CGN. Because raw total scores do not have a meaningful scale, whenever possible, we report results concerning this scale in terms of effect sizes: either correlations or standardized mean differences.

To explore the interrater reliability of the CGN measure in the present study, we also included a five-item scale in which twins rated their own relevant childhood behaviors compared with those of their co-twins (e.g., “Who was more athletic?”), using response choices “Me,” “My twin,” or “About the same.” Higher scores on this scale (Co-Twin Comparative CGN) indicated that the respondents considered themselves more gender nonconforming than their twins. Coefficient alpha for this scale was .71 for males and .58 for females (same-sex pairs only). Same-sex co-twins' scores correlated -.56 for men and -.65 for women, indicating that when one twin recalled being more masculine than the co-twin, the co-twin tended to recall being more feminine than the twin. For each same-sex pair, we then subtracted the second twin's scores from the first twin's scores so that the total score was highest when both twins considered the first twin to have been more gender nonconforming than the second. We then correlated this composite with the difference in CGN, the primary measure of childhood gender nonconformity used in this study. This correlation was .45 for men and .48 for women, indicating that twins' ratings of childhood gender nonconformity with respect to each other were moderately related to differences in the general CGN measure.

Continuous Gender Identity.

This scale (CGI) consisted of seven items taken from Finn (1987). The items assessed participants' self-concepts as masculine or feminine (e.g., “In many ways I feel more similar to women [men] than to men [women].”) using 7-point rating scales. A subscale including these items (as well as some other items, primarily concerning childhood gender nonconformity) distinguished homosexual from same-sex heterosexual individuals (Finn, 1987). Furthermore, a study of gay and lesbian couples found that scores on this scale were moderately correlated with partner's ratings of respondent's masculinity–femininity (Bailey et al., 1996).

Separate scree tests for each sex were both consistent with a single factor underlying CGI item intercorrelations. Items were summed to yield total scores, and coefficient alpha was .52 for men and .57 for women.

Data Analysis: Genetic and Environmental Model Fitting

Univariate analyses.

Two indices of twin similarity for sexual orientation were computed: the probandwise concordance and the polychoric correlation. The *probandwise concordance* is an estimate of the probability that a twin is nonheterosexual given that his or her co-twin is nonheterosexual. A proband in our study is any twin who is nonheterosexual by relevant criteria and who was recruited without regard to his or her co-twin's sexual orientation. Because we did not examine the sexual orientation data until data collection was complete, all twins were recruited blindly with respect to their co-twins' data. Thus, probands were all twins in complete pairs (both twins completed the questionnaire) who met the respective criterion for nonheterosexuality. Concordant pairs (++) thus contained two probands; discordant pairs (+-), one proband; and uninformative pairs (--), none. Probandwise concordance (McGue, 1992), provided for same-sex pairs, is the percentage of probands whose twins also met the criterion and is calculated as 2(++)/[2(++)+(+-)]. Probandwise concordance is preferable to pairwise concordance because, among other reasons, it does not depend on the probability of ascertainment. Furthermore, probandwise concordance is the appropriate index to compare with data concerning other types of relatives (e.g., the percentage of nontwin brothers of gay men who are also gay), as well as population prevalence estimates.

The *polychoric correlation* is computed from the full contingency table cross-classifying the sexual orientation status of a twin by the sexual orientation status of the co-twin. The polychoric correlation is the appropriate correlation if one assumes that scores on the observed ordinal variables can be explained by underlying normally distributed variables (Falconer, 1989; Gottesman & Carey, 1983; Reich, Cloninger, & Guze, 1975). In this case, the continuum underlying sexual orientation status was presumed to be the tendency to different degrees of homosexual feelings, which were measured using the primary Kinsey Scale (i.e., the average of scores from the Kinsey attraction items and Kinsey fantasy items). The Kinsey Scale itself is assumed to be ordinal rather than interval. The implied statistical model also includes thresholds, representing points on the underlying continuum at which influences are sufficient to cause progressively stronger degrees of homosexual attraction. Thresholds are inferred from the prevalences of the different Kinsey scores in the sample. The polychoric correlation is the correlation between co-twins' hypothesized normally distributed latent tendency to become nonheterosexual. The same basic statistical model is appropriate to the indexes of similarity for the two primary correlates of sexual orientation: CGN and CGI, and polychoric correlations were also computed for those variables. We computed polychoric correlations using the PRELIS computer program (Jöreskog & Sörbom, 1993b). Confidence intervals of polychoric correlations (and parameter estimates derived from them) can be asymmetric, and thus we provide confidence intervals rather than standard errors.

We emphasize that the validity of polychoric correlation estimates, as well as parameter estimates derived from them, depends on the validity of the model used to generate them. The implied etiological model is multifactorial; that is, both genetic and environmental influences are assumed to be manifold, with any single gene or environment having a small effect across the population. (This means that any gene or environment that has a large effect in particular cases is rare.) If, for example, either a single gene or an environment by itself exerted a substantial influence on sexual orientation, then estimates would be biased. However, heritability and environmentality cannot be estimated without making assumptions, and available evidence suggests that the assumptions underlying polychoric correlations are most reasonable, both in the general case (Lyons et al., 1997) and with respect to sexual orientation.

Most genetic variation is generated by multiple genes, each of modest effect, as required by the multifactorial model (Plomin, Owen, & McGuffin, 1994). Even if the evidence that a gene on the X chromosome affects sexual orientation (Hamer et al., 1993; Hu et al., 1995) is ultimately correct, polychoric correlations may provide reasonably accurate estimates. The X-linked gene appears to account for only a modest amount of variance in sexual orientation (Hu et al., 1995) and, thus, does not seriously violate the multifactorial assumption. Nor does the observation that sexual orientation is quite nonnormally distributed, in some cases even bimodal (Diamond, 1993), provide strong evidence against the multifactorial assumption. Multifactorial systems can generate nonnormal data due to either measurement biases (Grayson, 1987) or to nonlinear effects of genes and environments. The debate whether sexual orientation is dimensional or categorical (Gangestad, Bailey, & Martin, 1998; Haslam, 1997) is, for this reason, not especially germane to the validity of the multifactorial assumption. The multifactorial assumption can be falsified only by discovery of a gene or environment that is sufficiently large in effect to bias parameter estimates unduly. Such a gene or environment is unlikely to exist. Nevertheless, it is good to keep in mind that genetic and environmental parameter estimates require an assumption that cannot be directly tested.

Genetic and environmental structural equation models were fitted separately for men and women by maximum likelihood using the LISREL program (Jöreskog & Sörbom, 1993a) and using only the same-sex pairs. We thus examined the relative effects of additive genetic (A), shared environmental (C; i.e., between family or common environment), and nonshared environmental (E; i.e., within family or individual-specific environment, including measurement error) factors on the latent normal variable underlying variation in sexual orientation.

After estimating parameters, they were tested for significance. In order to test parameter estimates (i.e., A, C, and E) for significance, one compares the fit of the full model allowing estimation of all three parameters with that of models in which each parameter (or combination of parameters) of interest has been set to zero. Each model is associated with a chi-square value, which reflects its fit (the higher the chi-square, the worse the fit). More specifically, good-fitting models have implied correlation matrices that are similar to the obtained correlation matrices used to estimate parameters. Models can be compared using a likelihood ratio chi-square test. Confidence intervals of parameter estimates were computed using the method of successive approximation, by raising or lowering each parameter until a significant decrement in fit occurs; such confidence intervals are typically asymmetric (Neale & Miller, 1997).

Multivariate analyses.

Genetic and environmental model fitting is not only useful to understanding the causes of variation in single traits but can also be used to examine why multiple traits covary (Heath, Neale, Hewitt, Eaves, & Fulker, 1989; Neale & Cardon, 1992). Multivariate models use as their input not only the correlations between co-twins for the same variable, but also the correlations among variables within each twin (e.g., the first twin's sexual orientation and childhood gender nonconformity), and the correlation between different variables across co-twins (e.g., the first twin's sexual orientation with the second twin's childhood gender nonconformity). In this study, we fitted a common pathway model, which specifies that covariation among measured variables is due to a latent phenotypic factor. We hypothesized that the latent phenotypic factor represents the degree of sex-atypical sexual orientation, childhood gender nonconformity, and continuous gender identity due to early hormonal influences. Prior research supports a role for prenatal hormones in influencing sexual orientation (Ellis & Ames, 1987), aspects of childhood gender nonconformity (e.g., Berenbaum & Hines, 1992), and gender identity (Zucker et al., 1996). Furthermore, as we have noted, these variables tend to be associated, albeit imperfectly so. It is plausible, then, to hypothesize that this covariation reflects hormonal processes, although our genetic analyses cannot test this, and the validity of our analyses does not depend on the accuracy of this hypothesis.

Genetic and environmental influences on the latent phenotypic factor can be estimated from the twin correlations; they depend especially on the cross-twin, cross-trait correlations (e.g., the correlation between one twin's sexual orientation and the other twin's CGN). Furthermore, each variable may have genetic, shared environmental, or nonshared environmental influences specific to that variable. Estimation and inferential tests of the general and specific influences are performed as in the univariate case.

Testing the equal environments assumption.

A fundamental assumption of the classical twin method that we adopted is that the trait-relevant environments are no more similar for MZ than for DZ twins. There are some environmental respects in which MZ twins are typically more similar than DZ twins (e.g., they are more likely to have been dressed similarly as children), but it is an empirical question whether these are trait-relevant respects. If they are, then similarity for the putative environmental influences should predict trait similarity. We used four standard items of similar childhood experiences (whether twins shared the same room, had the same playmates, were dressed alike, and were in the same classes at school). We summed the items to create an index of similarity in childhood experiences. Coefficient alphas for the composite ranged between .63 for male DZ twins to .69 for female MZ twins. Correlations between co-twins' composites ranged from .49 for male DZ twins to .66 for female MZ twins, suggesting that the twins' memories were fairly reliable. Thus, we summed co-twins' composites to form an overall index of similar childhood experiences and used it in a subsequent analysis to determine whether similar childhood experiences could explain similarity in sexual orientation.

Results

Distribution and Correlates of Sexual Orientation

The rate of exclusive heterosexuality (i.e., Kinsey scores of 0) was quite similar for men (91.8%; n = 1,683) and women (91.9%; n = 2,704). The distribution of nonheterosexual scores (i.e., Kinsey scores exceeding 0) differed markedly between the sexes, however (Figure 1). Women were more likely than men to have slight to moderate homosexual feelings (scores of 1–3), and men were more likely to be nearly exclusively homosexual (scores of 5–6). The distribution of scores differed significantly between the sexes, $[chi]^2(6, N = 4,387) = 71.7$, $p < .001$. The mean Kinsey score did not, however, differ by zygosity within sex. Nor was this variable correlated with age.

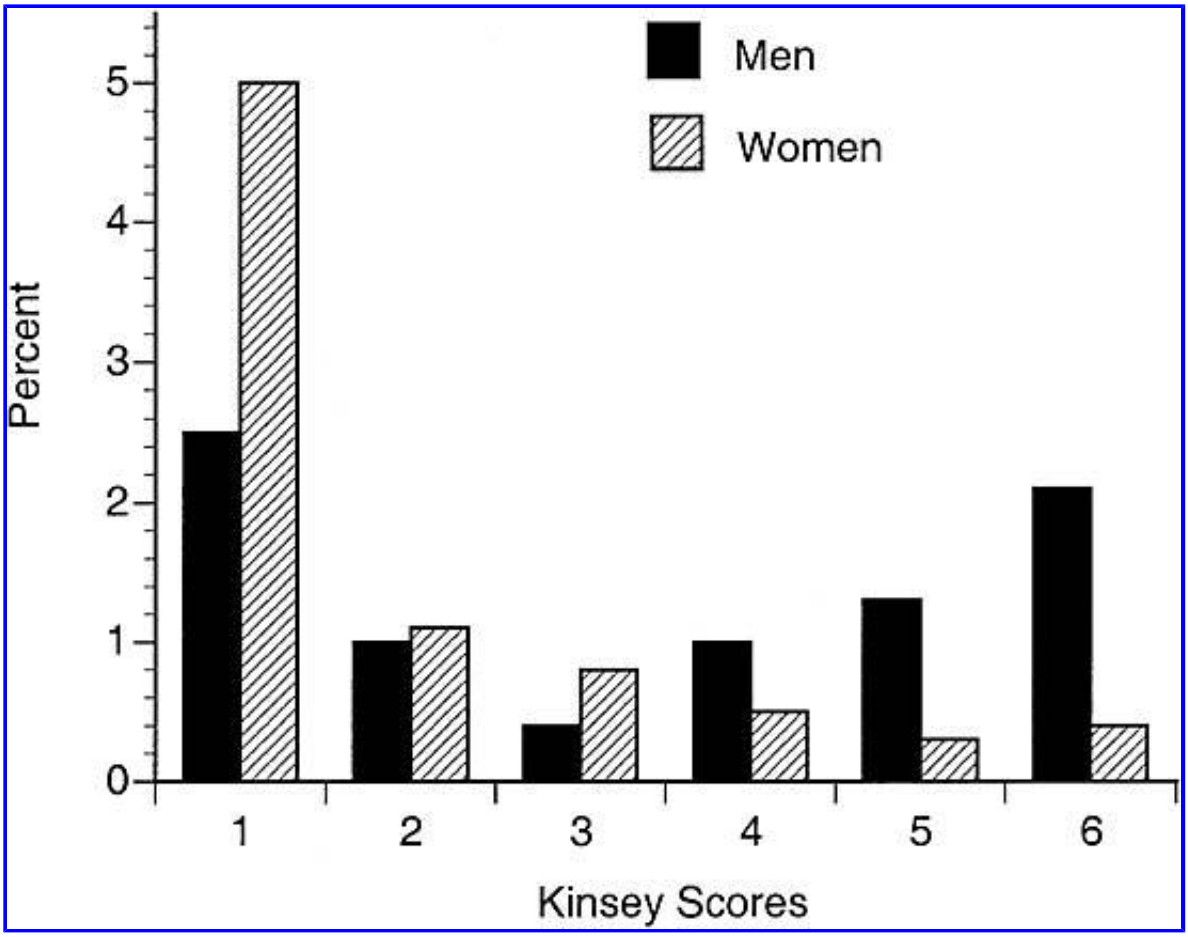

Figure 1. Distribution of Kinsey scores for all men (*n* = 147) and women (*n* = 238) with nonzero Kinsey scores (i.e., who were not exclusively heterosexual), as their percentage of the entire within-sex sample: 1 = *heterosexual with slight homosexual feelings;* 2 = *heterosexual with substantial homosexual feelings;* 3 = *equally heterosexual and homosexual;* 4 = *homosexual with substantial heterosexual feelings;* 5 = *homosexual with slight heterosexual feelings;* 6 = *completely homosexual*.

We examined whether the variables of hypothesized relevance to sexual orientation, CGN and CGI, were in fact associated with it. Furthermore, we examined whether Kinsey scores of 1 were similar to other nonheterosexual (i.e., nonzero) Kinsey scores with respect to these correlates, or whether they were indistinguishable in that sense from strictly heterosexual scores (i.e., Kinsey scores of zero). This was important because conventionally, a score of 2 is considered the lower bound of the bisexual range of the Kinsey Scale, and scores of 1 are often combined with scores of 0 as heterosexual (see, e.g., Pillard & Weinrich, 1986). Moreover, because scores of 1 are the most common nonzero score, particularly for women (Figure 1), their inclusion with the higher nonzero scores as nonheterosexual could substantially boost statistical power.

Figure 2 represents the comparison of both CGN and CGI between participants with strictly heterosexual Kinsey scores (i.e., 0) and those with either strongly homosexual scores (5–6), bisexual scores (2–4), or scores of 1. Effect sizes for these comparisons ranged from moderate (d = .5) to large (d > 1.5); all were statistically significant. Thus, the two measures appear to be valid correlates of sexual orientation, and Kinsey scores of 1 demonstrate similar (if somewhat smaller) associations with them as the higher scores do. Figure 3 (which represents actual percentages, rather than effect sizes, and thus includes Kinsey scores of 0) shows a similar pattern with respect to two indexes of homosexual experience. Participants with Kinsey scores of 1 were substantially, and significantly, more likely than those with Kinsey scores of 0 to have had at least one same-sex partner, and to have had at least two same-sex partners.

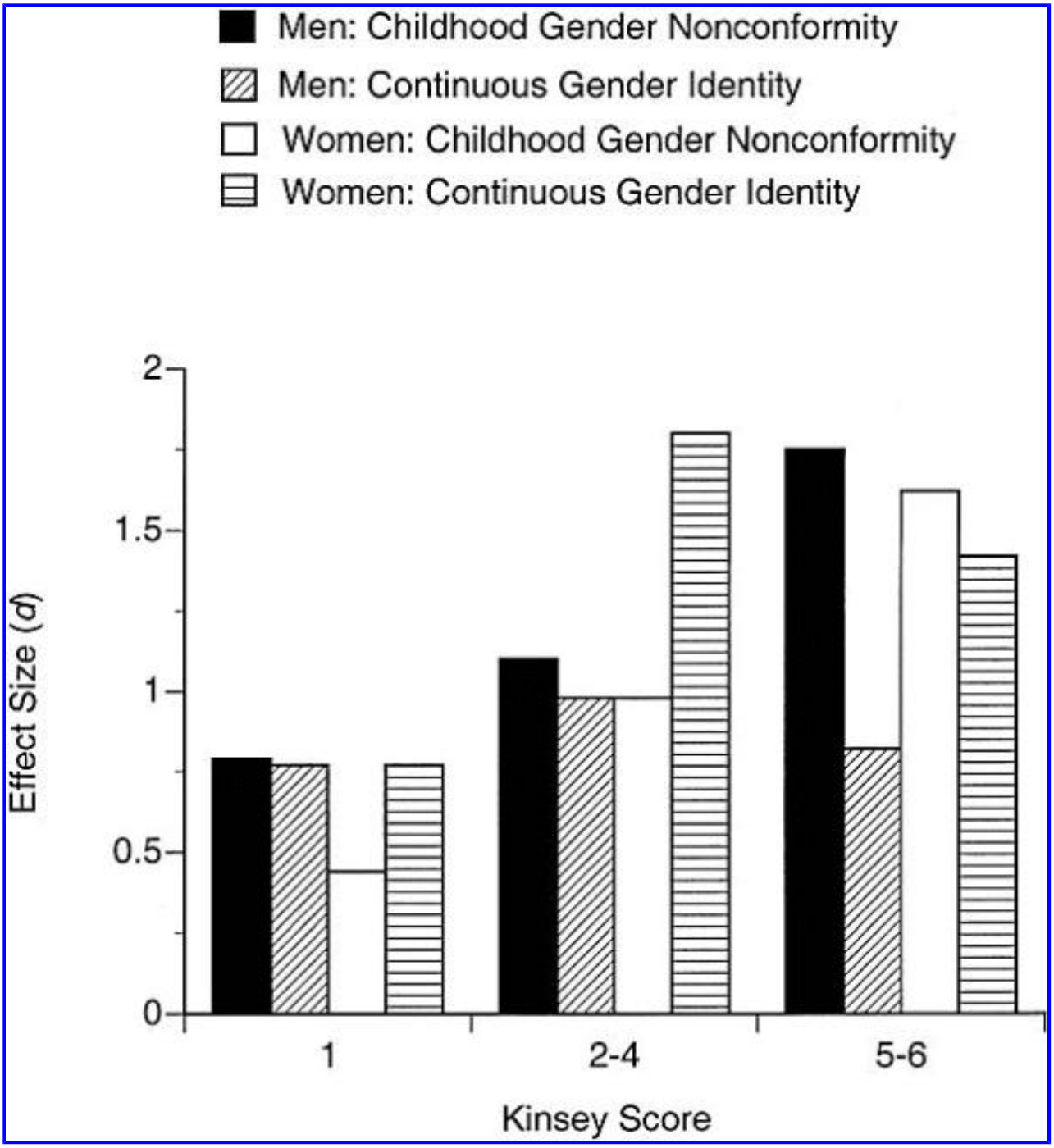

Figure 2. Correlates of sexual orientation measure: Childhood Gender Nonconformity and Continuous Gender Identity. Effect sizes represent the comparison of each subgroup with strictly heterosexual participants (i.e., Kinsey scores of 0), and were calculated as *d* (Cohen, 1987).

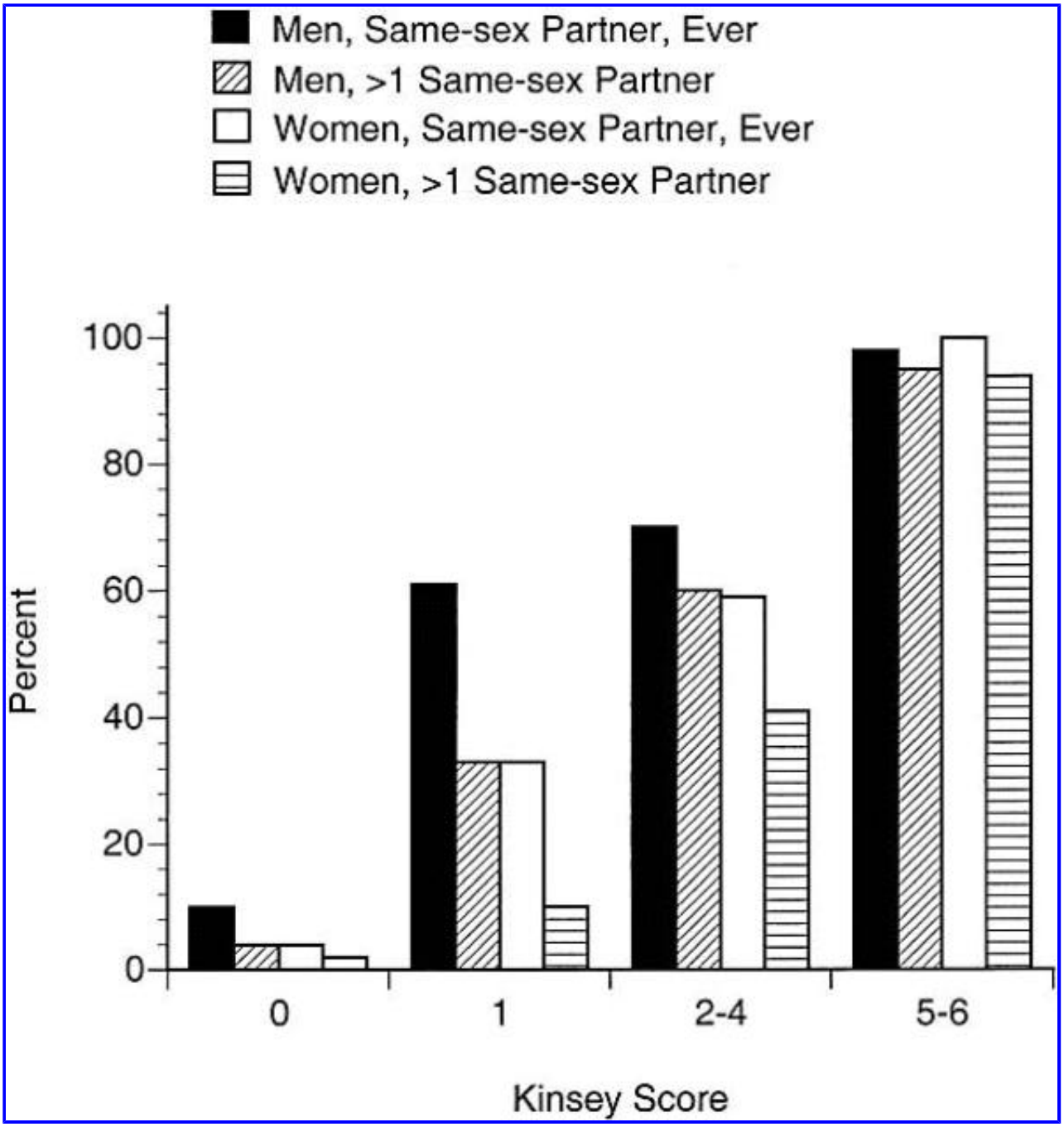

Figure 3. Correlates of sexual orientation measure: any homosexual experience and repeated homosexual experience.

We addressed one final issue related to construct validity of our CGN measure. The validity of the association between similar retrospective measures and sexual orientation has often been questioned (e.g., Baumrind, 1995; Ross, 1980) on the grounds that homosexual people may be especially prone to exaggerate (or heterosexual people to understate) their childhood gender nonconformity. We collected data with the potential to illuminate this issue. Participants were asked to rate their siblings' (including their twins') sexual orientations as “heterosexual,” “bisexual,” or “homosexual.” Participants were also asked to rate their own sexual orientations on the trichotomous scale. A number of twins (14 men and 14 women) were apparently unaware that their co-twins did not consider themselves to be heterosexual. (In these cases, the co-twins were “closeted” with respect to the twins.) In these cases, would they recall their nonheterosexual co-twins

as having been gender nonconforming children compared with themselves? We specifically tested the difference in the Co-Twin Comparative CGN Scale between twins whose nonheterosexual co-twins were closeted and those who accurately assessed their co-twins as heterosexual. Male participants recalled their closeted nonheterosexual co-twins as significantly more feminine than their heterosexual co-twins, $d = 0.7$, $t(448) = 2.4$, $p < .05$. The analogous comparison for women was not significant, $d = 0.3$, $t(952) = 1.4$, though it was in the predicted direction. Thus, male twins who did not know that their male co-twins considered themselves gay or bisexual still recalled them as having been more feminine than themselves.

Twin Similarity

Table 1 provides the probandwise concordances for same-sex twin pairs, using two criteria for nonheterosexuality. For opposite-sex pairs, only pairwise concordances were appropriate (because of markedly different distributions of sexual orientation, using the strict criteria). The stricter criterion was a Kinsey score of at least 2, and the more lenient criterion was a score of at least 1. Men's same-sex concordance rate for MZ twins was significantly greater than that for same-sex DZ twins by a directional test, both for the strict criterion (Fisher's exact $p = .04$) and the lenient criterion ($p < .001$). Women's same-sex concordance rates did not differ significantly by zygosity, for either definition.

Table 1
Probandwise Concordances for Two Criteria of Nonheterosexual Orientation

Proband's sex and criterion	Monozygotic				Same-sex dizygotic				Opposite-sex dizygotic			
	n pairs			Probandwise concordance	*n* pairs			Probandwise concordance	*n* pairs			Probandwise concordance
	++	+−	−−		++	+−	−−		++	+−	−−	
Male												
Strict (Kinsey ≥ 2)	3	24	260	20.0	0	16	146	0.0	2	17	287	10.5
Lenient (Kinsey ≥ 1)	9	30	260	37.5	1	30	146	6.3	3	24	287	11.1
Female												
Strict (Kinsey ≥ 2)	3	19	539	24.0	1	17	293	10.5	2	9	287	18.2
Lenient (Kinsey ≥ 1)	14	65	539	30.1	8	37	293	30.2	3	23	287	11.5

Note. Figures for the strict criteria exclude pairs in which 1 twin had a Kinsey score of 1. For opposite-sex twins, probandwise concordances represent the rates of nonheterosexuality in the opposite-sex twins of the probands.

Table 1 Probandwise Concordances for Two Criteria of Nonheterosexual Orientation

To investigate the validity of the equal environments assumption with respect to sexual orientation, we compared our index of similar childhood experiences between concordant and discordant MZ twins, using the lenient criterion. (This analysis was restricted to MZ twins because there was an insufficient number of concordant DZ twin pairs for analysis and because for MZ but not for DZ twins, equal environments measures cannot reflect genetic similarity.) Consistent with the equal environments assumption, concordant pairs were not more similar in their childhood experiences than were discordant pairs. Indeed, for males, concordant pairs recalled significantly less similar childhood environments, $t(37) = 2.1$, $p < .05$; for females, $t(76) = 0.4$, $p > .05$. Thus, concordance in MZ pairs does not appear to have resulted from similarity of childhood experiences as we measured them.

Table 2 includes the polychoric correlations for sexual orientation, CGN, and CGI. MZ correlations were somewhat lower for CGI than for the other two variables, partly reflecting CGI's modest reliabilities. (This will also constrain CGI's genetic and shared environmental parameters in subsequent model fitting.) Among female same-sex pairs, MZ correlations exceeded DZ correlations, although the magnitude of the difference varied considerably. For the male same-sex pairs, the MZ correlations all exceeded twice the DZ

correlations, consistent with genetic dominance. Dominance variance is expected when a trait is related to fitness, as sexual orientation surely is. Twin methodology does not allow simultaneous estimation of shared environment and both dominance and additive genetic effects, because there are too many unknowns. In the following analyses, we estimated additive genetic and shared environmental effects, but the possibility of genetic dominance should not be forgotten.

Table 2
Twin Similarity for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity

Trait	Male MZ	Male DZ	Female MZ	Female DZ	Opposite-sex DZ
Sexual orientation	.51	−.11	.49	.45	.14
95% confidence interval	.22–.78	−.85–.27	.32–.66	.26–.71	−.81–.47
Childhood gender nonconformity	.54	.14	.42	.06	−.02
95% confidence interval	.45–.65	−.03–.30	.31–.49	−.13–.11	−.11–.12
Continuous gender identity	.32	.12	.32	.21	.06
95% confidence interval	.15–.43	−.07–.29	.19–.41	.03–.34	−.07–.21

Note. Figures represent polychoric correlations. MZ = monozygotic; DZ = dizygotic.

Table 2 Twin Similarity for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity

The correlations for opposite-sex DZ twins were all small and nonsignificant, consistent with the possibility that very different genetic and shared environmental factors affect these traits in the two sexes (i.e., that they are sex-limited; see Neale & Cardon, 1992). Because, given the pattern of DZ correlations (i.e., most were relatively small), we had little power to detect sex limitation, and because of the possibility that the traits have different causes between the sexes, we subsequently estimated genetic and environmental models separately for each sex, using only the same-sex pairs.

We examined the equal environments assumption for CGN and CGI by correlating our index of similar childhood experiences with the absolute intrapair difference of each variable. For men, these correlations were low and nonsignificant for both zygosities. In contrast, women did show some reliable, albeit small, correlations consistent with violation of the equal environments assumption. For MZ pairs, the relevant correlations were $r(628) = -.14$ and $r(615) = -.12$ for CGN and CGI, respectively, $p < .01$; and for DZ pairs, $r(353) = -.13$, $p < .05$, and $r(340) = -.02$, *ns*. The sign of the correlations indicated that more similar childhood experiences was associated with smaller intrapair trait differences.

Genetic and Environmental Model Fitting: Univariate Analyses

Table 3 contains the parameter estimates (and 95% confidence intervals) for the full (A, C, E) models. In these tables, the sexual orientation variable was a 3-point ordinal scale (Kinsey scores of 0, 1, and 2–6, respectively). With one exception, heritabilities (computed as A^2) were moderate and shared environmentalities (computed as C^2) were small. The exception, female sexual orientation, showed the reverse pattern. The confidence intervals for the heritabilities and shared environmentalities tended to be wide, reflecting in part the compensatory relation between C and A. That is, to an extent, as one diminishes, the other can increase to account for the data. The largest confidence intervals, for sexual orientation, also reflect that variable's unfavorable distribution. Polychoric correlations (and parameter estimates derived from them) are most precisely measured when thresholds are evenly spaced across the distribution; they become

much less so when a large fraction of the distribution is on one side of all the thresholds (Neale, Eaves, & Kendler, 1994), as is true of sexual orientation. CGN had (on the basis of visual inspection) the most favorable distribution, and for both men and women, heritability was substantial and highly significant.

Table 3
Parameter Estimates for Univariate Models

Trait	Men			Women		
	A^2	C^2	E^2	A^2	C^2	E^2
Sexual orientation						
Parameter estimate	.45	.00	.55*	.08	.41	.50*
95% confidence interval	.00–.71	.00–.41	.18–.85	.00–.67	.00–.64	.30–.69
Childhood gender nonconformity						
Parameter estimate	.50*	.00	.50*	.37*	.00	.64*
95% confidence interval	.28–.62	.00–.18	.35–.64	.25–.46	.00–.08	.52–.74
Continuous gender identity						
Parameter estimate	.31	.00	.69*	.24	.09	.67*
95% confidence interval	.00–.44	.00–.30	.53–.85	.00–.42	.00–.35	.56–.79

Note. Sexual orientation is the 3-point ordinal Kinsey variable (scores of 0, 1, and 2–6, respectively). Parameter abbreviations: A = additive genetic; C = shared environmental; E = nonshared environmental.
* $p < .05$.

Table 3 Parameter Estimates for Univariate Models

Because of the aforementioned tradeoff between A and C, it is useful to consider their joint effect, which can be conceptualized as familial influences. ("Familial influences" refers to those influences that cause traits to run in families, and should not be interpreted as all processes involving the family; some such processes cause family members to differ from one another.) The proportion of variance attributable to familial factors can be estimated using Table 2 as $1\text{-}E^2$, and it ranged from .31 for male AGI to .50 for male CGN. In order to test the significance of familial factors, we fitted a model for each variable and sex omitting both A and C. The difference in chi-square ($df = 2$) ranged between 13.3 (for male sexual orientation) and 74.9 (for male CGN), and in every case was significant ($p < .01$). Thus, familial factors were demonstrably important for all the traits, though it was more difficult to resolve the relative importance of additive genetic and shared environmental factors.

Genetic and Environmental Model Fitting: Multivariate Analyses

Tables 4 and 5 contain the correlation matrices analyzed in the multivariate models for men and women, respectively. Figure 4 depicts the common pathway model (for 1 twin) and gives parameter estimates for both sexes. For both men and women, the model chi-squares were significant, $[chi]^2(19, N = 494) = 43.9$, and $[chi]^2(19, N = 1{,}044) = 41.9$, respectively, $p < .01$, and thus the models could be rejected on formal grounds. However, with sufficient sample size, any interesting model will be rejected because all scientific models are approximations, and hence have error (Browne & Cudeck, 1993). The more important issue of statistical inference, then, concerns whether a given model's particular components are statistically necessary.

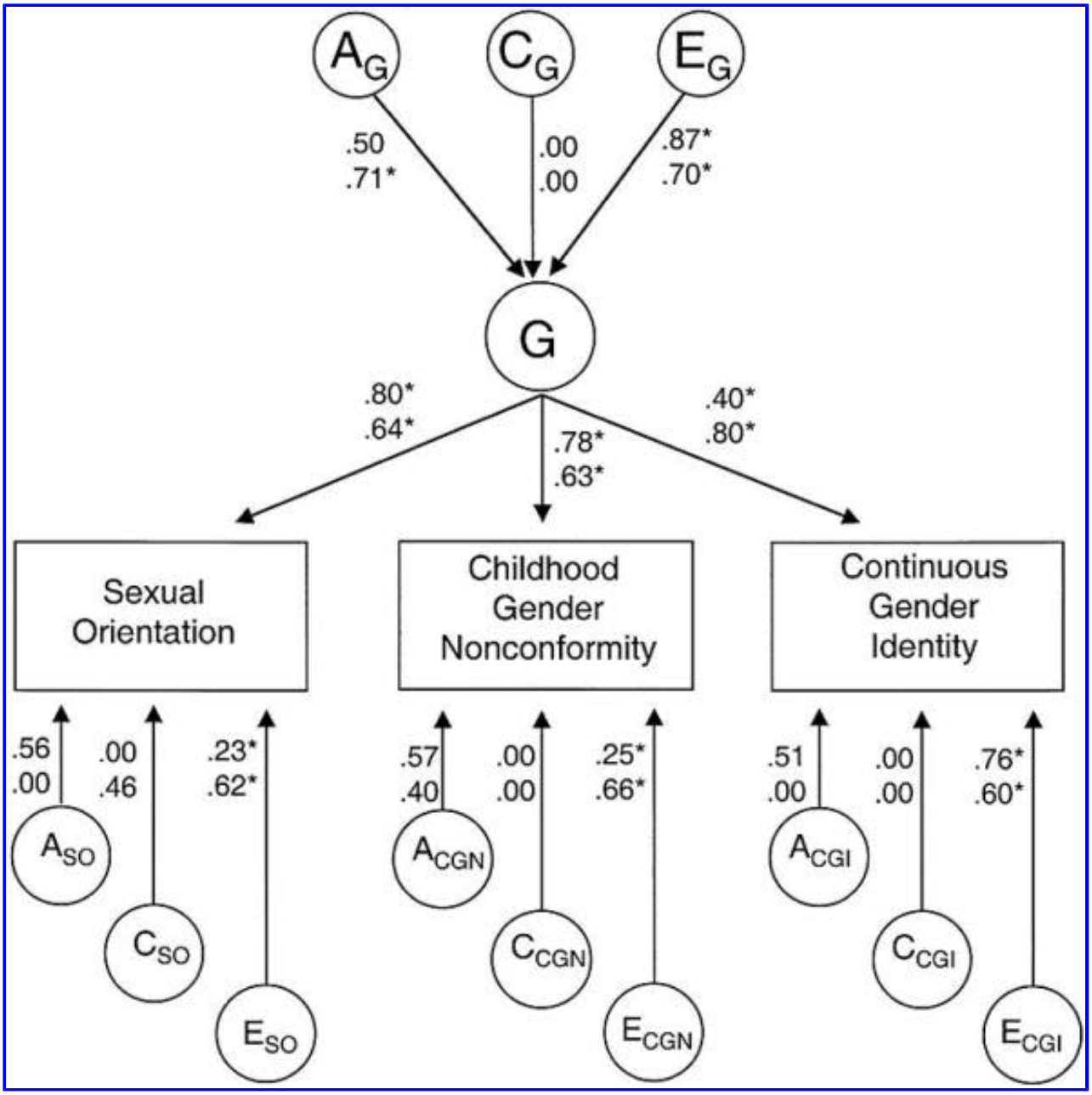

Figure 4. Common pathway model and parameter estimates (A = additive genetic; C = shared environmental; E = nonshared environmental). Parameter estimates for males are above respective estimates for females. SO = sexual orientation; CGN = childhood gender nonconformity; CGI = continuous gender identity. *$p < .05$.

Table 4
Within-Individual and Cross-Pair Correlations for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity: Men

Trait	1	2	3	4	5	6
1. Sexual orientation, Twin 1	—	.51	.54	.20	.36	.17
2. Sexual orientation, Twin 2	−.11	—	−.06	.50	.20	.31
3. Childhood gender nonconformity, Twin 1	.59	.13	—	.54	.26	.09
4. Childhood gender nonconformity, Twin 2	.03	.64	.14	—	.12	.36
5. Continuous gender identity, Twin 1	.25	−.35	.35	.14	—	.33
6. Continuous gender identity, Twin 2	−.05	.52	.14	.31	.12	—

Note. Correlations for monozygotic pairs are above the diagonal; dizygotic correlations are below the diagonal.

Table 4 Within-Individual and Cross-Pair Correlations for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity: Men

Table 5
Within-Individual and Cross-Pair Correlations for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity: Women

Trait	1	2	3	4	5	6
1. Sexual orientation, Twin 1	—	.49	.36	.17	.51	.37
2. Sexual orientation, Twin 2	.45	—	.28	.45	.34	.58
3. Childhood gender nonconformity, Twin 1	.14	.01	—	.42	.50	.21
4. Childhood gender nonconformity, Twin 2	−.07	.35	.02	—	.29	.49
5. Continuous gender identity, Twin 1	.38	.12	.47	.09	—	.32
6. Continuous gender identity, Twin 2	.10	.31	.09	.50	.21	—

Note. Correlations for monozygotic pairs are above the diagonal; dizygotic correlations are below the diagonal.

Table 5 Within-Individual and Cross-Pair Correlations for Sexual Orientation, Childhood Gender Nonconformity, and Continuous Gender Identity: Women

One basic issue is whether, in fact, the parameter estimates differ reliably between the sexes. Constraining parameters to be equal across both sexes dramatically worsened fit, difference $[chi]^2(15, N = 1{,}538) = 76.7$, $p < .001$. This indicates that either the phenotypic model (i.e., loadings on the latent phenotypic variable, G) or the genetic and environmental architecture, or both, differed between the sexes.

The loadings on the latent phenotypic variable, G, differed markedly between the sexes; constraining these to be equal across the sexes while allowing the genetic and environmental architecture to be estimated separately for each sex worsened fit considerably, difference $[chi]^2(3, N = 1{,}538) = 51.3$, $p < .001$. This difference primarily reflects the different pattern of correlations among variables within each sex. For women, all three measured variables were good indicators of G, with CGI especially so (factor loading of .80). For men, both sexual orientation and childhood gender nonconformity were good indicators of G, but CGI was much less so (factor loading of .40).

The sex difference in parameter estimates was not restricted to the phenotypic level. Allowing the sexes to differ in their loadings on G, but constraining the genetic and environmental architecture to be equal across the sexes worsened fit significantly, difference $[chi]^2(12, N = 1{,}538) = 55.9$, $p < .001$. Of particular interest

in the present case were the genetic and environmental influences on the latent variable, G. For both men and women, the shared environmental parameter was estimated as .00. In contrast, the additive genetic parameter was substantial for both sexes, but was significant only for women. For men, the decrement in fit when A_G was set to zero was only marginally significant, $[chi]^2(1, N = 494) = 3.1$, $p = .08$. For both men and women, setting A_G and C_G simultaneously to zero significantly worsened fit, $[chi]^2(2, N = 494) = 6.3$, $p < .05$, and $[chi]^2(2, N = 1,044) = 67.2$, $p < .001$, suggesting that familial factors are important influences on the covariation among the traits we studied. Nonshared environmental influences on G, E_G, were also substantial, especially for men.

Discussion

In a large representative sample of adult Australian twins, we found consistent evidence that familial factors influence sexual orientation and two related traits, childhood gender nonconformity and continuous gender identity. It was difficult, in general, to disentangle genetic and shared environmental contributions to the familial variance, though childhood gender nonconformity was significantly heritable in both sexes. Multivariate analyses showed that familial factors were important causes in the covariation among the three traits, and provided some support for genetic factors per se. The multivariate analyses, as well as examination of the distribution of sexual orientation, provided evidence that male and female sexual orientation should be analyzed separately and probably require different theoretical accounts. As is clear from the confidence intervals of the univariate parameter estimates, however, only fairly general statements about genetic and environmental influences can be made with confidence.

Distribution and Correlates of Sexual Orientation

Our finding that male and female sexual orientation were distributed quite differently, with higher scores representing homosexual orientations much more common in men, suggests that sexual orientation development is not symmetrical between the sexes. Something about male sexual orientation development makes extreme departures from heterosexual development especially likely compared with female sexual orientation development. Furthermore, evidence from our study suggests that sexual orientation, as we measured it, may have had somewhat different meanings for male and female participants. The correlation between sexual fantasy and sexual attraction was lower for women ($r = .67$) than for men ($r = .92$), for whom the two items appeared to be essentially equivalent. These phenotypic sex differences increase the a priori likelihood that the influences on male and female sexual orientation differ.

For both men and women, the most common nonzero Kinsey score was 1. Individuals with scores of 1 are certainly underrepresented in studies that ascertain participants through homophile publications or organizations, as most prior genetic studies and, indeed, most studies of sexual orientation have done. Our study yielded the most thorough existing analysis of the issue whether Kinsey scores of 1 should be differentiated from scores of 0 and thus considered nonheterosexual. Participants with scores of 1 were more atypical in their childhood behavior and gender identity, and they were more likely to have had homosexual experience, compared with those with scores of 0. This is consistent on a general level with the results of a recent study of women with congenital adrenal hyperplasia (CAH), a condition in which females are exposed to high levels of androgens prenatally. This condition has been hypothesized to be be a model for some biological influences on female sexual orientation, and if so, women with CAH should have elevated rates of bisexuality and homosexuality. Women with CAH were more likely than controls to have Kinsey scores of 1 (Zucker et al., 1996). Further research should elucidate the meaning and correlates of rare homosexual feelings. Such research might explore, for example, whether people with Kinsey scores of 1

show a different pattern of physiologically measured sexual arousal to male or female sexual stimuli compared with those with Kinsey scores of 0.

Genetic and Environmental Analyses

The most striking difference between our results and those of past twin studies of sexual orientation concerns the probandwise concordance rates. In a recent review the lowest concordances for single-sex MZ samples were 47% and 48%, for men and women, respectively (Bailey & Pillard, 1995). In contrast, our MZ concordances were 20% and 24%, respectively, for the strict criterion that is most similar to those used in prior studies. These rates are significantly lower than the respective rates for the two largest prior twin studies of sexual orientation: for men, 52% (Bailey & Pillard, 1991), $[chi]^2(1, N = 550) = 8.2$, $p < .01$, and for women, 48% (Bailey, Pillard, et al., 1993), $[chi]^2(1, N = 1{,}115) = 4.3$, $p < .05$. This suggests that concordances from prior studies were inflated because of concordance-dependent ascertainment bias (Kendler & Eaves, 1989). In those studies, twins deciding whether to participate in a study clearly related to homosexuality probably considered the sexual orientation of their co-twins before agreeing to participate. In contrast, both the more general focus of our study (i.e., on sexuality in general) and its anonymous response format made such considerations less likely. We are less confident of this explanation for women because prior studies had a preponderance of women with Kinsey scores of 5 and 6 and because there were relatively few such women in our sample (e.g., only 7 MZ twins in complete pairs had scores that high). It is conceivable that female homosexuality (indicated by Kinsey scores of 5 or 6) has a higher true probandwise concordance than does female bisexuality (indicated by Kinsey scores of 2–4). Larger studies will be necessary to determine if this is so.

Reviews of past twin studies of sexual orientation have questioned the plausibility of the equal environments assumption that the trait-relevant environment is no more similar for MZ than for DZ twins (Byne & Parsons, 1993; McGuire, 1995). Our results provided some support for this assumption. Concordant MZ pairs did not recall more similar parental treatment compared with discordant MZ pairs, and, indeed, for males the opposite pattern occurred. Although our limited set of items cannot provide a definitive test of the equal environments assumption (and because we do not know what the relevant environment for sexual orientation is, no definitive test is presently possible), they do suggest that increased MZ similarity is not due to the most obvious ways in which MZ twins are treated more similarly by their parents. Although there was some evidence that the equal environments assumption was violated for childhood gender nonconformity and continuous gender identity for women, the magnitude of this violation was not large, accounting for, at most, 2% of the variance.

Consistent with several studies of siblings (Bailey & Bell, 1993; Bailey & Benishay, 1993; Pillard, 1990; Pillard & Weinrich, 1986), we found that sexual orientation is familial. In contrast to most prior twin studies of sexual orientation, however, ours did not provide statistically significant support for the importance of genetic factors for that trait. This does not mean that our results support heritability estimates of zero, though our results do not exclude them either. Our findings are also consistent with moderate to large heritabilities for both male and female sexual orientation, and the confidence intervals of our estimates include estimates from earlier studies (Bailey & Pillard, 1991; Bailey, Pillard, et al., 1993; Buhrich, Bailey, & Martin, 1991). Our findings demonstrate the necessity of very large sample sizes to resolve familial variance into its genetic and shared environmental components, when one is studying traits with unfavorable distributions, such as sexual orientation.

In contrast, childhood gender nonconformity was significantly heritable for both men ($h^2 = .50$) and women ($h^2 = .37$). This provides some limited support for Bem's (1996a) hypothesis that childhood gender

nonconformity is the inherited component of sexual orientation development. Although as we have noted, childhood gender nonconformity was assessed retrospectively, with obvious attendant validity concerns, we provided some evidence for the validity of our scale. For example, co-twins' memories of their relative gender nonconformity were moderately related to each other and to differences on the CGN scale. This is consistent with past research, which has also supported the reliability and validity of such retrospective reports (Bailey, Miller, & Willerman, 1993; Bailey et al., 1995). In contrast, no study to date has identified clear biases in retrospective reports of childhood gender nonconformity. Finally, we note that we included a retrospective measure of childhood gender nonconformity because such measures have been associated robustly with adult sexual orientation (Bailey & Zucker, 1995). To the extent that we are interested in sexual orientation, then, it does not matter whether the childhood gender nonconformity measures are valid but rather that they are associated with sexual orientation. Our results suggested that they were strongly associated for males, with an average within-twin, cross-trait correlation of .57 (see Table 4). For females, this correlation was weaker, .33, but still moderate (see Table 5).

Multivariate models showed that familial factors also contribute to the covariation among sexual orientation, childhood gender nonconformity, and continuous gender identity. Furthermore, for both men and women, genetic factors were implicated (though for men, they were only marginally significant). Environmental factors were important, but appeared primarily to be of the nonshared variety. This is generally consistent with research on other personality (see Rowe, 1994, for a review).

Although we considered the multivariate model we tested to be highly plausible, others are also possible (Heath et al., 1989; Neale & Cardon, 1992). For example, Bem's (1996a) hypothesis that childhood gender nonconformity is causally related to homosexual orientation could, in principle, be tested by means of the comparison of alternative multivariate models, but in practice, this would require both very large samples and multiple indicators of both traits (Heath et al., 1993). It is important to acknowledge that the parameter estimates and associated significance tests depend on the validity of the underlying models, and there are more alternatives in the multivariate than in the univariate case.

Recent evidence that male sexual orientation is influenced by an X-linked gene (Hamer et al., 1993; Hu et al., 1995) has received widespread attention but remains controversial (Bailey, 1995). Our male MZ concordance figure suggests, however, that any major gene for strictly defined homosexuality has either low penetrance or low frequency.

Limitations and Future Research

We have already acknowledged the primary limitation of our study. Despite the large number of participants, there was an insufficient number of nonheterosexual (and especially homosexual) participants to guarantee a high degree of statistical power in the genetic and environmental analyses. For example, we had only about even odds of detecting significant heritability in our male sample, assuming the concordances we obtained using our strict definition. In order to increase power to .80, it would be necessary to double our sample size. Several twin registries exist that (either singly or, better, collectively) could ensure such power. In informal conversations with researchers associated with these registries, we have noted a great deal of hesitancy to ask registry twins to participate in studies of sexuality. We thus conclude with the observations that our participants generally indicated that they enjoyed completing the survey and that, to our knowledge, only 2 individuals from the original 9,112 approached (0.02%) withdrew from the registry because of the study.

References

Bailey, J. M. (1995). Sexual orientation revolution. *Nature Genetics, 11,* 353–354. [Medline Link] [Context Link]

Bailey, J. M., & Bell, A. P. (1993). Familiality of female and male homosexuality. *Behavior Genetics*, *23*, 313–322. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., & Benishay, D. S. (1993). Familial aggregation of female sexual orientation. *American Journal of Psychiatry*, *150*, 272–277. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., Finkel, E., Blackwelder, K., & Bailey, T. (1996). *Masculinity, femininity, and sexual orientation.* Unpublished manuscript. [Context Link]

Bailey, J. M., Miller, J. S., & Willerman, L. (1993). Maternally rated childhood gender nonconformity in homosexuals and heterosexuals. *Archives of Sexual Behavior*, *22*, 461–469. [Medline Link] [PsycINFO Link] [Context Link]

Bailey, M. J., Nothnagel, J., & Wolfe, M. (1995). Retrospectively measured individual differences in childhood sex-typed behavior among gay men: Correspondence between self- and maternal reports. *Archives of Sexual Behavior*, *24*, 613–622. [Medline Link] [PsycINFO Link] [Context Link]

Bailey, J. M., & Oberschneider, M. J. (1997). Sexual orientation and professional dance. *Archives of Sexual Behavior*, *26*, 433–444. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., & Pillard, R. C. (1991). A genetic study of male sexual orientation. *Archives of General Psychiatry*, *48*, 1089–1096. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., & Pillard, R. C. (1995). Genetics of human sexual orientation. *Annual Review of Sex Research*, *6*, 126–150. [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., Pillard, R. C., Dawood, K., Miller, M. B., Farrer, L. A., Trivedi, S., & Murphy, R. L. (1999). A family history study of male sexual orientation using three independent samples. *Behavior Genetics*, *29*, 79–86. [Medline Link] [PsycINFO Link] [Context Link]

Bailey, J. M., Pillard, R. C., Neale, M. C., & Agyei, Y. (1993). Heritable factors influence sexual orientation in women. *Archives of General Psychiatry*, *50*, 217–223. **[Fulltext Link]** [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Bailey, J. M., & Zucker, K. J. (1995). Childhood sex-typed behavior and sexual orientation: A conceptual analysis and quantitative review. *Developmental Psychology*, *31*, 43–55. **[Fulltext Link]** [PsycINFO Link] [Context Link]

Baker, L. A., Treloar, S. A., Reynolds, C., Heath, A. C., & Martin, N. G. (1996). Genetics of educational attainment in Australian twins: Sex differences and secular changes. *Behavior Genetics*, *26*, 89–102. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Baumrind, D. (1995). Research on sexual orientation: Research and social policy implications. *Developmental Psychology*, *31*, 130–136. **[Fulltext Link]** [PsycINFO Link] [Context Link]

Bell, A. P., Weinberg, M. S., & Hammersmith, S. K. (1981). *Sexual preference: Its development in men and women*. Bloomington, IN: Alfred C. Kinsey Institute of Sex Research. [Context Link]

Bem, D. J. (1996a). Exotic becomes erotic: A developmental theory of sexual orientation. *Psychological Review*, *103*, 320–335. **[Fulltext Link]** [PsycINFO Link] [Context Link]

Bem, D. (1996b). *Exotic becomes erotic: A political postscript*. Unpublished manuscript. [Context Link]

Berenbaum, S. A., & Hines, M. (1992). Early androgens are related to childhood sex-typed toy preferences. *Psychological Science*, *3*, 203–206. [PsycINFO Link] [Context Link]

Biggar, R. J., & Melbye, M. (1992). Responses to anonymous questionnaires concerning sexual behavior: A method to examine potential biases. *American Journal of Public Health*, *82*, 1506–1512. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Blanchard, R., & Freund, K. (1983). Measuring masculine gender identity in females. *Journal of Consulting and Clinical Psychology*, *51*, 205–214. [Medline Link] [PsycINFO Link] [Context Link]

Blanchard, R., McConkey, J. G., Roper, V., & Steiner, B. W. (1983). Measuring physical aggressiveness in heterosexual, homosexual, and transsexual males. *Archives of Sexual Behavior*, *12*, 511–524. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage. [Context Link]

Buhrich, N., Bailey, J. M., & Martin, N. G. (1991). Sexual orientation, sexual identity, and sex dimorphic behaviors in male twins. *Behavior Genetics*, *21*, 75–96. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Buss, D. M. (1994). *The evolution of desire: Strategies of human mating*. New York: HarperCollins.

Byne, W., & Parsons, B. (1993). Human sexual orientation: The biological theories reappraised. *Archives of General Psychiatry*, *50*, 228–239. **[Fulltext Link]** [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Cohen, J. (1987). *Statistical power analysis for the behavioral sciences*, (Rev. ed.). Hillsdale, NJ: Erlbaum. [Context Link]

Diamond, M. (1993). Homosexuality and bisexuality in different populations. *Archives of Sexual Behavior*, *22*, 291–310. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Duffy, D. L. (1994). *Asthma and allergic diseases in Australian twins and their families*. Unpublished doctoral dissertation, University of Queensland, Brisbane, Australia. [Context Link]

Dunne, M. P., Martin, N. G., Bailey, J. M., Heath, A. C., Bucholz, K. K., Madden, P. A. F., & Statham, D. J. (1997). Participation bias in a sexuality survey: Psychological and behavioural characteristics of responders and non-responders. *International Journal of Epidemiology*, *26*, 844–854. [Medline Link] [BIOSIS Previews Link] [Context Link]

Dunne, M. P., Martin, N. G., Pangan, T., & Heath, A. C. (1997). Personality and change in the frequency of religious observance. *Personality and Individual Differences*, *23*, 527–530. [Context Link]

Ellis, L., & Ames, M. A. (1987). Neurohormonal functioning and sexual orientation: A theory of homosexuality–heterosexuality. *Psychological Bulletin*, *10*, 233–258. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Falconer, D. S. (1989). *Introduction to quantitative genetics* (3rd ed.). New York: Longman. [Context Link]

Finn, S. E. (1987). *The structure of masculinity and femininity self ratings*. Unpublished manuscript. [Context Link]

Freund, K., Langevin, R., Satterberg, J., & Steiner, B. (1977). Extension of the Gender Identity Scale for males. *Archives of Sexual Behavior*, *6*, 507–519. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Gangestad, S. W., Bailey, J. M., & Martin, N. G. (1998). *Taxometric analyses of sexual orientation and gender identity*. Unpublished manuscript. [Context Link]

Gottesman, I. I. (1998, June). Twins: En route to QTLs for cognition. *Science*, *276*, 1522 [Context Link]

Gottesman, I. I., & Carey, G. (1983). Extracting meaning and direction from twin data. *Psychiatric Developments*, *1*, 35–50. [Medline Link] [Context Link]

Gottesman, I. I., & Shields, J. (1972). *Schizophrenia and genetics: A twin study vantage point*. New York: Academic Press. [Context Link]

Grayson, D. A. (1987). Can categorical and dimensional views of psychiatric illness be distinguished? *British Journal of Psychiatry*, *151*, 355–361. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Green, R. (1987). *The "sissy boy syndrome" and the development of homosexuality*. New Haven, CT: Yale University Press. [Context Link]

Greenberg, A. S., & Bailey, J. M. (1993). Do biological explanations of homosexuality have moral, legal, or policy implications? *Journal of Sex Research*, *30*, 245–251. [PsycINFO Link] [Context Link]

Grellert, E. A., Newcomb, M. D., & Bentler, P. M. (1982). Childhood play activities of male and female homosexuals and heterosexuals. *Archives of Sexual Behavior*, *11*, 451–478. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Hamer, D., & Copeland, P. (1994). *The science of desire: The search for the gay gene and the biology of behavior*. New York: Simon & Schuster. [Context Link]

Hamer, D. H., Hu, S., Magnuson, V. L., Hu, N., & Pattatucci, A. M. L. (1993, July). A linkage between DNA markers on the X chromosome and male sexual orientation. *Science*, *261*, 321–327. [Context Link]

Haslam, N. (1997). Evidence that male sexual orientation is a matter of degree. *Journal of Personality and Social Psychology*, *73*, 862–870. [Context Link]

Heath, A. C., Bucholz, K. K., Slutske, W. S., Madden, P. A., Dinwiddie, S. H., Dunne, M. P., Statham, D. J., Whitfield, J., Martin, N. G., & Eaves, L. J. (1994). The assessment of alcoholism in the general community: What are we measuring? Some insights from the Australian twin panel interview survey. *International Review of Psychiatry*, *6*, 295–307. [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Heath, A. C., Kessler, R. C., Neale, M. C., Hewitt, J. K., Eaves, L. J., & Kendler, K. S. (1993). Testing hypotheses about direction of causation using cross-sectional family data. *Behavior Genetics*, *23*, 29–50. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Heath, A. C., Neale, M. C., Hewitt, J. K., Eaves, L. J., & Fulker, D. W. (1989). Testing structural equation models for twin data using LISREL. *Behavior Genetics*, *19*, 9–36. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Hu, S., Pattatucci, A. M. L., Patterson, C., Li, L., Fulker, D. W., Cherny, S. S., Kruglyak, L., & Hamer, D. H. (1995). Linkage between sexual orientation and chromosome Xq28 in males but not in females. *Nature Genetics*, *11*, 248–256. [Medline Link] [BIOSIS Previews Link] [Context Link]

Jardine, R., & Martin, N. G. (1984). Causes of variation in drinking habits in a large twin sample. *Acta Geneticae Medicae Gemellologiae*, *33*, 435–450. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Jardine, R., Martin, N. G., & Henderson, A. S. (1984). Genetic covariation between neuroticism and the symptoms of anxiety and depression. *Genetic Epidemiology*, *1*, 89–107. [Medline Link] [BIOSIS Previews Link] [Context Link]

Johnson, A. M., Wadsworth, J., Elliott, P., Prior, L., Wallace, P., Blower, S., Webb, N. L., Heald, G. I., Miller, D. L., & Adler, M. W. (1989). A pilot study of sexual lifestyle in a random sample of the population of Great Britain. *AIDS*, *3*, 135–141. [Medline Link] [PsycINFO Link] [Context Link]

Jöreskog, K. G., & Sörbom, D. (1993a). LISREL 8 [Computer software]. Chicago: Scientific Software. [Context Link]

Jöreskog, K. G., & Sörbom, D. (1993b). PRELIS 2 [Computer software]. Chicago: Scientific Software. [Context Link]

Kasriel, J., & Eaves, L. J. (1976). A comparison of the accuracy of written questionnaires with blood-typing for diagnosing zygosity in twins. *Journal of Biosocial Science*, *8*, 263–266. [Medline Link] [BIOSIS Previews Link] [Context Link]

Kendler, K. S., & Eaves, L. J. (1989). The estimation of probandwise concordance in twins: The effect of unequal ascertainment. *Acta Geneticae Medicae et Gemellologiae, Twin Research*, *38*, 253–270. [Medline Link] [BIOSIS Previews Link] [Context Link]

Kinsey, A. C., Pomeroy, W. B., & Martin, C. E. (1948). *Sexual behavior in the human male*. Philadelphia and London: W. B. Saunders. [Context Link]

Laumann, E. O., Gagnon, J. H., Michael, R. T., & Michaels, S. (1994). *The social organization of sexuality: Sexual practices in the United States*. Chicago: The University of Chicago Press. [Context Link]

LeVay, S. (1991, September). A difference in hypothalamic structure between heterosexual and homosexual men. *Science*, *253*, 1034–1037. [Context Link]

LeVay, S. (1996). *Queer science: The use and abuse of research into homosexuality*. Cambridge, MA: MIT Press. [Context Link]

Lippa, R. A. (1998). Gender-related traits in gay men, lesbians, and heterosexual men and women: The virtual identity of homosexual–heterosexual diagnosticity and gender diagnosticity. *Unpublished manuscript.* [Context Link]

Lippa, R., & Hershberger, S. (1999). Genetic and environmental influences on individual differences in masculinity, femininity, and gender diagnosticity: Analyzing data from a classic twin study. *Journal of Personality*, *67*, 127–155. [Medline Link] [PsycINFO Link] [Context Link]

Lykken, D. T., McGue, M., & Tellegen, A. (1987). Recruitment bias in twin research: The rule of two-thirds reconsidered. *Behavior Genetics*, *17*, 343–362. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Lyons, M. J., Faraone, S. V., Godberg, J., Eaves, L. J., Meyer, J. M., & True, W. R. (1997). Another view on the “right” statistical measure of twin concordance. *Archives of General Psychiatry*, *54*, 1126–1128. **[Fulltext Link]** [Medline Link] [PsycINFO Link] [Context Link]

Martin, N. G., Eaves, L. J., Heath, A. C., Jardine, R., Feingold, L. M., & Eysenck, H. J. (1986). Transmission of social attitudes. *Proceedings of the National Academy of Sciences*, *83*, 4364–4368. [Medline Link] [BIOSIS Previews Link] [Context Link]

Martin, N. G., & Martin, P. G. (1975). The inheritance of scholastic abilities in a sample of twins: I. Ascertainment of the sample and diagnosis of zygosity. *Annals of Human Genetics*, *39*, 213–218. [Medline Link] [BIOSIS Previews Link] [Context Link]

McGue, M. (1992). When assessing twin concordance, use the probandwise not the pairwise rate. *Schizophrenia Bulletin*, *18*, 171–176. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

McGuire, T. R. (1995). Is homosexuality genetic? A critical review and some suggestions. *Journal of Homosexuality*, *28*, 115–145. [Medline Link] [PsycINFO Link] [Context Link]

Meyer-Bahlburg, H. F. L. (1987). Psychoendocrine Research and the Societal Status of Homosexuals: A Reply to DeCecco. *Journal of Sex Research*, *23*, 114–120. [PsycINFO Link] [Context Link]

Mitchell, J., & Zucker, K. J. (1992, June). *The Recalled Childhood Gender Behaviors Questionnaire: Psychometric properties*. Paper presented at the International Academy of Sex Research, Barrie, Ontario, Canada. [Context Link]

Money, J. (1988). *Gay, straight, and in-between*. New York: Oxford University Press. [Context Link]

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families*. Dordrecht, the Netherlands: Kluwer. [Context Link]

Neale, M. C., Eaves, L. J., & Kendler, K. S. (1994). The power of the classical twin study to resolve variation in threshold traits. *Behavior Genetics*, *24*, 239–258. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Neale, M. C., & Miller, M. B. (1997). The use of likelihood-based confidence intervals in genetic models. *Behavior Genetics*. [Context Link]

Pattatucci, A. M. L., & Hamer, D. H. (1995). Development and familiality of sexual orientation in females. *Behavior Genetics*, *25*, 407–420. [Medline Link] [PsycINFO Link] [Context Link]

Pillard, R. C. (1990). The Kinsey Scale: Is it familial? In D. P. McWhirter, S. A. Sanders, & Reinisch, J. M. (Eds.), *Homosexuality/heterosexuality: Concepts of sexual orientation. The Kinsey Institute series* (Vol. 2, pp. 88–100). New York: Oxford University Press. [Context Link]

Pillard, R. C. (1991). Masculinity and femininity in homosexuality: “Inversion” revisited. In J. C. Gonsiorek & J. D. Weinrich (Eds.), *Homosexuality: Research implications for public policy* (pp. 32–43). Newbury Park, CA: Sage. [Context Link]

Pillard, R. C., & Weinrich, J. D. (1986). Evidence of familial nature of male homosexuality. *Archives of General Psychiatry*, *43*, 808–812. [Medline Link] [PsycINFO Link] [Context Link]

Plomin, R., Owen, M. J., & McGuffin, P. (1994, June). The genetic basis of complex human behaviors. *Science*, *264*, 1733–1739. [Context Link]

Reich, T., Cloninger, C. R., & Guze, S. B. (1975). The multifactorial model of disease transmission: I. Description of the model and its use in psychiatry. *British Journal of Psychiatry*, *127*, 1–10. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Rice, G., Anderson, C., & Ebers, G. (1995, September). *Male homosexuality: Absence of linkage to microsatellite markers at Xq28*. Paper presented at the Twenty-First Annual Meeting of the International Academy of Sex Research, Provincetown, MA. [Context Link]

Ross, M. W. (1980). Retrospective distortion in homosexual research. *Archives of Sexual Behavior*, *9*, 523–531. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Rowe, D. (1994). *The limits of family influence: Genes, experience, and behavior*. New York: Guilford Press. [Context Link]

Schmalz, J. (1993, March 5). Poll finds an even split on homosexuality's cause. *New York Times*, p. 11 [Context Link]

Stein, E. (1994). The relevance of scientific research about sexual orientation to lesbian and gay rights. *Journal of Homosexuality*, *27*, 269–308. [Medline Link] [PsycINFO Link] [Context Link]

Sundet, J. M., Magnus, P., Kvalem, I. L., Samuelsen, S. O., & Bakketeig, L. S. (1992). Secular trends and sociodemographic regularities in coital debut age in Norway. *Archives of Sexual Behavior*, *21*, 241–252. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Zucker, K. J., & Bradley, S. J. (1995). *Gender identity disorder and psychosexual problems in children and adolescents* (pp. 4–5). New York: Guilford Press. [Context Link]

Zucker, K. J., Bradley, S. J., Oliver, G., Blake, J., Fleming, S., & Hood, J. (1996). Psychosexual development of women with congenital adrenal hyperplasia. *Hormones & Behavior*, *30*, 300–318. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Zuger, B. (1988). Is early effeminate behavior in boys early homosexuality? *Comprehensive Psychiatry*, *29*, 509–519. [Medline Link] [PsycINFO Link] [BIOSIS Previews Link] [Context Link]

Accession Number: 00005205-200003000-00009
