

The Heritability of Gender Identity Disorder in a Child and Adolescent Twin Sample

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The heritability and prevalence of the gender identity disorder (GID) was examined, as well as its comorbidity with separation anxiety and depression, in a nonretrospective study of child and adolescent twins. The parents of 314 twins (ages 4–17 years; 96 monozygotic pairs [MZ] and 61 dizygotic [DZ] pairs) completed the Coolidge Personality and Neuropsychological Inventory (CPNI) containing a six-item *DSM-IV*-based GID scale. Prevalence of clinically significant GID symptomatology in the twin sample was estimated to be 2.3%. Univariate model fitting analyses were conducted using an ordinal transformation of the GID scale. The model that best described the data included a significant additive genetic component accounting for 62% of the variance and a nonshared environmental component accounting for the remaining 38% of the variance. Results suggested no heterogeneity in the parameter estimates resulting from age. The correlation between GID and depression was modest, but significant ($r = .20$; $P < .05$), whereas the correlation between GID and separation anxiety was nonsignificant ($P > .05$). Overall, the results support the hypothesis that there is a strong heritable component to GID. The findings may also imply that gender identity may be much less a matter of choice and much more a matter of biology.

KEY WORDS: Gender identity disorder; child and adolescent twins; heritability; nonretrospective design.

INTRODUCTION

Gender identity refers to one's sense of self or conviction in being male or female. Sexual orientation refers to one's degree of sexual attraction to males or females. A gender identity disorder (GID), as defined by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*; American Psychiatric Association, 1994), consists of two components: (1) a strong and persistent cross-gender identification and (2) persistent discomfort with one's biological sex or gender role behavior associated with one's sex. Heritability studies related

to gender issues, employing twin and family prevalence studies, more often have investigated genetic influences upon sexual orientation.

A review of heritability studies of sexual orientation reveals a fairly consistent finding that male homosexuality appears to be familial, is not controlled by a single major gene, is probably not X-linked, but has a genetic component (e.g., Bailey and Bell, 1993; Bailey *et al.*, 1999; Whitam *et al.*, 1993). There are fewer heritability studies of female homosexuals. A review of the latter research reveals somewhat the same pattern as for male homosexuals. It appears that female homosexuality has an appreciable genetic basis (e.g., Bailey, *et al.*, 1993), but there appears to be some evidence that the genetic etiological mechanisms for homosexuality may be different for the two genders. One of the important differences lies in the prevalence rates between the two genders for homosexuality. Female homosexuality is generally considered more rare than male homosexuality (e.g., Bailey

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et al., 1993; Gebhard, 1972). It also appears that the prevalence of female homosexuality among the relatives of homosexual females is much greater than the prevalence of male homosexuality among the relatives of homosexual males. Both of these lines of evidence suggest different mechanisms contributing to male and female sexual orientation, but the data is far from convincing.

As noted earlier, there are far fewer twin and familial studies of GID compared to studies of homosexuality. One recent retrospective study (Bailey *et al.*, 2000) reported on the heritability of childhood gender nonconformity in 1,891 adult twins (median age 29 years). They found a significantly heritable pattern for childhood gender nonconformity for both men and women, although the heritability estimates (univariate and multivariate) were stronger in men (males .50-.57; females .37-.40). The authors concluded that their findings provided some support for a hypothesis by Bem (1996) that childhood gender nonconformity is the heritable component of adult sexual orientation. However, Bailey *et al.* not only found different patterns of heritability for adult sexual orientation and for adult gender identity than they did for childhood gender nonconformity, but also varying patterns between men and women. The latter findings may alternatively suggest that childhood gender nonconformity does not have a simple predictive relationship with adult sexual orientation for both genders.

The controversy continues over the issue of the relationship of a childhood GID diagnosis with an adult homosexual orientation. In a study by Green (1987), 75% of boys who may have met criteria for GID reported homosexual or bisexual fantasies by the age of 19. About 80% of the individuals in this sample who have had sexual experiences were classified as homosexual or bisexual. Di Ceglie (2000), in a review of two long-term follow-up studies of 160 children with GID, found that from about half to three-quarters of the children were homosexual or bisexual as adolescents or adults. However, adult retrospective studies of homosexual men and women show that most do not report a childhood history of GID (e.g., Bailey and Zucker, 1995). From these and other studies, Bradley and Zucker (1997) have argued that the relationship "between GID and homosexuality is not a perfect one and that GID is not simply the early manifestation of homosexuality . . ." (p. 875). Menvielle (1998) counter-argues that "A 75% homosexual outcome makes GID an unusually strong predictor . . . This means that there is a strong correspondence between GID behaviors and

adult homosexuality . . ." (p. 243). In summary, it appears that although GID may be a fair predictor of later adult homosexuality, most adult homosexual orientations cannot be explained by a childhood GID diagnosis. However, it is also possible that retrospective adult studies are flawed by such factors as memory, self-report, social desirability, etc., and perhaps underestimate the real childhood prevalence of GID in this population.

DSM-IV (1994) notes that the prevalence of GID is unknown, but it has been considered to be a "very rare syndrome" (e.g., Coates and Wolfe, 1995) or "relatively rare" (Bradley and Zucker, 1997). In children clinically referred for GID, cross-gender interests and behavior typically onsets between 2 and 4 years, and a diagnosis of GID commonly occurs about school age (*DSM-IV*, 1994). Bradley and Zucker have noted that 6% of nonreferred 4- to 5-year-old boys and 12% of nonreferred 4- to 5-year-old girls sometimes or frequently behaved like the opposite sex (according to mothers' ratings). Furthermore, they noted that 1% of these boys and 5% of these girls sometimes or frequently wished to be of the opposite sex. In children referred for clinical evaluation, approximately 16% of 4- to 5-year-old boys behaved like the opposite sex and approximately 16% wished to be girls. Bradley and Zucker concluded that, although this data did not define rates of GID, cross-gender behaviors are not uncommon in children, and GID symptoms may not be uncommon in clinical populations. In their gender identity clinic, they also found a male-to-female sex ratio for GID symptoms of 1.4:1.

Studies of clinical samples of children have reported that there were five times as many boys for each girl diagnosed with GID (Zucker and Green, 1992). In adult clinical samples, GID appeared in men at about two to three times the rate of women. *DSM-IV* (1994) notes that referral bias might artificially increase the rate of GID in males, because there may be a greater stigma attached to cross-gender behavior for boys than for girls. In a study of GID familial patterns, Zucker *et al.* (1997) investigated the ratio of brothers to sisters for 444 boys with GID. They found an excess of brothers of male probands with GID and evidence that boys with GID are born relatively late among their brothers. They reported that this pattern may be reversed in girls, although the latter results were based on a small sample ($N = 22$). Psychosocial explanations for these results are equivocal: some suggest that children with GID may find it difficult to identify with more masculine older brothers, predisposing them to greater femi-

nity. However, other studies suggest that the presence of older brothers is associated with greater behavioral masculinity (Zucker *et al.*, 1997).

Biological explanations for sexual orientation have included an immunohormonal hypothesis (MacCulloch and Waddington, 1981) involving maternal antibodies to testosterone. Blanchard and Bogaert (1996) developed an alternative immunohormonal hypothesis involving the relationship between a mother's immune reaction and antibodies on the sexual differentiation of the fetal brain and a child's future gender identity and sexual orientation. Bradley and Zucker (1997) argued that most immunohormonal theories suggest that there are likely to be prenatal hormonal effects on sexual-dimorphic behavior. However, most studies of sexual chromosomes or hormonal abnormalities associated with GID have typically been negative (e.g., Coates and Wolfe, 1995).

Another issue in GID is the comorbidity with other disorders. Menvielle (1998) argues that comorbid disorders may more often be related to the stress experienced by the children with GID. Bradley and Zucker (1998) counter-argue that, whereas some psychological problems in children with GID are probably related to the stigma associated with the disorder, many of the problems are not simply reactive. Zucker and Bradley (1995) have noted that children with GID frequently have separation anxiety disorder, depression, and other behavioral problems. Coates and Person (1985) noted that 15 of 25 boys with GID met criteria for a diagnosis of separation anxiety disorder.

The study by Bailey *et al.* (2000) appears to be the first heritability study of childhood gender nonconformity in twins, although in a retrospective design. Prior speculations as to the heritability of GID appear to be based primarily upon genetic studies of adult sexual orientation. The purpose of the present study was to estimate heritability of GID in a sample of child and adolescent twins in a nonretrospective design. No twin studies to date have investigated the heritability of GID in a nonretrospective design. In addition, the present study attempted to estimate the prevalence of GID and the covariation of GID with symptoms of depression and separation anxiety. GID was measured in the present study by a six-item parent-as-respondent scale from the Coolidge Personality and Neuropsychological Inventory (CPNI; Coolidge, 1998) for children. There is substantial support from adult twin studies that heritability of many types of psychopathology may be appropriately estimated by the use of nonclinical samples (e.g., Jang *et al.*, 1996).

METHOD

Participants and Procedures

Participants consisted of parents of twins who were recruited through advertisements on the Internet and in local newspapers and through students in psychology classes at a midwestern university who earned extra credit by identifying parents of twins. Parents completed the CPNI on each child, as well as a demographic survey. Informed consent was obtained.

There were 314 twins, 96 MZ pairs (44 male pairs and 52 female pairs) and 61 DZ pairs (20 male pairs, 20 female pairs, and 21 male/female pairs). The mean age of the MZ pairs was 9.4 ($SD = 3.4$), and the mean age of the DZ pairs was 10.1 ($SD = 3.6$). The mean age of the parents was 39.5 years ($SD = 6.3$), and 85% of the parents had attained a level of education beyond high school. The mean maternal age at time of birth was 29.5 years ($SD = 5.3$), and ethnicity was as follows: MZ twins, Caucasian (83%), Hispanic (4%), Asian (6%), African American (2%), or other (5%); DZ twins, Caucasian (93%), Asian (2%), and other (5%). Conception followed the use of fertility drugs for 12% of the twins. Some parents reported having exposed the twins to potentially harmful substances prior to birth, such as alcohol (4%), tobacco (11%), injury (1%), serious illness (1%), and prescription drugs (11%).

Materials

The parents completed the 200-item, parent-as-respondent CPNI (Coolidge, 1998). The CPNI contains a six-item GID scale, based on the criteria in *DSM-IV* for GID. Each item is answered on a 4-point Likert-type scale ranging from (1) *strongly false*, (2) *more false than true*, (3) *more true than false*, to (4) *strongly true*. The CPNI is designed to be filled out by a primary caregiver who is intimately acquainted with the child's behavior. The CPNI normative sample consists of 329 children, ages 5 to 17 years. The internal scale reliability of the GID scale was .71, and the test-retest reliability was .78 (4-week test-retest interval). The CPNI manual states that a clinical diagnosis of GID may be warranted when a raw score sum is 15 or greater. A raw GID score of 15 results in a T score of about 92 (approximately 4 SDS above the normative mean), based on the CPNI normative sample and, thus, may be considered a conservative cutoff. The depression scale of the CPNI contains 14 items created from the *DSM-IV* criteria for major depressive disorder. The internal scale reliability is .81, and the test-retest

reliability of the scale is .89. The separation anxiety disorder scale of the CPNI contains 8 items from the *DSM-IV* criteria for separation anxiety disorder. The internal scale reliability is .83, and the test-retest reliability is .93. Validity studies support the use of the CPNI in a variety of clinical and community settings (e.g., Coolidge *et al.*, 1994; Coolidge *et al.*, 1990; Coolidge *et al.*, 1992; Coolidge *et al.*, 2001; Coolidge *et al.*, 2000; Friedman, 1998; Ellett, 2000; Snider, 2000).

Zygosity was determined by a 10-item questionnaire (see Cohen, *et al.*, 1975) that contained items regarding physical similarities (e.g., height, weight, hair and eye color) and confusion of the twins by parents, family, and strangers. The questionnaire has been demonstrated to be approximately 90% valid (compared to blood-typing).

In the present twin sample, the internal reliability of the GID scale was .84, the internal reliability of the separation anxiety disorder scale was .82, and the internal reliability of the depression scale was .80.

Twin Analyses

Because of the severely skewed distributional properties of the raw GID scale in this normative population, the raw scores were transformed in order to facilitate an ordinal analysis. Item scores, initially based on a 4-point Likert scale, were collapsed into binary categories representing either a positive (1) response or a negative (0) response. The six binary items were summed and collapsed into three ordered categories. The categories included "0," representing no symptoms endorsed, "1," representing minimal (1–3) symptoms endorsed, and "2," representing clinically significant (4 or more) symptoms endorsed. In ordinal data analysis, we assume that our ordered categories represent imprecise measurement of an underlying normal distribution of liability for the disorder, and that the liability distribution has one or more threshold (normal deviate) values that discriminate between categories (Neale and Cardon, 1992; Neale and Maes, 1999; Sham, 1998).

RESULTS

GID Scale Characteristics

Individual GID scores were first examined for complete data. Five cases were eliminated from the analyses because they were missing 50% or greater of the items on the GID scale. The remaining 309 twins ranged in age from 4 to 17 years, and GID raw scores tended to decrease with age ($r = -.19$, $P < .01$). Likewise,

gender differences in GID raw scores were significant, with females being rated significantly higher ($t(307) = 2.23$, $P < .05$). Before adjusting the data for these effects, the relative contributions of the individual GID items to a latent GID factor were evaluated using a standard principal components analysis, comparing males and females as well as younger (ages 4–10 years) and older (ages 11–17 years) subject groups. In each analysis, factor loadings on the first principal component were .49 or higher for each of the GID items (average loading = .75) suggesting that, despite differences in mean scores for males and females as well as for younger and older children, comparable constructs are being assessed in these groups. Cronbach's alpha coefficients were also high, ranging from .77 to .86 for the four groups, suggesting strong internal consistency among GID items, which varied minimally across gender and age.

Genetic Analyses

As previously described, raw scores were transformed into ordinal scales in order to facilitate the use of a univariate threshold model. To account for the gender differences in the prevalence of GID symptoms, thresholds were fixed separately for males and females. Twin pairs were also grouped into younger (ages 4–10) and older (ages 11–17) cohorts, to assess possible heterogeneity in our parameter estimates. Standard χ^2 difference tests were applied to compare the fit of the nested model. Both the ADE and ACE models were used as base models, and each fit the data equally well ($\chi^2_{\text{diff}} = 0.26$). As shown in Table I, the parameter estimates in the ACE model differed somewhat for the younger and older cohorts, suggesting that, in the older cohort, GID scores may be more influenced by shared environmental factors. The ADE model results supported the presence of dominance effects in the younger cohort only. However, a comparison of these base models with a more parsimonious model that estimated only additive genetic and nonshared environmental influences did not result in a poorer fit to the data (ACE vs. AE: $\Delta\chi^2_2 = 0.330$, $P = .85$; ADE vs. $\Delta\chi^2_2 = 0.070$, $P = .97$). A model that estimated only shared and nonshared environmental influences also fit the data (ACE vs. CE: $\Delta\chi^2_2 = 1.508$, $P = .47$), but less well than the AE model. Finally, models that constrained estimates across the older and younger cohorts were examined to test for heterogeneity related to age. The constrained CE model, which estimated a single parameter for shared environmental effects and a single parameter for nonshared environmental effects, did not result in a significant

Table I. Model Fitting Results

Age-group	Model	Variance components				-2LL ^a	df	$\Delta\chi^2$ (df)	P
		A	C	D	E				
Younger	ACE	.50	.00	—	.50	287.735	302	Base	
Older	ACE	.37	.36	—	.27				
Younger	ADE	.17	—	.35	.48	287.995	302	Base	
Older	ADE	.75	—	.00	.25				
Younger	AE	.50	—	—	.50	288.065	304	0.330 (2) ^b	.85
Older	AE	.75	—	—	.25			0.070 (2) ^c	.97
Younger	CE	—	.36	—	.64	289.243	304	1.508 (2)	.47
Older	CE	—	.68	—	.32				
Young-Old	CE	—	.50	—	.50	291.421	305	2.178 (1)	.14
Young-Old	AE	.62	—	—	.38	289.317	305	1.252 (1)	.26

^a Minus two times the log-likelihood of the GID data.

^b The AE model was compared to the ACE base model.

^c The AE model was compared to the ADE base model.

decrement in fit when compared to the CE model with separate estimates ($CE_{(Y/O)}$ vs. $CE_{(Y=O)}$; $\Delta\chi^2_1 = 2.178$, $P = .14$). Similarly, the constrained AE model did not result in a significant decrement in fit when compared to the full AE model ($AE_{(Y/O)}$ vs. $AE_{(Y=O)}$; $\Delta\chi^2_1 = 1.252$, $P = .26$), suggesting that age differences in heritability were nonsignificant. Based on parsimony and a comparison of log-likelihood fit statistics, the AE model, which estimates heritability at .62, is considered the best fitting model for these GID data.

Comorbidity of GID with Separation Anxiety and Depression

Ordinal scales were generated from item scores on the separation anxiety scale and the depression scale

(using the same method applied to the GID scores), resulting in three symptom levels: no symptoms, minimal symptoms, and clinically significant symptoms. Table II describes the percentage of children categorized for three symptom levels for GID, separation anxiety disorder (SAD), and depression (DEP). Parents reported their female children as having higher levels of GID than their male children in both the minimal and clinically significant categories. In contrast, males were rated as having more depressive symptoms than females. Scores for SAD were quite comparable for males and females. To estimate the rates of comorbidity among ordinal scores of GID, DEP, and SAD, polychoric correlations were estimated using Mx. The correlation between the GID and the DEP was significant ($r = .20$, $CI_{.95} = .01$ to $.37$), as was the correlation

Table II. Prevalence of Symptom Levels for Gender Identity Disorder (GID), Separation Anxiety Disorder (SAD), and Depression (DEP)

	Males			Females		
	No symptoms (%)	Minimal (%)	Clinically significant (%)	No symptoms (%)	Minimal (%)	Clinically significant (%)
GID	90.5	8.8	0.7	81.5	14.8	3.7
SAD	65.3	30.6	4.1	65.4	30.9	3.7
DEP	38.1	55.1	6.8	54.9	35.8	9.3

between SAD and DEP ($r = .48$, $CI_{(95)} = -.34$ to $.60$). However, the correlation between GID and SAD was nonsignificant ($r = .11$, $CI_{(95)} = -.10$ to $.31$).

DISCUSSION

The present study appears to be the first to estimate heritability of GID in a sample of child and adolescent twins in a nonretrospective design. Although a model including only shared and nonshared environmental effects could not be rejected, the best fitting model suggested that GID is highly heritable. Although GID appears to be somewhat more heritable in the older cohort compared to the younger cohort, this difference was not significant in our sample. This finding is similar to a recent study by Bailey *et al.*, (2000), who also found that childhood gender nonconformity was significantly heritable for adult twins in a retrospective design. The present findings are also similar to studies that have found appreciable heritable components of other personality traits and psychopathology in children and adolescents (e.g., Eaves *et al.*, 2000; Coolidge *et al.*, 2001; Coolidge *et al.*, 2000). The present findings also complement an adult twin study by Kirk *et al.*, (2000), who found a strong heritable basis for sexual orientation and homosexuality. Even if the size of the present heritability estimate may have been inflated somewhat by parental ratings as opposed to self-ratings (e.g., Eaves *et al.*, 2000), the estimate remains substantial and in the general range of a prior retrospective study of gender nonconformity by Bailey *et al.*, (2000).

A second purpose of the present study was to estimate the prevalence of GID symptomatology. Clearly, there was no external validity for high scores on the GID scale, thus any interpretation must be viewed with caution. Nevertheless, the current finding of 2.3% of the children who scored in the clinically significant range in this study would appear to counter claims that GID is a rare or very rare phenomenon. This rate is similar but lower than estimates of cross-gender behavior and gender nonconformity in a review article by Bradley and Zucker (1997). There was also a modest but significant relationship between gender and GID scores and between age and GID scores in the present study. Because of the relatively small number of twins in the present study, a determination of reliable prevalence rates by age was not feasible. Thus, to be conservative, the present prevalence estimate may be considered an upper limit of GID prevalence. A final note of caution: The ratio of boys to girls with GID symptomatology in the present study was 1:5. Bradley and

Zucker cite evidence that in nonreferred children, this ratio may range from about 1:2 to 1:3. However, in clinical samples, the rates range from about 7:1 to 1.4:1. Thus, the present estimate appears to have a greater similarity to nonreferred samples rather than clinical samples. It might still be argued, however (e.g., Bradley and Zucker, 1997), that cross-gender behaviors in girls are more acceptable to peers and adults than cross-gender in boys. Thus, girls may have a higher threshold for clinical referral. It appears clear from the present study and previous research that GID prevalence in future studies should be estimated separately by gender and in much larger samples.

A third purpose of the present study was to investigate the hypothesis that GID would be associated with higher rates of psychopathology, specifically SAD and DEP. Although the present study found a significant association between GID and DEP ($P < .05$), the relationship between GID and SAD scores was nonsignificant ($P > .05$); however, the strength of both relationships was weak. Future studies may also wish to address the issues raised by Bradley and Zucker (1997) and Menvielle (1998), who have argued the causal nature of the relationship between GID and associated psychopathology. This relationship is an important one as well as the issue of whether GID may be considered a precursor or prodromal stage of adult homosexuality. Certainly, both of these issues deserve further attention.

There are a few additional limitations of the present findings. The power in the present study was limited by the small sample size, although the power was sufficient to detect a heritability of the present magnitude (.62). However, because of the small sample size, we were unable to discriminate between competing models, which may be plausible and could not appropriately examine differential heritability in males and females. Nevertheless, a principal components analysis of the GID scale for gender and age did reveal a similar single factor structure and stable internal reliabilities. Future studies may wish to address the complexity of gender role development as a function of pre- and post-pubescence. Additional studies employing combinations of rater sources and clinical interviews with the parents and children are also needed. The parents in the present twin sample were also highly educated, and future studies may wish to ascertain whether less educated twin samples yield similar results. Overall, however, the central finding of this study cannot be lightly dismissed. It appears that the variation in GID has an appreciable heritable component, and the

implications of the latter finding in terms of intervention, therapy, and counseling appear to loom large.

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