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Sexual Orientation and Psychiatric Vulnerability: A Twin Study of Neuroticism and Psychoticism

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Abstract Recent evidence indicates that homosexuals and bisexuals are, on average, at greater risk for psychiatric problems than heterosexuals. It is assumed with some supporting evidence that prejudice often experienced by nonheterosexuals makes them more vulnerable to psychiatric disorder, but there has been no investigation of alternative explanations. Here we used Eysenck's Neuroticism and Psychoticism scales as markers for psychiatric vulnerability and compared heterosexuals with nonheterosexuals in a community-based sample of identical and nonidentical twins aged between 19 and 52 years ($N = 4904$). Firstly, we tested whether apparent sexual orientation differences in psychiatric vulnerability simply mirrored sex differences—for our traits, this would predict nonheterosexual males having elevated Neuroticism scores as females do, and nonheterosexual females having elevated Psychoticism scores as males do. Our results contradicted this idea, with nonheterosexual men *and* women scoring significantly higher on Neuroticism *and* Psychoticism than their heterosexual counterparts, suggesting an overall elevation of psychiatric risk in nonheterosexuals. Secondly, we used our genetically informative sample to assess the viability of explanations invoking a common cause of both nonheterosexuality and psychiatric vulnerability. We found significant genetic correlation between sexual orientation and both Neuroticism and Psychoticism, but no corresponding environmental correlations, suggesting that if there is a common cause of both nonheterosexuality and psychiatric vulnerability it is likely to have a genetic basis rather than an environmental basis.

Keywords Sexual orientation · Personality · Neuroticism · Psychoticism · Twins

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Introduction

Several recent large-scale studies have indicated that homosexuals and bisexuals (i.e., nonheterosexuals) are at much greater risk for psychiatric symptoms and disorders, including mood disorders (e.g., major depression, bipolar disorder), anxiety disorders (e.g., generalized anxiety disorder, phobic disorders, obsessive compulsive disorder), eating disorders, conduct disorder, substance misuse, suicidal ideation, and suicide attempts (Fergusson, Horwood, & Beautrais, 1999; King et al., 2008; Meyer, 2003; Mills et al., 2004; Sandfort, Bakker, Schellevis, & Vanwesenbeeck, 2006; Sandfort, de Graaf, Bijl, & Schnabel, 2001). For example, a recent meta-analysis revealed that, compared to heterosexuals, nonheterosexuals are at approximately twice the risk of depression and anxiety disorders, deliberate self harm and attempted suicide, and drug and alcohol dependence (King et al., 2008). With nonheterosexuals comprising up to a tenth of the adult population (Grulich, de Visser, Smith, Rissel, & Richters, 2003; Sell, Wells, & Wypij, 1995; Zietsch et al., 2008), it is obviously of great importance to public health to understand the causes of their elevated psychiatric vulnerability, but these causes remain unclear.

There is some evidence for the "minority stress" hypothesis, whereby social prejudice and discrimination provoke mental health problems in nonheterosexuals (Mays & Cochran, 2001; Meyer, 1995, 2003). Mays and Cochran (2001) found that controlling for reported levels of discrimination attenuated the relationship between sexual orientation and mental health. However, even after controlling for levels of discrimination there remained a large effect, and other studies show the relationship between sexual orientation and mental health is strong even in very liberal countries like The Netherlands (Sandfort et al., 2001, 2006), where prejudice against homosexuals is likely to be lower. Thus, it seems likely that other mechanisms (Bailey, 1999) may also contribute to the link between nonheterosexuality and psychiatric vulnerability, but these have not been investigated.

To investigate alternative hypotheses, we employed Eysenck's orthogonal personality scales Neuroticism and Psychoticism, putative measures of vulnerability to neuroses and psychoses, respectively (Claridge, 1981; Eysenck, 1967; Eysenck & Eysenck, 1976). Neuroticism has been shown to be a premorbid predictor of depression (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Ormel, Oldehinkel, & Vollebergh, 2004), accounts for comorbidity with anxiety (Andrews, 1996; Khan, Jacobson, Gardner, Prescott, & Kendler, 2005), and is also associated with eating disorders (Cassin & von Ranson, 2005). Similarly, Psychoticism is a premorbid predictor of schizophrenia and other psychotic disorders (Claridge, Robinson, & Birchall, 1983; Laurent, Gilvarry, Russell, & Murray, 2002) and is related to antisocial behavior and conduct problems (Cale, 2006; Miller & Lynam, 2001; Tranah, Harnett, & Yule, 1998). Both Neuroticism and Psychoticism are related to suicidal behavior (Nordstrom, Schalling, & Asberg, 1995). Thus, here we used Neuroticism and Psychoticism as markers for two broad types of psychiatric vulnerability.

It has been consistently found that women score higher on Neuroticism (Escorial & Navas, 2007), paralleling their greater vulnerability to depression/anxiety (Leach, Christensen, Windsor, Butterworth, & Mackinnon, 2008; Piccinelli & Wilkinson, 2000) and eating disorders (American Psychiatric Association, 1994), while men score higher on Psychoticism (Escorial & Navas, 2007), paralleling higher rates of antisocial and conduct disorders (Moffitt, Caspi, Rutter, & Silva, 2001). On many traits, nonheterosexual men tend to be more feminine and nonheterosexual women more masculine than their heterosexual counterparts (Miller, 2000; Pillard, 1991; Zietsch et al., 2008). Here, we tested whether sexual orientation differences in psychiatric vulnerability mirror sex differences. If true, we would expect observe higher Neuroticism scores in nonheterosexual males than heterosexual males, and higher Psychoticism scores in nonheterosexual females than heterosexual females. Lippa (2005) found that sexual orientation differences in Big Five personality traits tended to mirror sex differences, but some small studies on Eysenck's more clinically focused scales of Neuroticism and Psychoticism run counter to the "mirroring" effect. Bozkurt et al. (2006) found in a sample of Turkish men that homosexuals scored significantly higher on Psychoticism (as well as nonsignificantly higher on Neuroticism) than a matched heterosexual control group and Eisinger et al. (1972) found that homosexual women scored significantly higher than Neuroticism norms. In a small study, however, Wilson (1982) found that homosexual women scored *lower* than heterosexual women. As such, it is uncertain whether a mirroring effect exists for psychiatric vulnerability, but in any case, a simple mirroring of sex differences would not be a satisfactory explanation for the *overall* higher rates of psychiatric disorder observed in nonheterosexuals.

If, instead, both Neuroticism and Psychoticism scores are higher in nonheterosexual males *and* females, this would be more in line with the overall higher rates of psychiatric disorder in nonheterosexuals and would suggest a generalized elevation of psychiatric vulnerability in that group. Such an effect could be explained by the minority stress hypothesis if social prejudice causes nonheterosexuals to score higher on Neuroticism and Psychoticism. Alternatively, or in addition, it could be that genetic or environmental factors that predispose to nonheterosexuality also cause elevated levels of Neuroticism and Psychoticism. Without data on social prejudice, we could not directly test the minority stress hypothesis in the present study, but we used our genetically informative sample to assess the viability of alternative explanations invoking genetic and/or environmental influences common to both sexual orientation and psychiatric vulnerability.

Twin and family studies indicate that individual differences in sexual orientation, Neuroticism, and Psychoticism are partly ($\approx$40–50%) inherited (Birley et al., 2006; Keller, Coventry, Heath, & Martin, 2005; Kendler, Thornton, Gilman, & Kessler, 2000; Pillard & Bailey, 1998). If genetic variation underlying sexual orientation also influences Neuroticism and/or Psychoticism scores, then this genetic correlation could help to explain the observed greater psychiatric vulnerability in nonheterosexuals. A possible mechanism could involve genetic variation manifesting in structural differences in the brains of nonheterosexuals (Swaab, 2008), if those brain differences also affected psychiatric vulnerability. Similarly, environmental (non-genetic) factors that underlie sexual orientation could also influence psychiatric vulnerability. A possible example of such an environmental factor is childhood abuse, which is reported at much higher rates in nonheterosexuals than in the general population (Balsam, Rothblum, & Beauchaine, 2005; Corliss, Cochran, & Mays, 2002; Garcia, Adams, Friedman, & East, 2002; Hughes, Johnson, & Wilsnack, 2001; Tomeo, Templer, Anderson, & Kotler, 2001) and is also associated with a range of mental health issues as well as elevated Psychoticism and Neuroticism (Friedman, Marshal, Stall, Cheong, & Wright, 2008; Hughes

et al., 2001; Roy, 2002; Sun et al., 2008; Young, Harford, Kinder, & Savell, 2007).

In so far as Eysenck's Neuroticism and Psychoticism scales reflect psychiatric vulnerability, we investigated the above hypotheses using a sample of 4904 Australian twins who completed a survey on sexual behaviors and attitudes, including the revised EPQ shortened version. If sexual orientation differences in psychiatric vulnerability mirror sex differences, then we would expect higher Neuroticism and lower Psychoticism in nonheterosexual than heterosexual males, and vice versa for females. If both sexes exhibit elevated general vulnerability to mental illness in nonheterosexuals than heterosexuals, then Neuroticism and Psychoticism should be higher in both male and female nonheterosexuals than their heterosexual counterparts. If the latter case is true, then genetic analysis of the twin data should reveal whether elevated psychiatric vulnerability is due to a genetic correlation with sexual orientation, an environmental correlation, or both.

Method

Participants

The community-based sample consisted of 4904 Australian twins, both identical (monozygotic; MZ) and nonidentical (dizygotic; DZ), reared together. Of those, 107 were not used in genetic analysis due to missing zygosity (MZ/DZ) information. The final sample for genetic analysis was 4797, comprising 666 female MZ, 312 male MZ, 376 female DZ, 185 male DZ, 366 opposite-sex DZ pairs, and 987 single twins. Though data from single twins do not contribute directly to genetic analyses, they were included to increase the accuracy of sample statistics in the modeling.

Ages ranged from 19 to 52, with a mean (and *SD*) of 30.5 years (±8.3) for males and 31.1 years (±8.5) for females. In 1991–1992, participants anonymously completed a mailed questionnaire regarding their sexual behavior and attitudes, as well as personality and demographic information. Zygosity of the same-sex twins was pre-determined from an earlier study, based on twins' responses to standard items about physical similarity and being mistaken for each other. A more comprehensive description of the data collection procedure, the sample, and zygosity determination can be found in Kirk, Bailey, Dunne, and Martin (2000).

Measures

Sexual Orientation

Sexual orientation is thought to vary on a continuous spectrum (Kinsey, Pomeroy, & Martin, 1948). The measure we use to assess where individuals lie on this spectrum is Kinsey's attraction scale, where participants were asked to rate on a 7-point scale their sexual feelings ("Which of the following best describes your sexual feelings at present?") from exclusive attraction to the opposite sex (heterosexuality; Kinsey score 0), through degrees of attraction to both sexes (bisexuality; Kinsey scores 1–5), to exclusive attraction to the same sex (homosexuality; Kinsey score 6). Underlying this spectrum of sexual orientation, though, there appears to be a dichotomous factor with exclusive heterosexuals in one group and those with at least some homosexual tendencies in the other—a taxonometric analysis of our data suggested that up to 15% of men and 10% of women belong to the taxon associated with homosexuality (Gangestad, Bailey, & Martin, 2000). Note that we used sexual attraction as the indicator rather than sexual behavior or sexual identity, because we believe that attraction is less affected by mate availability and social/cultural constraints and is, in this sense, more fundamental (Sell et al., 1995).

We dichotomized scores on the Kinsey attraction scale to enable analyses which would be impossible with the raw scale due to low prevalences in Kinsey groups 1–6. As per Zietsch et al. (2008), we operationally defined those with any degree of sexual attraction to the same sex (Kinsey scores 1–6) as nonheterosexuals, and the associated trait as nonheterosexuality. In total, 11% of men and 13% of women in our sample fell into this category, in broad accordance with previous population estimates of the prevalence of nonheterosexual attraction (Sell et al., 1995). Those exclusively attracted to the opposite sex (Kinsey score 0) were defined as heterosexuals. Inspection of the mean Neuroticism and Psychoticism scores for each of the seven Kinsey groups supported the dichotomization of sexual orientation for the purpose of our analyses. Table 1 shows that Kinsey groups 1–6 (i.e., nonheterosexuals) had similar mean scores which were consistently higher than scores in Kinsey group 0 (i.e., heterosexuals). The one exception was that Kinsey group 4

Table 1 Mean Neuroticism and Psychoticism scores for Kinsey groups 0–6

Kinsey group	*N*	Neuroticism		Psychoticism	
		M	*SD*	*M*	*SD*
0	3916	5.07	3.40	1.98	1.67
1	340/344	5.97	3.35	2.65	1.89
2	82/81	6.35	3.41	2.73	1.99
3	41/43	5.95	3.89	2.67	2.02
4	25/26	4.56	3.12	2.38	2.23
5	30	5.93	3.37	2.70	1.91
6	54	5.69	3.61	2.08	1.98

Note: Sample sizes vary due to missing data. Absolute range for each scale, 0–12

had a slightly lower mean Neuroticism score than the heterosexuals, but it was also the smallest group ($n = 27$), so substantial sampling error could be expected.

Neuroticism and Psychoticism

We measured personality using the 48-item revised Eysenck Personality Questionnaire (EPQ-R) shortened version (Eysenck, Eysenck, & Barrett, 1985). For this study, we used the Neuroticism and Psychoticism scales, each consisting of 12 dichotomous (Yes/No) items each. Example Neuroticism items are "Are you a worrier?" and "Do you ever feel 'just miserable' for no reason?" Example Psychoticism items are "Would you take drugs which may have strange or dangerous effects?" and "Do you enjoy cooperating with others?" Both Neuroticism and Psychoticism are stable from age 12 to age 18 (Macaskill, Hopper, White, & Hill, 1994) and though there is a modest downward trend for both traits across the adult lifespan, individual differences are largely preserved (Loehlin & Martin, 2001). Stability in Neuroticism and Psychoticism is largely due to stability in their genetic influences (Loehlin & Martin, 2001; Macaskill et al., 1994).

Where only one personality item was missing, it was imputed using PRELIS (Joreskog & Sorbom, 1999), which substitutes with values from other cases with similar response patterns. This led to imputation of 0.13% of participant responses, and retained 3.2% of cases which would otherwise have missing data. Those cases still missing Neuroticism or Psychoticism items after imputation were treated as missing for that scale ($\approx$5% of cases for both Neuroticism and Psychoticism). The mean Neuroticism score was 4.40 ($\pm$3.39) for males and 5.65 ($\pm$3.34) for females. For Psychoticism, males averaged 2.59 ($\pm$1.85) and females 1.77 ($\pm$1.57). To be analyzed jointly with dichotomous sexual orientation, it was necessary to convert Neuroticism and Psychoticism into ordinal variables. Thus, for both variables we created six ordered categories of roughly equal frequencies (Neale, Eaves, & Kendler, 1994), which is optimal for threshold modeling in the statistical package Mx (Neale, Boker, Xie, & Maes, 2006).

Data Analysis

Maximum-likelihood modeling procedures were employed using the statistical package Mx (Neale et al., 2006), which accounts for the relatedness of twin pairs. Variables were analyzed as ordinal data, where it is assumed that thresholds delimiting the ordered categories overlay a normally distributed continuum of liability. In maximum-likelihood modeling, the goodness-of-fit of a model to the observed data was distributed as chi-square (χ^2), and the number of unknown parameters (those to be estimated) was reflected in the degrees of freedom (*df*). By testing the change in chi-square ($\Delta\chi^2$) against the change in degrees of freedom (Δdf), we tested whether dropping model parameters, or constraining them to be equal, significantly worsened the model fit. In this way, we tested hypotheses regarding those parameters.

Genetic Modeling

This study used the classical twin design, where variance in traits, and covariance between them, was partitioned into genetic (additive, A, and non-additive, D) and environmental (shared within twin pairs, C, and unshared, E) sources. This is possible when using a sample of twins because identical (monozygotic; MZ) twins share all their genes, while non-identical (dizygotic; DZ) twins share only half their genes on average. Thus, if A were the sole source of variance in a trait, we would expect a twin correlation of 1.0 for MZ pairs and 0.5 for DZ pairs. If D were the sole source of variance in a trait, we would expect a twin correlation of 1.0 for MZ pairs, and 0.25 for DZ pairs (for an explanation, see Posthuma et al., 2003). Twins reared together share many of their early environmental influences (C), including shared home environment, parental style, uterine environment, and so on. If C were the sole source of variance in a trait, we would expect a twin correlation of 1.0 for both MZ and DZ pairs. Twins also experience environmental influences which are unshared with their co-twin (E). If E were the sole source of variance in a trait, we would expect a twin correlation of zero for both MZ and DZ pairs. Variance due to measurement error also contributes to E.

In reality, observed MZ and DZ twin correlations reflect a combination of these genetic and environmental influences. Structural equation modeling allows us to determine the combination of these influences that best matches the observed data. A limitation of the design when only twin pairs reared together are used is that there is insufficient information to estimate both C and D for the same variable in the same model. For variables where the DZ twin correlation is more than half the MZ twin correlation, C is estimated, and for variables where the DZ twin correlation is less than half the MZ correlation, D is estimated. Cross-twin cross-trait correlations allow us to partition covariance between traits into A, C or D, and E in the same way as we do for variance in a single trait. An assumption of the classical twin design is that trait-relevant environments are equally similar for MZ and DZ twin pairs; tests of this assumption for personality traits (Loehlin, 1992) and homosexuality (Kendler et al., 2000) suggest it is valid. Further details of the classical twin design can be found elsewhere (Neale & Cardon, 1992; Posthuma et al., 2003).

To test for relationships between sexual orientation and the personality traits Neuroticism and Psychoticism, and whether the relationships could be explained by genetic or environmental correlation, we fitted two bivariate Cholesky

models: one with sexual orientation and Neuroticism, and the other with sexual orientation and Psychoticism. (As Neuroticism and Psychoticism were orthogonal (in our data, $r = -.06$ in males and $-.04$ in females), a computationally and conceptually more complex trivariate analysis would not be more informative than two bivariate analyses.) We tested for significant genetic and shared environmental influences by dropping them from each trait independently and comparing model fit. Similarly, we tested for significant genetic and environmental correlations between the variables by independently dropping the corresponding Cholesky cross-paths and comparing the fit. An alpha level of .005 was adopted as an approximate Bonferroni correction for the number of hypothesis tests performed. For ease of interpretation, the models were transformed from Cholesky forms into "correlated factors" models in Fig. 2 as suggested by Loehlin (1996).

Results

Heterosexuals Versus Nonheterosexuals on Neuroticism and Psychoticism

Figure 1 shows a comparison of the Neuroticism and Psychoticism scores of heterosexuals and nonheterosexuals. Independent sample *t*-tests on the raw personality data showed that nonheterosexual males had higher scores on Neuroticism, $t(1703) = 5.04, p < .001, d = .40$, and Psychoticism, $t(1716) = 4.82, p < .001, d = .36$, than heterosexual males. Likewise, nonheterosexual females had higher Neuroticism, $t(2858) = 3.21, p < .005, d = .17$, and Psychoticism, $t(2856) = 9.87, p < .001, d = .57$, than heterosexual females. Cohen's *d* was calculated by dividing the difference between the heterosexual and nonheterosexual groups by the standard deviation of the heterosexual group. The significance of these effects was confirmed in Mx, which accounts for the non-independence of twin pairs; dropping (separately for males and females) the covariance of sexual orientation with Neuroticism and Psychoticism from the models led to a significant drop in model fit in each case ($p < .005$). Genetic modeling was then used to determine whether the association of sexual orientation with Neuroticism and Psychoticism scores was due to genetic and/or environmental correlation between the variables.

Genetic Modeling: Preliminary Analyses

Before modeling variance components, we tested for heterogeneity in thresholds and twin pair correlations across age, sex, and zygosity. Neuroticism and Psychoticism showed significant age effects (older people had lower scores) and sex effects (females scored higher on Neuroticism and lower on Psychoticism) on the thresholds. These effects were accounted for by including sex and age as covariates in subsequent modeling.

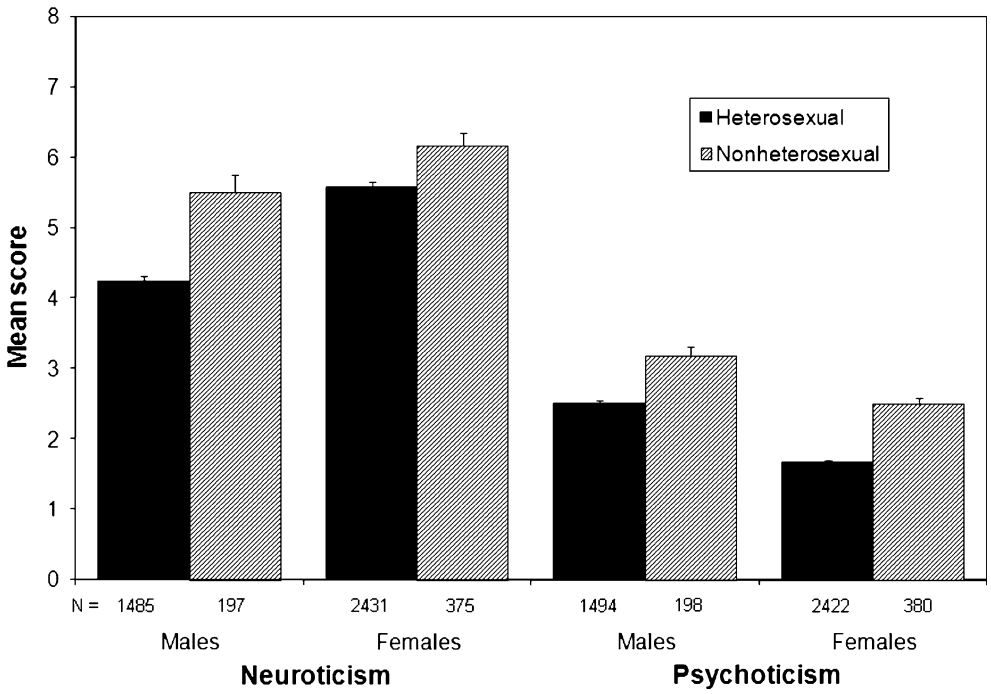

Fig. 1 Mean Neuroticism and Psychoticism scores (and *SE*) for heterosexuals (*dark bars*) versus nonheterosexuals (*light bars*)

Table 2 Twin pair polychoric correlations (with 95% CI) by zygosity group for sexual orientation (dichotomous), Neuroticism (ordinal), and Psychoticism (ordinal)

Zygosity group	*N* (pairs)	Twin pair correlation (95% CI)		
		Sexual orientation	Neuroticism	Psychoticism
MZ female	572–601	.47 (.30, .61)	.41 (.33, .48)	.44 (.37, .50)
MZ male	277–286	.57 (.33, .74)	.49 (.38, .58)	.38 (.27, .48)
DZ female	319–339	.37 (.15, .56)	.20 (.08, .31)	.13 (.02, .24)
DZ male	156–170	.20 (−.11, .49)	.11 (−.05, .26)	.00 (−.15, .15)
DZ opposite-sex	307–331	−.01 (−.26, .25)	.18 (.07, .29)	.20 (.08, .31)

Table 3 Genetic modelling results showing the change in model fit ($\Delta\chi^2$) and degrees of freedom (Δdf) when the specified parameters are dropped

	$\Delta\chi^2$	Δdf	p[a]
Sexual orientation			
Genetic influence on sexual orientation	53.06	1	<.001
Neuroticism			
Genetic influence on Neuroticism	173.11	1	<.001
Genetic correlation with sexual orientation	10.879	1	<.001
Environmental correlation with sexual orientation	.063	1	ns
Psychoticism			
Genetic influence on Psychoticism	179.26	1	<.001
Genetic correlation with sexual orientation	23.25	1	<.001
Environmental correlation with sexual orientation	4.59	1	.03

[a] Significant parameters are those that cause a significant worsening of model fit ($p < .005$) when dropped

Polychoric twin pair correlations are displayed in Table 2. For each trait, MZ twin correlations were higher than DZ twin correlations, suggesting genetic influences. Twin correlations were not significantly different for male versus female MZ pairs, nor for male versus female versus opposite-sex DZ pairs, suggesting no sex differences in the relative contributions of genetic and environmental influences. Based on the size of the DZ correlations relative to the MZ correlations, C was estimated as a specific influence on sexual orientation, whereas a specific D influence was estimated for Neuroticism and Psychoticism.

The low opposite-sex twin pair correlation in sexual orientation suggested that different factors may influence sexual orientation in males and females. We initially accounted for this by allowing for different sources of genetic influence in males and females (i.e., by not fixing the opposite-sex twin genetic correlation at 0.5), but this sex-limitation could be removed (i.e., the correlation fixed at 0.5) without significant loss of model fit ($\Delta\chi^2_1 = 0.37, p = .55$). Similarly, the *magnitudes* of the genetic and environmental influences could be

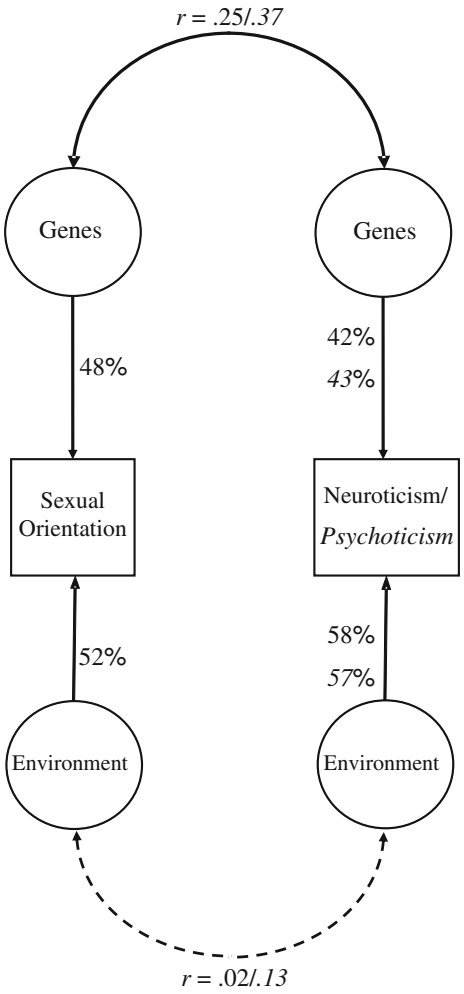

Fig. 2 Results for two bivariate correlated factors models, the first of sexual orientation with Neuroticism, and the second of sexual orientation with Psychoticism (results in italics). Percentages show the proportion of variance in each trait which is accounted for by additive genetic (Genes) and unshared environmental (Environment) influences. Note that Environment includes all non-genetic effects, including measurement error. Double headed arrows represent genetic and environmental correlations, indicating the degree to which the same genetic or environmental factors are influencing the different traits. The dashed line indicates that the environmental correlations are non-significant

equated between the sexes in both the sexual orientation-Neuroticism model ($\Delta\chi^2_8 = 15.03$, $p = .06$) and the sexual orientation-Psychoticism model ($\Delta\chi^2_8 = 11.62$, $p = .17$), indicating the relative contribution of genetic and environmental influences to the variance and covariance of the traits did not differ significantly between males and females. The effect C was nonsignificant for sexual orientation ($\Delta\chi^2_1 = 0.01$, $p = .92$) and D was nonsignificant for Neuroticism ($\Delta\chi^2_1 = 0.94$, $p = .33$) and Psychoticism ($\Delta\chi^2_1 = 1.78$, $p = .18$), so hypotheses regarding genetic and environmental effects were tested for the sample as a whole against AE models with males and females equated.

Genetic Modeling: Hypothesis Testing

Genetic model testing (Table 3) showed that sexual orientation was significantly influenced by genetic factors (48% of the variance). Neuroticism and Psychoticism were also significantly influenced by genetic factors (42% and 43% of the variance, respectively). Genetic correlations between sexual orientation and both Neuroticism and Psychoticism were highly significant, whereas the corresponding environmental correlations were not significant. Figure 2 displays these results in the form of a path diagram.

These results suggest that some of the genetic variance underlying sexual orientation also influences Neuroticism and Psychoticism levels, driving significantly higher Neuroticism and Psychoticism scores in nonheterosexuals as displayed in Fig. 1. In contrast, environmental factors influencing sexual orientation did not appear to affect levels of Neuroticism or Psychoticism.

Discussion

It has previously been found that nonheterosexuals are at much greater risk of psychiatric disorder than are heterosexuals (Fergusson et al., 1999; Meyer, 2003; Mills et al., 2004; Sandfort et al., 2001). We sought to investigate the etiology of this phenomenon by using Eysenck's personality scales Neuroticism and Psychoticism as markers for psychiatric vulnerability. Firstly, we found that scores on both Neuroticism and Psychoticism were significantly elevated in nonheterosexuals compared with heterosexuals, indicating greater vulnerability to neurotic and psychotic disorders, respectively. Secondly, analyses with our genetically informative sample revealed significant genetic correlations between sexual orientation and both Neuroticism and Psychoticism, but corresponding environmental correlations were not significant. This suggests that some of the genetic variation underlying sexual orientation also affects levels of Neuroticism and Psychoticism.

These results have a number of implications. Firstly, sexual orientation differences in Eysenck's Neuroticism and Psychoticism did not mirror sex differences. As expected, women scored significantly higher on Neuroticism whereas men scored significantly higher on Psychoticism—an effect that was observed in nonheterosexuals as well as heterosexuals. In contrast, nonheterosexual men *and* women had significantly higher Neuroticism *and* Psychoticism scores than their heterosexual counterparts. This counters the idea that the apparent elevation of psychiatric risk in nonheterosexuals is simply due to a mirroring of sex differences, for example that nonheterosexual men are at elevated risk of female-typical disorders, such as depression or eating disorders. It also contradicts previous findings (Lippa, 2005, 2008) that personality differences in nonheterosexuals tend to mirror sex differences. The reason for the contrast with Lippa's findings is not clear; as he employed nonstandard personality measures which he constructed himself based on the Big Five model (Digman, 1990), it is difficult to assess how his neuroticism measure might differ from Eysenck's Neuroticism scale. Eysenck devised his Neuroticism and Psychoticism scales with reference to psychiatric vulnerabilities, and the short scales are especially clinically focused, whereas Big Five traits result from agnostic factor analysis techniques and are broader in scope. Future work on Big Five differences between sexual orientation groups could use a standard, widely used measure such as the NEO-PI-R (Costa & McCrae, 1994), as there is a large body of work validating those scales and investigating their relationship to scales in other standard personality measures.

A second implication from our results is that it is unlikely an environmental factor, such as childhood sexual abuse, drives the elevated psychiatric risk in nonheterosexuals by predisposing to both nonheterosexuality and elevated psychiatric vulnerability. Such an effect might have been expected by those suggesting childhood sexual abuse predisposes to homosexuality (Cameron & Cameron, 1995; James, 2006), given that it also predisposes to psychiatric problems (Friedman et al., 2008; Hughes et al., 2001; Roy, 2002; Sun et al., 2008; Young et al., 2007). Unknown non-genetic factors not shared between twin pairs, along with measurement error, account for over 50% of variance in sexual orientation. These factors could include prenatal effects, idiosyncratic experiences, unequal parental treatment, interactions with siblings, or influences outside the family (e.g., teachers and peers). Whatever the specific environmental factors are, they seem not to overlap much *at the population level* with the environmental factors underlying Neuroticism or Psychoticism levels, given the very low and nonsignificant environmental correlations we found. This does not discount the possibility, though, that in individual cases an environmental influence during development could lead to a nonheterosexual orientation as well as psychiatric vulnerability.

A third implication is that genetic variation underlying sexual orientation also underlies, to an extent, Neuroticism and Psychoticism scores. We found heritabilities for these traits in the 42–48% range, in broad accordance with previous studies (Birley et al., 2006; Keller et al., 2005; Kendler et al., 2000; Pillard & Bailey, 1998), and the genetic correlations between traits indicate that some of this genetic variance overlaps. The most direct interpretation of this would involve a pleiotropic genetic factor (genes or linked genes which, on the one hand, predispose to being nonheterosexual and, on the other hand, predispose to having higher Neuroticism and Psychoticism scores and hence more vulnerability to mental illness). Bailey (1999) suggested that elevated rates of non-righthandness that have been observed in both nonheterosexuals (Lippa, 2003) and the mentally ill (DeLisi et al., 2002; Elias, Saucier, & Guylee, 2001; Hicks & Pellegrini, 1978) may reflect a biological factor that manifests in unusual laterality, sexual orientation, and psychiatric problems. If there is a biological correlate of both sexual orientation and psychiatric vulnerability, though, it might be more clearly observed in brain imaging research. A possible example is amygdala connectivity, which has been shown to differ with sexual orientation (Savic & Lindstrom, 2008) and also plays a role in regulating emotion (Banks, Eddy, Angstadt, Nathan, & Phan, 2007; Phelps & LeDoux, 2005). Future research could investigate whether brain differences in nonheterosexuals (Swaab, 2008) are associated with higher psychiatric risk.

The finding in our data of genetic correlations between sexual orientation and psychiatric vulnerability should be interpreted with caution, as it does not necessitate that pleiotropic genetic factors are at work. Other causal relationships could also manifest as genetic correlation between sexual orientation and psychiatric vulnerability. For example, if nonheterosexuality caused elevated Neuroticism and Psychoticism (perhaps via prejudice experienced by nonheterosexuals), then the genetic *and environmental* variation underlying sexual orientation would also contribute to the variation in Neuroticism and Psychoticism. The aspect of our results that suggests something more to the story is the contrast between the highly significant genetic correlation between sexual orientation and psychiatric vulnerability and the lack of corresponding environmental correlation. This pattern of results is suggestive of pleiotropic genetic factors contributing to elevated psychiatric risk in nonheterosexuals, but our sample was insufficient in both size and data characteristics (non-continuous and/or non-normal variables, twins only, no data on social prejudice, magnitudes of genetic and environmental influences too similar) to directly test competing causal hypotheses (Duffy & Martin, 1994). It is likely that there are several contributing factors to the elevated psychiatric risk in nonheterosexuals, genetics being one of these factors.

Another reason for caution is the low opposite sex twin pair correlation for sexual orientation. Although the confidence intervals for this correlation were very wide, it suggests that the etiology of male and female sexual orientation may differ (i.e., there may be sex-limitation in the influences on sexual orientation). To check that this was not biasing our results, we also ran sex-limited models that allowed for different etiology in males and females, as well as models with the opposite sex pairs excluded entirely. These models showed a similar pattern of results to those reported, so it does not appear that the low opposite sex twin pair correlation for sexual orientation inordinately affected the analyses. Nevertheless, the possibility of sex differences in the etiology of sexual orientation should be considered when interpreting the current results, and when designing future research.

Although the limitations of our study preclude definitive conclusions regarding the causes of increased psychiatric vulnerability in nonheterosexuals, our investigation yielded significant advances. Firstly, our results contradict the idea that sexual orientation differences in psychiatric vulnerability simply mirror sex differences, suggesting instead a broad, overall elevation of psychiatric vulnerability in male and female nonheterosexuals. Secondly, the lack of environmental correlation between sexual orientation and either Neuroticism or Psychoticism render it unlikely that environmental factors during development (such as childhood abuse) drive the elevated psychiatric risk in nonheterosexuals. Thirdly, the finding of genetic correlations between sexual orientation and both Neuroticism and Psychoticism lends weight to the possibility that there may be genetic factors that predispose to both nonheterosexuality and psychiatric vulnerability, and that this contributes to the elevated rates of mental health problems observed in nonheterosexuals.

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