Sex Differences in Magical Ideation: A Community-Based Twin Study

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Sex Differences in Magical Ideation: A Community-Based Twin Study

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Two questions regarding sex differences in magical ideation were investigated in this study: (1) whether there are mean-level sex differences on the Magical Ideation Scale (MIS), and (2) whether there are quantitative and/or qualitative sex differences in the genetic contributions to variation on this scale. These questions were evaluated using data obtained from a large community sample of adult Australian twins (N = 4,355) that included opposite-sex pairs. Participants completed a modified 15-item version of the MIS within a larger assessment battery. Women reported both higher means and variability on the MIS than men; this was also observed within families (in opposite-sex twin pairs). Biometric modeling indicated that the proportion of variation in MIS scores due to genetic influences (indicating quantitative sex differences) and the specific latent genetic contributions to this variation (indicating qualitative sex differences) were the same in men and women. These findings clarify the nature of sex differences in magical ideation and point to avenues for future research.

Keywords: magical ideation, sex differences, twin study, schizotypy, schizotypal personality disorder

Magical ideation is conceptualized as the tendency to accept unconventional forms of causality (Meehl, 1962; Horan, Reise, Subotnik, Ventura, & Nuechterlein, 2008). This definition includes a broad range of unconventional thoughts, from relatively common beliefs to delusions (Eckblad & Chapman, 1983; Brugger & Graves, 1997). Magical ideation loads onto the positive factor of schizotypy, or the factor that is characterized by odd beliefs and unusual experiences (Vollema & van den Bosch, 1995; Claridge et al., 1996; Venables & Rector, 2000). Magical ideation is a common symptom of schizotypal personality disorder (DSM-IV; APA, 2000). Much of the research regarding magical ideation has relied on the use of psychometric inventories, mainly the Magical Ideation Scale (MIS; Eckblad & Chapman, 1983). The scale includes 30 items designed to tap into beliefs and experiences regarding forms of thinking that, in terms of conventional standards of the predominant culture, are regarded as invalid (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994). Magical ideation has been shown to be related to mania (e.g., Kwapil, Barrantes-Vidal, & Silvia, 2008) and the trait of absorption (similar to openness to experiences; Eckblad & Chapman, 1986).

The current study investigated the role of genetic and environmental factors in explaining individual differences in the MIS. Four previous studies have conducted behavioral genetic analyses of the MIS (Kendler & Hewitt, 1992; Miller, 1993; Hay et al., 2001; MacDonald, Pogue-Geile, Debski, & Manuck, 2001), but they have yielded discrepant findings, with only two of the studies obtaining evidence of a significant genetic component (Kendler & Hewitt, 1992; Miller, 1993). Three of the studies had very small sample sizes, with a combined total of only 271 MZ and 195 DZ twin pairs (Kendler & Hewitt, 1992; Miller, 1993, as cited in MacDonald et al., 2001; Hay et al., 2001). The Hay et al. (2001) study was based on a much larger sample of 614 MZ and 720 DZ twin pairs, but the assessment of MIS was based on an abbreviated 2-item scale.

Investigating sex differences can yield clues about the causes of a disorder, and is especially useful because it permits a “unidirectional interpretation,” because sex cannot be impacted by the disorder (Lewine, 1988; Aleman, Kahn, & Selten, 2003). In terms of schizophrenia, there has been research regarding sex differences in multiple facets of the disorder (Leung & Chue, 2000). However, the evidence remains inconclusive regarding sex differences in positive schizotypy. Although some investigations have found no differences (Salem & Kring, 1998; Leung & Chue, 2000), the majority of studies examining sex differences in positive schizotypy have found that females on average report more positive symptoms than males (Rawlings, Claridge, & Freeman, 2001; Fossati, Raine, Carretta, Leonardi, & Maffei, 2003; Mata, Mataix-Cols, & Peralta, 2005). In contrast, the results of a recent meta-
analysis of 29 studies comparing mean scores of men and women on the MIS obtained an overall effect size of essentially zero (Miettunen & Jääskeläinen, 2010). Of the four previous behavioral genetic studies of MIS, one reported overall sex differences for a 4-item composite perceptual aberration and magical ideation scale score ($d = .08$, with women having higher mean MIS scores than men). None of the four studies examined sex differences in the sources of individual differences in MIS scores.

The present study extends the small literature on the genetic epidemiology of magical ideation by fitting biometric models to data collected from a large community sample of Australian twins. Because the current study used an abbreviated version of the MIS, it was important to ascertain the construct validity of the abbreviated form before fitting biometric models. The inclusion of opposite-sex twin pairs permitted us to address two major questions concerning sex differences in magical ideation. The first was whether there were mean-level sex differences in the MIS. Studying opposite-sex twin pairs allowed for a rigorous test of sex differences while controlling for a host of potential confounds (i.e., environmental factors, such as factors related to familial environment, that would be shared between opposite-sex twin pairs) that could complicate research using unrelated individuals. The second question was whether there were quantitative or qualitative sex differences in the genetic contributions to variation in MIS. After establishing the construct validity of the abbreviated MIS in both men and women, biometric modeling of male and female MZ and DZ twin pairs was used to test whether the proportion of variance in the MIS that was attributable to latent genetic factors differed for men and women (quantitative sex differences). With the inclusion of opposite-sex twin pairs, we were able to extend these models to test the extent to which the latent genetic factors contributing to variation in MIS overlapped or differed in men and women (qualitative sex differences).

**Method**

**Participants**

The participants for this study were members of a national community-based Australian Twin Registry (Slutske et al., 2009). The data were collected from 2004–2007, when participants were between 32 and 43 years old ($M = 37.66$, $SD = 2.31$). Of the 4,764 participants who completed a telephone interview, 4,355 (91%) returned a personality questionnaire. These 4,355 individual twins included 1,139 monozygotic females (MZF), 761 monozygotic males (MZF), 864 females from same-sex dizygotic pairs (DZF), 576 males from same-sex dizygotic pairs (DZF), 576 females from opposite-sex dizygotic pairs (OSF), and 439 males from opposite-sex dizygotic pairs (OSM). (For more details, see Slutske et al., 2009). This study was approved by the Institutional Review Boards at the University of Missouri and the Queensland Institute of Medical Research. All of the participants provided informed consent.

**Measures**

Three of the measures for this study were included in the personality questionnaire (MIS, Chapman Infrequency Scale, and Multidimensional Personality Questionnaire [MPQ]; Tellegen, 1982, 1985), and a mania screen was included in the structured diagnostic telephone interview.

**Magical Ideation Scale.** Participants completed an abbreviated 15-item version of the MIS (Eckblad & Chapman, 1983). The abbreviated MIS consisted of 15 true-false items designed to measure true-false items designed to measure “beliefs in forms of causation that by conventional standards are invalid” (p. 215). The original MIS has good test–retest reliabilities among both women and men ($r = .82$ and .80, respectively; Chapman, Chapman, & Miller, 1982), and good construct validity in that it predicts future psychosis (Gogging, Tallent, & Marts, 2005), and is also associated with measures of schizotypal personality disorder (Cicero & Kerns, 2010). In the present study, the 15 MIS items had adequate internal consistency reliability (for women, $\alpha = .77$; for men, $\alpha = .73$).

**Chapman Infrequency Scale.** The 11-item Chapman Infrequency Scale (Chapman & Chapman, 1983) was included in order to exclude those participants who were responding in a random or invalid manner. Participants who endorsed three or more infrequency items were dropped from further study (Chapman & Chapman, 1983). On the basis of Chapman Infrequency Scale scores, 54 (0.01%) participants were excluded from all analyses.

**Multidimensional Personality Questionnaire.** The MPQ (Tellegen, 1982, 1985) is a self-report personality inventory of normal personality. The personality questionnaire included the MPQ Absorption Scale. The Absorption Scale is associated with fantasy and openness to experience (Glisky, Tataryn, Tobias, Kihlstrom, & McConkey, 1991). This measure was used in order to investigate the construct validity of the abbreviated MIS included in the personality questionnaire.

**Mania screen.** The mania screen categorized participants into three categories: no evidence of mania, possible mania, and probable mania. Individuals were assigned a diagnosis of possible mania if they endorsed experiencing a week or more of heightened energy and felt unusually good, and had experienced a week or more of rapid speech and impulsivity. Individuals were assigned a diagnosis of probable mania if they met criteria for possible mania and had been hospitalized or been treated with medication for their symptoms. Seventy-five participants (1.7%) were classified as having a history of possible mania, and 44 participants (1.0%) were classified as having a history of probable mania. Possible mania and probable mania were combined for analyses. The mania screen was also used to establish the construct validity of the abbreviated 15-item MIS used in the present study.

**Data Analysis**

Prior to conducting behavioral genetic analyses of the MIS, it was first necessary to establish that the scale was measurement invariant across sex and an equally valid measure among both men and women. These psychometric analyses were an essential step for guiding and interpreting the behavior genetic analyses.

Factor analyses were initially conducted in order to ensure that the 15 items of the abbreviated MIS yielded a unifactorial measure. Following these initial analyses, CFAs were conducted to test whether there was evidence of cross-sex measurement invariance of the MIS, that is, whether the MIS items function the same in men and women (Reise, Widaman, & Pugh, 1993; Byrne, 2008). This was accomplished by comparing a model in which the measurement parameters
for each of the items (i.e., factor loadings and thresholds) were freely estimated for men and women to a model in which the parameters were constrained to be equal for men and women. The relative fit of the constrained model to the fit of the freely estimated model provided a test of cross-sex measurement invariance. These analyses were conducted in Mplus (Muthén & Muthén, 1998), in which the twin pair data were treated as clustered observations.

Two methods were used to examine mean sex differences in MIS. The first method examined the between-family mean and variances differences using data from all of the men and women in the sample. Models in which either the means or variances (or both) were allowed to vary between men and women were tested in Mplus. The fits of pairs of nested models were compared via a chi-square difference test to determine whether there were significant sex differences in the means or variances.

The second method used data from the DZO twin pairs to determine whether there were significant within-family mean and variance sex differences in MIS. This method controls for all between-family differences that might contribute to or obscure a mean sex difference in MIS. For these DZO twin pairs, mean sex differences were investigated using a matched pairs $t$ test. The variances differences were tested the same way as in the between-family tests described above except that the analyses were restricted to the DZO twin pairs.

The genetic analysis for the MIS partitioned the variance into additive genetic ($A$), shared environmental ($C$), or nonadditive genetic ($D$) and nonshared environmental ($E$) influences. The models were fit directly to the raw twin data by the method of maximum likelihood using data from incomplete as well as complete twin pairs. For each of four models created, the estimates of $A$, $C$ or $D$ and $E$ were either constrained to be equal between men and women or allowed to vary, and the estimate of the genetic correlation ($r_A$) for opposite sex twin pairs was either fixed to 0.5 (because same-sex dizygotic twin pairs share on average half of their segregating genes) or allowed to vary. The univariate ACE sex differences twin model for an opposite-sex twin pair is shown in Figure 1. The figure displays a model in which the causal paths leading to the latent MIS trait ($Mf$, $Mm$) were freely estimated for women ($Af$, $Cf$, and $Ef$) and men ($Am$, $Cm$, and $Em$). Scaling factors from the latent MIS trait to the measured MIS trait were included for women and men ($Sf$ and $Sm$, respectively) to allow for sex differences in the variances in MIS. Means were included in the model for women and men ($Mean_f$ and $Mean_m$, respectively) to allow for mean sex differences in MIS. A more restricted model was fitted in which the ACE parameters were constrained to be equal for men and women, that is, $Af = Am$, compare $= Cm$, and $Ef = Em$. Comparing the fit of the constrained model to that of the freely estimated model indicated whether the etiologic structure of MIS was equivalent for men and women (a test of quantitative sex differences). To test for qualitative sex differences, we compared the fits of models in which the estimates of the genetic ($r_A$) correlation between the latent additive genetic factors contributing to MIS in opposite sex DZ pairs were either free to vary or fixed to 0.5. Scores on the MIS were not significantly associated with age [$r(4269) = -.01$, $p = .49$], so it was not necessary to include age in any of the biometric models. Models that included age yielded results that were identical to the results from models that did not include age.

Results

Factor Analyses

The 15 MIS items were subjected to an EFA using principal-axis factor analysis (using the WLSMV estimation technique) followed by an oblique rotation. We selected a one-factor solution based on the scree plot, eigenvalues, and previous literature (Chapman et al., 1982; Venables & Rector, 2000). In order to confirm these results, a CFA was also performed. The CFA confirmed the appropriateness of the one-factor structure (RMSEA = 0.03, CFI = 0.96, TLI = 0.96). Measurement invariance analyses were conducted in order to examine whether every MIS question functioned the same in men and women (see Table 1). The results of the measurement invariance analyses indicated that items 6 and 8 were sex specific, and therefore a 13-item cross-sex-invariant version of the MIS was used for all subsequent analyses. The results were virtually the same regardless of whether we used the 13- or 15-item MIS scale.

Construct Validity

We examined the construct validity of the abbreviated MIS to ensure that it was a valid measure of psychosis proneness among both men and women. The construct validity of the 13-item abbreviated MIS was evaluated by testing whether it significantly predicted a history of mania or the MPQ absorption scale. Higher
scores on the MIS significantly predicted a history of mania in the full sample (χ²(1) = 52.17, p < .01) and for women (χ²(1) = 27.30, p < .01) and men (χ²(1) = 29.21, p < .01). Furthermore, the MIS scores were significantly correlated with MPQ absorption scale scores in the full sample (r = .58, p < .01) and among women (r = .56, p < .01) and men (r = .56, p < .01). The strong associations with history of mania and the personality trait of psychosis proneness in women as in men. 

**Mean Differences**

There were significant between-family mean sex differences in MIS scores, Δχ²(1) = 39.77, p < .01, d = 0.26 with women (n = 1968, M = 2.18) having a higher mean than men (n = 1302, M = 1.71). The distribution of MIS scores for women and men can be seen in Figure 2. There were also significant between-family variance differences in MIS scores, Δχ²(1) = 17.00, p < .01, with women (σ² = 3.93) having a greater variance than men (σ² = 3.01). When variances were allowed to differ, there were still significant mean between-family sex differences for the MIS score.

A matched pairs t test indicated that there was a significant within-family mean difference between men and women on the total MIS score in DZO twins, t(323) = 3.94, p < .01; women: M = 2.36, SD = 2.04; men: M = 1.80, SD = 1.73; d = 0.29. There were also significant differences in the variances in MIS scores for men and women in DZO twins (Δχ²(1) = 7.62, p < .01) with women (σ² = 4.10) having a greater variance than men (σ² = 2.07).

### Genetic Analyses

Twin correlations for the total MIS score for each of the zyosity groups are presented in Table 2. For men and women, the MZ twin correlations were larger than the DZ twin correlations, and the correlations among male twins, both MZ and DZ were smaller than among female twins. Furthermore, there was a nonsignificant trend indicating a smaller twin correlation for DZO than same-sex DZ twins.

Based upon the results demonstrating mean and variance differences between men and women on the MIS scale, all of the biometric models allowed for mean and variance differences across sex. Based on the relative fits of the ACE (Δχ²(19) = 59.04,
SEX DIFFERENCES IN MAGICAL IDEATION

Table 2

Twin Correlations for the 13-Item Magical Ideation Scale (Omitting Items 6 and 8)

<table>
<thead>
<tr>
<th>Zygosity group</th>
<th>Pairs</th>
<th>Correlation</th>
<th>95% Confidence Interval</th>
<th>$\chi^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZf</td>
<td>472</td>
<td>0.40</td>
<td>0.32–0.46</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>MZm</td>
<td>284</td>
<td>0.31</td>
<td>0.20–0.41</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>DZf</td>
<td>325</td>
<td>0.26</td>
<td>0.16–0.36</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>DZm</td>
<td>160</td>
<td>0.12</td>
<td>−0.04–0.27</td>
<td>0.13</td>
<td></td>
</tr>
<tr>
<td>DZo</td>
<td>323</td>
<td>0.09</td>
<td>−0.02–0.20</td>
<td>0.10</td>
<td></td>
</tr>
</tbody>
</table>

$\chi^2$ test of differences in twin correlations (df = 1)

- MZ vs DZ: $\chi^2 = 7.09, p < .01$
- DZs vs DZO: $\chi^2 = 2.40, p = 0.12$
- MZm vs MZf: $\chi^2 = 4.10, p = 0.04$
- DZm vs DZF: $\chi^2 = 5.04, p = 0.02$
- MZf vs DZF: $\chi^2 = 2.53, p = 0.11$
- MZm vs DZm: $\chi^2 = 4.75, p = 0.03$
- DZf vs DZO: $\chi^2 = 6.03, p = 0.01$
- DZm vs DZo: $\chi^2 = 0.00, p = 0.96$

Note. MZ = monozygotic; DZ = dizygotic; s = same-sex twin pairs; o = opposite-sex twin pairs; m = male; f = female. The correlations remain the same when age was covaried out of the MIS scores.

$p < .01$ and ADE models ($\Delta \chi^2(19) = 60.33, p < .01$), ACE models were used in all analyses presented below (since the ACE model provided better fit indices). An ADE/ACE model (ADE in men, ACE in women) was not used because it would have precluded examining qualitative sex differences.

Prior to examining sex differences, tests were conducted to determine the overall significance of each of the components of A, C, and E. The fit of a model that did not include the A parameter was compared to a full model. The relative fits of these two models ($\Delta \chi^2(3) = 21.58, p < .01$) indicated that dropping A resulted in a substantial and significant deterioration in model fit. Next, the fit of a model that did not include the C parameter was compared to a full model. The relative fits of these two models, ($\Delta \chi^2(2) = 2.07, p = .12$) indicated that C could be dropped from the model without a significant reduction in model fit. (A model that did not include the E parameter could not be fit to the data.) Although we could have proceeded with a more parsimonious “best-fitting” AE model, we took a more conservative approach by testing sex differences within full models. The use of best-fitting models can sometimes yield misleading results because, for example, an AE model is a reduced model that provides estimates of A and E that would be obtained under the strict assumption that C has zero influence.

Quantitative sex differences were investigated by comparing a model in which Af = Am, compare = Cm, and Ef = Em (Table 3, Model 3) to a model that allowed the estimates to vary Af ≠ Am, compare ≠ Cm, and Ef ≠ Em (Table 3, Model 1). The relative fits of these two models ($\Delta \chi^2(3) = 31.09, p < .0001$) indicated that there were quantitative sex differences in the sources of variation in the MIS. Next, qualitative sex differences were tested by comparing the fit of a model that fixed the Af estimate of DZO at 0.5 to a model that allowed it to vary between 0.00 and 0.50 (Table 3, Model 2 vs. Model 1). The model in which Af was fixed to 0.5 for DZO did not provide a significantly worse fit compared to the model in which it was allowed to take on a value less than 0.5 ($\Delta \chi^2(1) = 0.04, p = .84$). The results of this test of

Table 3

<table>
<thead>
<tr>
<th>Zygosity group</th>
<th>DZO fixed, DZO free</th>
<th>DZO fixed, DZO fixed</th>
<th>DZO free, DZO free</th>
<th>DZO fixed, DZO free</th>
<th>DZO free, DZO free</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model 1</td>
<td>472</td>
<td>16.51</td>
<td>15.59</td>
<td>16.51</td>
<td>15.59</td>
</tr>
<tr>
<td>Model 3</td>
<td>472</td>
<td>17.60</td>
<td>17.60</td>
<td>17.60</td>
<td>17.60</td>
</tr>
<tr>
<td>Model 4</td>
<td>472</td>
<td>17.60</td>
<td>17.60</td>
<td>17.60</td>
<td>17.60</td>
</tr>
</tbody>
</table>

Note. A = estimate of genetic correlation for DZO twins; A = additive genetic influence; C = shared environmental influence; E = nonshared environmental influence; f = female; m = male.
qualitative sex differences suggest that there are not significant differences in the latent genetic factors contributing to variation in MIS in men and women. Based on finding quantitative sex differences but not qualitative sex differences, Model 2 was selected as the final model (see Table 3).

After the selection of Model 2 as the final model, follow-up analyses were conducted to probe the quantitative sex difference in the sources of variation in MIS. A model in which ACE were allowed to vary between men and women was compared to a model in which CE were allowed to vary (and A was fixed), $\Delta \chi^2(1) = 0.02, p = .89$. This indicated that there were not significant differences in the contribution of genetic influences on variation in MIS between men and women. Likewise, a model in which ACE were allowed to vary was compared to a model in which AE were allowed to vary (and C was fixed), $\Delta \chi^2(1) = 3.90, p < .05$. This indicated that there were significant differences in the contribution of shared environmental influences on variation in MIS between men and women. Lastly, a model in which ACE were allowed to vary was compared to a model in which AC were allowed to vary (and E was fixed), $\Delta \chi^2(1) = 0.02, p = .89$. This indicated that there were also significant differences in the contribution of the nonshared environmental influences on variation in MIS between men and women.

Discussion

Research on the causes of sex differences in positive schizotypy (Claridge & Hewitt, 1987; Del Giudice, Romina, Adelina, & Marco, 2010; Macare, Bates, Heath, Martin, & Ettinger, 2012) can provide important clues to better understand the phenomenon more generally (Rutter, Caspi, & Moffitt, 2003; Weisberg, DeYoung, & Hirsh, 2011). The present study represents an investigation of sex differences in one aspect of positive schizotypy, magical ideation, as indicated by scores on the MIS. We found that men and women differed in mean levels, variability, and causes of variation in magical ideation. Each of these findings is elaborated on below.

Women scored 0.26 standard deviations higher than men on the MIS in the full sample (i.e., in between-family comparisons). These results were confirmed in a more stringent within-family comparison among opposite-sex twin pairs ($d = 0.29$). Opposite-sex twin pairs provide the ideal comparison because they represent matched pairs that are equated on age, childhood family socioeconomic status, parental and neighborhood characteristics, religion, and all other between-family characteristics (even including those unforeseen) that might contribute to or obscure the mean-level sex difference in MIS. These twin pairs also partially control for genetic differences. In these male-female pairs, both the mean levels and variability of MIS scores were greater in women than in men. Not only were women more likely to endorse items from the MIS, but they were more likely to be in the extremes of the MIS distribution. Finding both higher means and variances in women in magical ideation supports previous research indicating that women have higher levels of positive schizotypy. One explanation for this sex difference is the potential role of estrogen in the development of positive schizotypy. Specifically, there is evidence of higher rates of psychosis among women with lowered estrogen production (e.g., Jacobs & D’Esposito, 2011). Estrogen level may constitute a risk factor for psychosis that is specific to women, pointing to a potential explanation for increased levels of positive schizotypy in women. Furthermore, there is evidence of higher rates of schizophrenia spectrum disorders among women with both lowered estrogen production and variations at the COMT gene locus, which is considered an estrogen metabolism gene (Min et al., 2012). Higher levels of magical ideation variation in women may be in part explained by individual differences in estrogen levels. Thus, there are several pieces of evidence indicating sex-specific factors that may contribute to higher means and variances in women.

The results of the present study are inconsistent with the previous meta-analysis (Miettunen & Jääskäläinen, 2010) that obtained an effect size near zero of the difference between men and women on the MIS. The major difference between the present and previous studies included in the meta-analysis is that the present study was based on a middle-aged community sample and the meta-analysis was primarily based on young adult college student samples. Thus, one logical explanation may be that the magnitude of the sex difference increases with age; however, this explanation was tested and ruled out (Miettunen & Jääskäläinen, 2010). Another possibility is that there are systematic differences (other than age) between college students and community residents that also vary in men versus women (Chmielewski, Fernandez, Yee, & Miller, 1995). College student samples are inherently selective compared to general community samples and the forces that select one into college may differ in men and women. However, Miettunen and Jääskäläinen (2010) found no significant differences between student and nonstudent samples, and therefore this explanation was tentatively tested (with far fewer community than student samples and individuals). It is interesting to note that Miettunen and Jääskäläinen (2010) did report a significant difference between the student and nonstudent samples on another measure of positive schizotypy, perceptual aberration, with women obtaining significantly higher scores than men in nonstudent samples, but not in student samples. The bulk of the evidence suggests that there might be systematic differences between college and community samples that might obscure mean differences between men and women on the MIS (and other indicators of positive schizotypy) when using a college student sample, as has been reported for other characteristics (e.g., Gladstone & Koenig, 1994; Tolin & Foa, 2006).

This study improves upon the weaknesses of the previous studies, with a sample size that far exceeds the total number of participants included in all four of the previous twin studies of MIS, and convincingly demonstrates the importance of genes in contributing to individual differences in MIS scores. The previous studies obtained far-ranging estimates of heritability, accounting for between 0 to 56% of the variation in MIS scores; only two of the four studies obtained evidence for significant genetic influences. In the present study, we obtained a heritability estimate of 28% for men and 29% for women, with no evidence for sex differences. The heritability estimate for this psychosis proneness scale is low compared to the high heritabilities obtained for psychosis per se, that is, schizophrenia and bipolar disorder, which have heritability estimates of at least 80% (Cardno & Gottesman, 2000; McGuffin et al., 2003).

It is worth considering whether differences across the extant studies might be explained by age differences because three of the four previous studies were based on younger adult samples. There is accumulating evidence from the behavioral genetic literature that as individuals age, the proportion of phenotypic variation that is explained by genetic influences increases, potentially through the strengthening of gene–environment correlations in which people seek out environments to which they are genetically predis-
posed (Bergen, Gardner, & Kendler, 2007). One especially interesting comparison is with the previous study of Hay et al. (2001), because it was based on an overlapping sample from the Australian Twin Registry. Many of the individuals who participated in the Hay et al. (2001) study as young adults (18–25 years of age) also participated in the present study some 15 years later (when they were 32–43 years of age). The assessments of MIS were quite different in the two studies and included only one item in common (item #6 in Table 1). Yet the results were remarkably similar, with a heritability of 33% in young adulthood (Hay et al., 2001) and 28–29% in middle adulthood (present study). The scant evidence available suggests that the heritability of scores on the MIS may be relatively stable from early- to midadulthood.

There were significant differences between men and women in the proportion of variation in MIS scores that was due to shared environmental influences, even though the shared environment did not significantly contribute to variation in MIS scores, either overall or in men or women considered individually. Furthermore, although there were not statistically significant qualitative sex differences, when the genetic correlation for opposite-sex twin pairs was freely estimated in a biometric twin model, it was estimated at 0.08, compared to the assumption of a correlation of 0.50 among same-sex DZ pairs. In fact, a genetic correlation of zero between men and women could not be statistically ruled out. Future research might profit from following up on these intriguing clues suggesting that the shared environment may make a stronger contribution to variation in MIS in men than in women, and that the genetic risk factors for MIS may differ in men and women.

Conclusions

In sum, we found that men and women differed in mean levels and variability, but not necessarily in the causes of variation in magical ideation. On average, women scored higher on a measure of magical ideation, and their distribution of scores on magical ideation showed more variability than among men. The proportion of variation in magical ideation scores due to genetic influences and the specific latent genetic factors contributing to MIS variation did not differ between men and women.

These results have implications for future research. First, the within-family comparisons of men and women convincingly demonstrated a mean-level sex difference in magical ideation scores. These findings raise doubts about the use of college student samples for making broader inferences about mean-level sex differences (in magical ideation as well as other traits) in the general population. The mean and variance differences indicate that women are not only endorsing more MIS items, but also occupy more of the extremes of the distribution. This supports previous research indicating higher rates of positive schizotypy in women, and indicates the possibility of sex-specific risk factors (i.e., estrogen) contributing to increased magical ideation in women. Second, the biometric analyses convincingly demonstrated that genetic factors explain variation in magical ideation in both men and women. However, much work is left to do in examining whether there are differences in the contributions of genetic and environmental factors to variation in magical ideation across development, or whether there are certain environmental contexts in which genetic influences on magical ideation are amplified. An obvious next step will also be to identify the specific genes that are associated with magical ideation. Because magical ideation is a quantitative trait that can be easily measured in large surveys of the general population, it might serve as a useful endophenotype in the search for susceptibility genes for the psychotic disorders.

References


