Are There Sex Differences in the Genetic and Environmental Effects on Mental Rotation Ability?

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Probably the most robust sex difference in cognitive abilities is that on average males outperform females in tests of mental rotation. Using twin data we tested whether there are sex differences in the magnitude of genetic and environmental effects on mental rotation test performance and whether the same or different genetic effects operate in females and males. The present study replicated the well-known male advantage in mental rotation ability. The relative proportion of variance explained by genetic effects did not differ between females and males, but interestingly, absolute additive genetic and unique environmental variances were greater in males reflecting significantly greater phenotypic variance in mental rotation test performance in males. Over half of the variance in mental rotation test performance was explained by genetic effects, which suggest that mental rotation ability is a good phenotype for studies finding genes underlying spatial abilities. Results indicate that females and males could be combined for such genetic studies, because the same genetic effects affected mental rotation test performance in females and males.

Keywords: genetic modeling, heritability, mental rotation test, sex difference, twins

There exists a robust sex difference in mental rotation ability, with males on average outperforming females (for a review see Voyer et al., 1995). Both biological and environmental explanations for the sex difference in mental rotation ability have been raised. The biological basis is supported by the fact that the sex difference in mental rotation ability is not only restricted to adults, but is evident also before puberty (Kerns & Berenbaum, 1991) and seems to appear already in infancy at 3–5 months (Moore & Johnson, 2008; Quinn & Liben, 2008) Furthermore, it is evident in different cultures (Peters et al., 2006; Silverman et al., 2007) and also other species do exhibit sex differences in spatial abilities (Jonasson, 2005; Jozet-Alves et al., 2008). There is also evidence showing that social environment can affect mental rotation ability. Spatial abilities in general can be improved by training of spatial skills (for a meta-analysis see Baenninger & Newcombe, 1989). Spatial activities and computer experience has been suggested to correlate positively with mental rotation test performance in females, but not in males (Quaiser-Pohl & Lehmann, 2002) and playing an action video game has been reported to reduce the sex difference in mental rotation test (Feng et al., 2007).

Given the large number of studies concerning sex differences in mental rotation ability it is surprising that to date, no studies have investigated the magnitude of possible sex-specific genetic and environmental effects in mental rotation ability using a twin design. Previously, only one study has investigated mental rotation ability in twins (Vandenberg, 1969). That study indicated a significant role of genetic effects on mental rotation ability, but having no twins from opposite-sex pairs that study was not aimed to investigate the possible sex specific genes underlying this spatial ability. Functional magnetic resonance imaging studies have suggested that different brain regions are activated in females and males while performing the mental rotation test (e.g., Butler et al., 2007; Clements-Stephens et al., 2009; Hugdahl et al., 2006), which could reflect underlying sex-specific genetic or environmental effects.

Here, we concentrated on the sex difference in the variance of mental rotation ability (we have reported the sex difference in the mean performance on Mental

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Rotation Test in more detail elsewhere, see Vuoksimaa et al., in press). The three aims of this study were: (1) to examine the sex difference in phenotypic variance of mental rotation test performance; (2) to study the proportion of variance explained by genetic and environmental effects, and to test whether the magnitude of genetic and environmental effects differ between females and males; and (3) to test whether same or different genetic effects contribute to mental rotation ability in females and males.

Methods

Subjects

Our study sample consisted of 804 young adult twins, aged 21–24, from the population-based FinnTwin12 study, which includes nearly all Finnish twin born in 1983–1987. Mental rotation ability was assessed as a part of a laboratory study protocol in the fourth wave of data collection between 2006 and 2009. To this study phase we invited all twins who had participated in the intensive study protocol at age 14. Detailed information about FinnTwin12 and intensively studied twins can be found elsewhere (Kaprio et al., 2002). We studied mental rotation ability in 358 twin pairs including 97 monozygotic female pairs (MZF), 46 monozygotic male pairs (MZM), 71 dizygotic female pairs (DZF), 50 dizygotic male pairs (DZM) and 94 opposite-sex pairs (DZOS). In addition, we had 88 twins whose co-twin did not participate. The zygosities were determined using a validated questionnaire (Sarna et al., 1978), with added questions for younger twins (Goldsmith, 1991). Zygosity was confirmed from DNA for all but one same-sex twin pairs. All subjects gave written informed consent before participating in the study. The FinnTwin12 study protocol was approved by the ethical committee of Helsinki and Uusimaa hospital district and IRB of Indiana University, Bloomington.

Stimulus

We used the redrawn version of Vandenberg and Kuse Mental Rotation test (MRT) (Peters et al., 1995), which is based on the stimuli by Shepard and Metzler (1971). Before administering the scored test trials, subjects were given 5 minutes to complete four practice trials and the correct answers for these trials were shown. The actual test consisted of two parts with 12 trials in each part. Each trial consisted of a target figure and four additional test figures. Subjects had to decide which two of the four test figures represented the target figure rotated to a different angle through the vertical axis. The subjects were given one point if they marked both correct alternatives. One correct and one incorrect answer yielded zero points. Thus, possible total score ranged from 0 to 24. There was a 3-minute time limit to complete each part.

Modeling of Genetic and Environmental Effects

Using Mx-software (Neale at al., 2003) we examined how much of the phenotypic variance in MRT is explained by additive genetic (A), dominant genetic (D), common environmental (C), and unique environmental (E) effects. A reflects the cumulative effects of multiple additive genes, and correlates 1.0 in MZ and 0.5 in DZ twins. D signify the dominant genetic effects with 1.0 correlation in MZ and 0.25 in DZ twins. C stands for environmental effects that make the twins alike and correlates 1.0 both in MZ and DZ twins. E refers to all environmental effects that make twins unlike and is uncorrelated both in MZ and DZ twins and includes also measurement error. With twin data it is not possible to include D and C components in the same model, thus ACE and ADE models have to be tested separately. Mx software calculates both absolute (unstandardized) and relative (standardized) estimates for the genetic and environmental variance components. Absolute genetic and environmental variances sum to the phenotypic variance, whereas relative variance reflects the proportion of variation explained by the A, D, C or E effects.

We first computed a saturated model, allowing free variation of parameters across zygosity and sex effects on means, variances and covariances on MRT performance. Full ACE and ADE models were tested against the saturated model. After that we compared several sub-models against the full sex limitation model to see whether dropping A, D or C effects or constraining female and male A, D or C components to be equal worsened the fit of the model. By using maximum likelihood method with raw data Mx calculates minus two times log-likelihood of data for each model. By comparing the difference in minus two times log-likelihood values when taking into account the difference in the degrees of freedom (df) the p value is calculated. This indicates whether the submodel reduces the fit of the model or not. Significant p value (less than 0.05) indicates the poorer fit of the submodel. If two models fit the data equally, the more parsimonious model is preferred. In other fit index, the Akaike's information criterion (AIC), the lowest value indicates to best fitting model.

Other Statistical Analyses

We used the Adjusted Wald test for studying the sex difference in the mean MRT score and χ^2-test was used to test sex differences in the group of top and bottom scorers. Confidence intervals (CI) for variance ratio were calculated with the bootstrap method using percentile-based confidence intervals. The clustered family data was taken into account in all analyses.

Results

There was a significant sex difference in MRT showing that on average males (mean = 13.15, 95%CI 12.58–13.72) performed better than females (mean = 9.33, 95%CI 8.90–9.76), $F(1,445) = 120.43$, $p < .001$, $d = 0.84$, and males had also greater variance than females (variance ratio 1.34, 95% confidence intervals, 1.13–1.61). There were significantly more males

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than females in the top 5%, $F(1,445) = 36.5$, $p < .001$, and 10%, $F(1,445) = 67.8$, $p < .001$, scorers, whereas number of females was significantly greater in subjects who scored in the bottom 4%, $F(1,445) = 8.37$, $p < .01$, or 8%, $F(1,445) = 20.1$, $p < .001$ (Table 1). The mean MRT scores by sex and zygosity are reported elsewhere (see Vuoksimaa et al., 2010).

The intrapair twin correlations indicated genetic effects on MRT performance: MZF = .54, DZF = .25, MZM = .47, DZM = .26, and DZOS = .39. In females the MZ correlation coefficient was about two times the DZ correlation, which suggests the possibility of both additive genetic and unique environmental effects, whereas in males the MZ correlation was less than twice higher than DZ correlation suggesting additive genetic effects and possible common environmental effects. The correlation in opposite-sex pairs was higher than DZ correlation in females or males from same-sex pairs, providing no evidence of different genes operating in males and females.

The saturated model indicated that there were no differences in means or variances between randomly ordered first and second twins, or between zygosity groups. But there was a significant sex effect on the means. After the saturated model, we ran the full sex-limitation model, which allowed different proportions of genetic and environmental effects for females and males. We used sex as a covariate and allowed different means for females and males due to sex effect on mean MRT scores. Among females, different means were allowed for females with male co-twins and for females with female co-twins due to significant difference in MRT performance between these two groups of females (see Vuoksimaa et al., 2010). The fit of the full ACE sex-limitation model was compared to the saturated model: the nonsignificant ($p = .09$) change in minus two times log likelihood was 17.77 (df = 11) indicating that full sex-limitation model was acceptable.

Next, we fitted several submodels against the full ACE sex-limitation model (see Table 2 for model-fit statistics). Our results indicated that the C component could be dropped both from females and males (Model 2), whereas dropping the A component from females and males significantly worsened the fit (Model 3). Also dropping both A and C component at the same time worsened the fit of the model (Model 4). When we tested AE, AC and E models against the full ADE sex-limitation model (model fitting statistics not shown), the results were similar than compared to ACE model indicating that AE model fit the data best.

Constraining the A and E components to be equal in females and males (Model 5) worsened the fit when compared against AE model where different magnitude of A and E effects were allowed for females and males. Finally, we constrained the opposite-sex pair genetic correlation at 0.5 (the expected value when no sex-specific gene effects are present): this model fitted the data well indicating that same set of genes operate in females and males (Model 6).

The AE model that allowed different magnitude of genetic variance for females and males best fit our data. In that model, the standardized (relative) additive genetic variance explained 55% (95%CI 42–67%) of the phenotypic variance of MRT in females and 53% (95%CI 33–68%) of the variance in males. The unstandardized (absolute) genetic variance was 9.88 in females and 13.06 in males and unstandardized unique environmental variance was 7.93 in females and 11.66 in males (Figure 1).

Table 1

Sex Difference in the Extreme Scores of the Mental Rotation Test: Proportion of Females and Males in the Tail Regions and Male:Female Ratios

Percentile	Females	Males	Male to female ratio
< 5%	82.4%	17.7%	0.21
< 10%	85.7%	14.3%	0.17
≥90%	15.5%	84.5%	5.45
≥95%	14.3%	85.7%	5.99

Note: Value higher than one indicates a greater proportion of males in the region.

Discussion

The large effect size for the sex difference in the MRT performance in our study replicates the well-documented male advantage in mental rotation ability (Voyer et al., 1995). We have earlier reported that the sex difference in

Table 2

Model Comparison Statistics for the Univariate Sex-Limitation Model for Mental Rotation Test Performance

Model	Change in χ^2	Change in *df*	*p*	AIC	Compared against model
1. ACE					
2. AE*	0.277	2	0.871	-3.723	ACE
3. CE	8.481	2	0.014	4.481	ACE
4. E	64.665	4	0.000	56.665	ACE
5. AEf = AEm	10.335	2	0.006	6.335	AE
6. ra = 0.5	0.000	1	1.000	-2.000	AE

Note: A = additive genetic effects, C = common environmental effects, E = unique environmental effects. AEf = model that includes A and E effects for females. AEm = model that includes A and E effects for males. ra = genetic correlation. * = best fitted model, where different magnitudes of additive genetic and unique environmental effects for females and males were allowed. Df = degrees of freedom. AIC = Akaike's information criterion.

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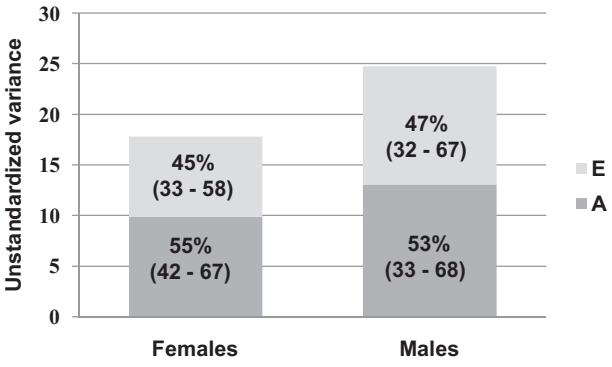

Figure 1

Unstandardized/absolute (on y-axis) and standardized/relative (indicated as percentages with 95%CI's in parentheses) additive genetic and unique environmental variances of mental rotation tests performance in females and males in the best fitting model. A = proportion of variance explained by additive genetic effects. E = proportion of variance explained by unique environmental effects.

this sample of twins is evident between females and males from same-sex pairs and also between females and males from the opposite-sex pairs (see Vuoksimaa et al. 2010). In the present study, we found that males showed significantly higher phenotypic variance than females. Earlier studies have indicated that males have higher variance than females in variety of cognitive domains, including spatial abilities (Hedges & Nowell, 1995; Lohman & Lakin, 2009). The sex difference in means of MRT was reflected on the tails of the distribution of MRT scores. In the top 5% of scorers the proportion of males was six times higher than in females whereas in the bottom 4% of scorers the proportion of females was five times higher than in males.

Our model fitting results indicated that the best-fitted model comprised additive genetic and unique environmental effects and allowed these components to vary in females and males. This model fitted the data better than the AE model where genetic effects could be constrained to be equal between sexes even though the proportion of variance explained by genetic effect varied only two percent in females and males. In fact, as indicated by greatly overlapping confidence intervals, the proportion of variance explained by additive genetic effects was equal between sexes. Despite the similarity in relative genetic variance, there was a clear sex difference in absolute genetic variance: males had substantially greater genetic variance than females. Additive genetic effects accounted for over half of the variance in MRT, which is comparable to additive genetic effects on general cognitive ability in young adults (Haworth et al., in press; Lyons et al., 2009). Our results indicate that the male advantage in MRT is not explained by males' and females' different magnitude of genetic and/or environmental effects. It is possible that the male advantage in MRT is created by gene environment interactions. Such interactions have been reported for example in general cognitive ability: socioeconomic status moderates the heritability of intelligence quotient (Turkheimer et al., 2003). Moreover, prematurity moderates the genetic effects on both verbal and non-verbal cognitive development (Koeppen-Schomerus et al., 2000). Gene-environment interactions can be also sex specific; thus future studies should investigate whether environmental factors modify genetic effects differently in males and females. Earlier research suggests that prenatal testosterone levels (Vuoksimaa et al., in press) and computer game playing experience (Feng et al., 2007) could be such factors.

What might be the cause for greater phenotypic variance and greater absolute genetic variance in males compared to females? Our results do not offer definitive answers to this question, and we can only speculate reasons for the greater variance as well as greater absolute genetic variance of MRT in males than in females in the present study.

One of the limitations of our study, even though we had a large sample for an intensive laboratory assessment, was inadequate power to detect small common environmental effects that were suggested by twin correlations in males from same-sex pairs. Larger samples will be needed to assess whether there are common environmental effects in males. In females from same-sex pairs, the twin correlations were not suggestive of common environmental effects.

Our results have important implications for seeking genes that affect spatial abilities that show male advantage. Since additive genetic effects explained over half of the variance in MRT, genome wide association studies could be effective in detecting the genetic regions that underlie mental rotation ability that shows robust and large sex difference. Based on our results, females and males could be combined for such studies, because the same genetic effects are likely to affect MRT performance in both.

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