Genetic and Environmental Contributions to ADHD Using the Conners’ Rating Scales-Revised

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Objective

Means, variances, and twin correlations were calculated using the statistical software program Mx and are presented below. Differences in mean scores were tested by likelihood-ratio \( \chi^2 \) tests. Because the ADHD-I scale from the Conners-Revised Form was not normally distributed, the data were square-root transformed to approximate normal distribution.

All model fitting was performed on transformed data with Mx. The basic model was an ACE or ADE model with additive and without sex and interaction effects. The possible presence of an interaction component was tested by equating the variances between MZ and DZ twins. The basic model is shown below.

The significance of the A, D, and C factors or sibling interaction was tested by dropping these variance components, using the \( \chi^2 \) difference test. We also computed likelihood-based 95% confidence intervals.

Discussion

The Conners-Revised ADHD-I combines the strengths of both DSM and CBCL taxonomic approaches in the study of ADHD. Here we report on heritability estimates for DSM ADHD more in line with those reported for AP and AGG of the CBCL (70%) than with studies which use DSM-IV categorical, yes/no data, which report heritability estimates in the 90-95% range.

In addition, these data, which allow for gender sensitive analyses (comparing data on females to females, males to males), illustrate the perception that the DSM-IV ADHD items identify too few females as suffering from ADHD.

Finally, our model fitting identifies the contribution of genetic dominance. This model, which identifies genetic dominance as the primary influence on ADHD, has not been reported previously for ADHD. Our group has found evidence of genetic dominance on AP from the CBCL, but not in all age groups and not for all informants. In the ADE model we identified additive genetic influences (29%), moderate dominant genetic influences (15%), and modest unique environmental contributions (21%). If such models are replicated, existence of genetic dominance argues for a different approach to identifying heritable phenotypes of the genetic study of ADHD. The issue of the ADE model versus models reported by others such as the AG model can only be settled as we increase the sample sizes, types of samples (such as adopted sib designs, unrelated designs, and multi-informant designs).

Summary of Model Fitting

The difference in \( \chi^2 \) indicates the goodness-of-fit of the model, compared to a saturated model. First, variance differences between MZ and DZ twins were tested. The fit of a model that constrained the variances to be equal was compared to the fit of a fully saturated model in which all variances and covariances were freely estimated. The variances were not significantly different, as a result an interaction component was not included.

Second, an ADE model was fit to the data. This model provided a very good fit to the data (\( \chi^2 = 316.38 \), \( p = 0.05 \)). The model had equal loadings of the variance, the dominant genetic factor 50%, and the unique environmental factor 21%. The confidence intervals are provided above.

Questions

1. What percentage of children, by gender, meet CRS criteria for clinical deviance on the ADHD-I?

2. Are rates of ADHD-I more or less common in our general community twin sample compared to DSM rates of ADHD?

3. What are the estimates of the genetic and environmental contributions to ADHD-I and are there gender differences?

Results

- Most square root transformed ADHD-I scores and standard deviations by sex and zygosity:
  - Monozygotic Males: 2.60 (1.33)
  - Dizygotic Males: 2.63 (1.36)
  - Monozygotic Females: 2.09 (1.32)
  - Dizygotic Females: 2.16 (1.36)

- Summary of Model Fitting:
  - The ADE model was the best fitting model. The additive genetic factor explained 29% of the variance, the dominant genetic factor 50% and the unique environmental factor 21%. The confidence intervals are provided above.

Limitations

1. Like other factor analytically derived approaches the ADHD-I does not retain all 18 items of the DSM-IV, thus use of the ADHD-I is not a direct test of DSM-IV ADHD. The ADHD-I is closer in content to DSM than other measures of ADHD.

2. Data on maternal report may not generalize to children of older ages or to other informants. Our group is currently collecting data on older twins by father and teacher report in order to test for these factors.

3. We did not directly interview the parents or children in this study and therefore cannot present data on the number of children who exceeded ADHD-I cut-off’s who also met DSM-IV criteria for ADHD. In order to test for these data, our group is currently interviewing a subset of this sample in order to determine those relations.

Conclusions

Use of the Conners’ Parent Report Scale-Revised ADHD-I to estimate genetic and environmental contributions to ADHD combines the strengths of categorical and quantitative taxonomic approaches in the study of ADHD.

Our data are consistent with prior reports that ADHD is predominantly influenced by genetic factors that are both dominant and additive.

Table: Twin Estimates

<table>
<thead>
<tr>
<th>Source of Variance</th>
<th>Standardized Estimate</th>
<th>CI (low-high)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>.288</td>
<td>.051 - .515</td>
</tr>
<tr>
<td>D</td>
<td>.492</td>
<td>.209 - .740</td>
</tr>
<tr>
<td>E</td>
<td>.212</td>
<td>.156 - .294</td>
</tr>
</tbody>
</table>

Diagram: Twin Correlations

![Diagram of Twin Correlations]

- Twin Type: Monozygotic Males / Monozygotic Females / Dizygotic Males / Dizygotic Females / Dizygotic Opposite Sex

- Twin-Twin Correlations:
  - Monozygotic Males: .794
  - Dizygotic Males: .289
  - Monozygotic Females: .287
  - Dizygotic Females: .253
  - DOS - male eldest: .263
  - DOS - female eldest: .263