DCD and ADHD: A genetic study of their shared aetiology

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Abstract

Previous studies have found that rates of attention deficit hyperactivity disorder (ADHD) and developmental coordination disorder (DCD) are very similar, both being approximately 7% in sample populations [Kadesjö, B., & Gillberg, C. (1999). Developmental coordination disorder in Swedish 7-year-old children. Journal of the American Academy of Child and Adolescent Psychiatry, 38, 820–828; Milberger, S., Faraone, S., Biederman, J., Testa, M., & Tsuang, M. (1996). New phenotype definition of attention deficit hyperactivity disorder in relatives for genetic analyses. American Journal of Medical Genetics, 67, 369–377]. The rate of comorbidity between the two has been found to be close to 50% [Barkley, R. (1990). Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford Press]. Investigations into the comorbidity of the disorders points to a shared aetiology between them. The aim of the present investigation was to examine the extent to which the shared aetiology is due to common genetic factors to both disorders. We also investigated whether particular subtypes of each disorder were more linked than others. Mailed questionnaires were completed by parents (predominantly mothers) of 1285 twin pairs aged 5 and 16 years from the volunteer Australian Twin Registry (ATR). Included were a DSM-IV-based ADHD form, the alternative SWAN (Strengths and Weaknesses of ADHD Symptoms and Normal Behaviour scale) and the Developmental Coordination Disorder Questionnaire (DCDQ). Statistical analyses including structural equation modelling were carried out to explore the genetic factors of both disorders. The modelling showed a strong shared additive genetic component between most subtypes of ADHD and DCD to the subtypes of the other disorder. Analyses comparing the two ADHD measures showed an overlap of the symptoms captured by each measure but also significant differences. The DCD-fine motor and ADHD-Inattentive were most strongly linked using the

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DSM-IV based scale. On the SWAN scale the results were similar but the general coordination scale was also very strongly linked. Implications for the use of different assessment tools are discussed.

PsycINFO classification: 2330; 2346; 2510

Keywords: Developmental coordination disorder; Attention deficit hyperactivity disorder; Twins; Genetics; Comorbidity

1. Introduction

Attention deficit hyperactivity disorder (ADHD) and developmental coordination disorder (DCD) are both childhood disorders identified in the DSM-IV (APA, 1994) which have a population prevalence of approximately 7% (Kadesjö & Gillberg, 1999; Milberger, Faraone, Biederman, Testa, & Tsuang, 1996). Studies investigating ADHD have found that around 50% of ADHD cases also have motor problems severe enough to be diagnosed as DCD (Barkley, 1990; Piek, Pitcher, & Hay, 1999; Pitcher, Piek, & Hay, 2003). Further, children initially diagnosed with DCD have been found to also meet moderate to severe diagnosis for ADHD (Kadesjö & Gillberg, 1999).

Given the joint evolution of ADHD and DCD from the Minimal Brain Dysfunction classification, it is not surprising that these two disorders may be associated (Piek et al., 1999). Not only are they found together, but many studies report a higher prevalence of boys compared with girls for both ADHD (Eiraldi, Power, & Nezu, 1997; Pennington & Ozonoff, 1996) and DCD (e.g., Henderson & Hall, 1982; Kadesjö & Gillberg, 1999). However other studies suggest there are equal numbers of boys and girls affected in DCD (e.g., Hoare & Larkin, 1991). Also, both disorders have been linked to psychosocial problems such as socially inappropriate behaviour, emotional problems, reduced academic performance, and reading and spelling difficulties (Levy, Hay, Bennett, & McStephen, 2005; Taylor et al., 2004). Furthermore, when ADHD and DCD are comorbid, the outcome tends to be more severe than when each disorder occurs alone (e.g., Gillberg, 1992; Pitcher et al., 2003; Visser, 2003). This implies that there may be an exclusive shared aetiology of the comorbidity which is distinct from the factors influencing either of the separate disorders.

One of the difficulties in exploring the comorbidity of these two disorders is the difference between the bodies of research existing for each disorder. ADHD has been extensively researched (Voeller, 2004) with many genetic studies confirming the high heritability of ADHD and its subtypes (Levy & Hay, 2001). Several candidate loci, such as DAT1 (Chen et al., 2003), DRD4 (Langley et al., 2004), and MAO (Payton et al., 2001) have been identified and replicated. There are also many well developed measures in questionnaire or interview form and numerous neuropsychological measures, all of which are comprehensively covered in the recently revised European guidelines (Taylor et al., 2004). DCD, however, is much less extensively researched and what literature exists on it and its aetiology is often confusing. This confusion is often due to varying selection criteria, such as different cut-off scores being used in different studies (Piek & Edwards, 1997), and also due to the overlap that many symptoms of DCD have with other disorders such as learning difficulties (Peters, Barnett, & Henderson, 2001). Performance tests such as the Movement Assessment Battery for Children (Henderson & Sugden, 1992) and the
McCarron Assessment of Neuromuscular Development (McCarron, 1997) are generally used to identify DCD. Recently, screening tools have been developed, such as the Developmental Coordination Disorder Questionnaire (Wilson, Kaplan, Crawford, Campbell, & Dewey, 2000), which allows the testing of much larger samples of children.

Although the link between ADHD and motor problems has been recognised for many years, there have been few studies that have investigated the motor problems in relation to the three distinct subtypes identified by the DSM-IV (APA, 1994). These subtypes are based on the behavioural symptomatology identified in the child; either predominantly inattentive (ADHD-PI), predominantly hyperactive/impulsive (ADHD-HI), or a combination of both types of symptoms (ADHD-C). One link that appears to have been established is that between inattentive symptomatology and poor motor skills, in particular fine motor control (McGee, Williams, & Feehan, 1992; Piek et al., 1999; Pitcher et al., 2003). There is also evidence that suggests that gross motor deficits are more likely to occur in ADHD-C compared to the ADHD-PI (Piek et al., 1999). Differences in the prevalence of boys and girls for different subtypes also suggests a greater relationship between DCD and ADHD-PI, as a greater proportion of girls are found with ADHD-PI compared with ADHD-C (Lahey et al., 1994).

The aim of the current study was to take validated questionnaire measures for both ADHD and DCD and look for a shared genetic heritability to both of them using quantitative genetic methodology in a very large twin sample. While this methodology is powerful, it does require extremely large sample sizes (Neale & Cardon, 1992). It can answer the question of why the two disorders co-occur at such a high frequency and with a different outcome than either disorder alone. By studying the heritability in this way we will be able to tease apart the genetic influences from the environmental effects acting upon the disorders to better understand how they come about. From looking at the comorbid disorder we can examine whether it is a general link between ADHD and DCD or whether particular subtypes of each are linked more strongly with each other. Specific comorbidities may point to particular aetiological pathways which can then be explored more fully. By studying the subtypes of the disorders we can also examine whether there are different factors influencing each one as well as common factors which act on all of them.

Different screening measures of ADHD were also tested against each other for equivalence and to explore the extent that different ADHD measures have on finding shared heritability between it and DCD. There has been much concern that different measures of ADHD may yield different genetic results. For example on some questionnaires, particularly short ones such as the Rutter A, parents may exaggerate differences between dizygotic twins and thus inflate the heritability (Thapar, Harrington, Ross, & McGuffin, 2000).

2. Method

2.1. Participants

The participants consisted of 1285 families of twins from the Australian Twin ADHD Project (ATAP) summarised in Levy and Hay (2001). Families were ascertained from the Australian Twin Registry (ATR: http://www.twins.org.au), a nation-wide, volunteer based database of twins and higher order multiple birth families born in Australia. Each family was sent a questionnaire package for returning by pre-paid mail. The project was approved by Curtin University Human Research Ethics Committee and by the Australian Twin Registry.
The twin pairs were aged 5–16 years. There were 588 MZ pairs, 667 DZ pairs, and 30 pairs for which zygosity could not be assigned (who were excluded from further analyses and are not included in the following counts). There were 472 pairs of male twins of which 289 were MZ and 183 were DZ. There were 473 female pairs of which 299 were MZ and 174 were DZ. There were 310 male/female pairs. This means there were 1254 males and 1256 females.

2.2. Measures

2.2.1. Zygosity

Zygosity was assigned using the following process. First the parents were asked if there had been DNA testing to determine zygosity. If they answered no, then a twin similarity questionnaire of demonstrated validity (Cohen, Dibble, Grawe, & Pollin, 1975; Nichols & Bilbro, 1966) was used as results from such questionnaires have been shown to have good agreement with results from tests using blood or genetic markers (McGuffin, Owen, O’Donovan, Thapar, & Gottesman, 1994). The questionnaire described by Levy and Hay (2001) contains 12 questions such as, “Does their mother ever confuse them in appearance?” “Do they have very similar personalities?” and, “Do they have the same blood group?” to assess how similar the twins appear to be in terms of appearance, personality, and biology. If zygosity still could not be assigned then a question asking the parents whether they thought their twins were monozygotic or dizygotic was used. All opposite sex pairs were assigned as dizygotic.

2.2.2. Australian twin behaviour rating scale (ATBRS)

ADHD symptoms were assessed using the ATBRS, (Levy, Hay, McLaughlin, Wood, & Waldman, 1996; Levy, Hay, Waldman, & McStephen, 2001, Chapters 2 and 3), a parent report questionnaire containing 18 items assessing DSM-IV criteria for ADHD as observed in the child over the last 12 months. It contains items such as, “Has difficulty keeping attention on work or games”, and, “Has difficulty organising tasks or activities” which follow the language of the diagnostic items in DSM-IV. For each item, responses could range from, “Not at all” (scored as 0) to, “Very much/often” (scored as 3). The scores are then used to define whether each child is affected and with which subtype (inattentive, hyperactive–impulsive, or combined). This method is consistent with the procedures adopted in other studies (e.g., Lahey et al., 1998), and has produced similar estimates of the prevalence of the three subtypes, and of the latent structure, when comparing Australian and Missouri samples (Rasmussen et al., 2002). Studies using DSM-III-R ADHD criteria with a previous version of the ATBRS have reported kappa coefficients of .561 to .648 and an alpha coefficient of .86 (Levy, Hay, McStephen, Wood, & Waldman, 1997). This measure has been found to be a reliable measure of the presence of inattentive symptoms ($r = .93$) and hyperactive/impulsive symptoms ($r = .95$) (Levy et al., 2001). If anything, this questionnaire is conservative, with parents identifying more ADHD symptoms in face-to-face interviews (Levy et al., 1997).

2.2.3. Strengths and weaknesses of ADHD symptoms and normal behaviour (SWAN)

ADHD symptoms were also assessed using the SWAN (Swanson et al., 2001), an 18 item parent report questionnaire with observations based on the last month. It contains items such as, “Organise tasks and activities”, and, “Ignore extraneous stimuli”, which
while pointing to behaviours underlying ADHD, are phrased quite differently to the items in DSM-IV. For each item the responses range from “Far below average” (scored as +3) to “Far above average” (scored as −3) and the scores totalled in a similar way to the ATBRS above to define the affected status of each child. Unlike the above scale which uses a fixed score for the cut-off between affected and unaffected, the cut-off is calculated from the distribution of scores using:

\[
\text{Cut-off score} = \text{Mean} \pm (1.65 \times \text{standard deviation})
\]

As this is a relatively new scale there are not yet many results detailing the reliability of this scale but preliminary results point to this being an efficient measure. An important difference between this and most other measures is that this is scored from −3 to 3. This produces a normal distribution of scores which makes subsequent analysis much easier as it alleviates the need for transformation to normality which the results from most other measures require.

2.2.4. Developmental coordination disorder questionnaire (DCDQ)

DCD symptoms were assessed using the DCDQ (Wilson et al., 2000). In addition to the full scale, this questionnaire provides four subtypes, namely control during movement, fine motor/handwriting, gross motor/planning, and general coordination. This is a relatively new measure and this study is the largest dataset to have used it to date. The scale contains 17 items such as “Throws a ball in a controlled and accurate fashion, compared to other children the same age”, “Learned to ride a bike later than his/her friends”, and “Runs easily and smoothly, and stops with control”. It is rated on a 5-point scale ranging from “Not at all like this child” (scored as 1) to “Extremely like this child” (scored as 5). For the last seven items the scores are reversed so that the response, “Not at all like this child” is scored as 5 and vice versa. The total score of the scale is 85 with lower scores indicating affectedness, i.e. scores of 17–48 are affected, 49–57 are suspect, and 58–85 are non-DCD (Wilson et al., 2000). Because the scale was being incorporated into a larger questionnaire, the decision was made for the convenience of the family member completing the form to change it to the same 4-point scale as the ADHD questions, omitting the middle category and using a 1,2,4,5 coding for comparability.

Before any analysis was performed the inter-item reliability of the scale was tested using SPSS. Cronbach’s alpha was .88 for the full scale and for each item if deleted ranged from .86 to .88. The acceptable value for Cronbach’s alpha is .70 and hence the scale shows good reliability. Concurrent validity has been established by correlating the scores on the DCDQ with other test measures, namely, the Bruininks–Oseretsky Test of Motor Performance \((r = .46–.54, p < .0001)\) and the Movement Assessment Battery for children \((r = −.59, p < .0001)\) (Wilson et al., 2000). Construct validity was demonstrated by Wilson and colleagues, as they found significant differences in scores between a DCD group, a suspect DCD group and a non-DCD group.

2.3. Analyses

Exploratory analysis of the data was performed using SPSS (SPSS Incorporated, 1999). Point prevalences were calculated to find the proportion of our sample affected by ADHD as defined by the published cut-offs for the DSM-IV and DCD scales. Prevalences were also calculated for all three scales using the formula contained in literature for the SWAN
scale (Swanson et al., 2001), namely mean ± 1.65sd. Differences in groups defined by zygosity, age and sex were then investigated and regression and Mann–Whitney tests (Greene & D’Oliveira, 1993; McGuffin, Shanks, & Hodgson, 1984) were performed to explore the significance of any differences found. Only sex differences were found and so the data were standardised by sex as described below.

The raw scores for the ATBRS were very negatively skewed with a ‘floor effect’ whereby a high proportion of the sample had low scores. To achieve a closer approximation to normality, the data were transformed by taking the logarithm of each score. The DCDQ data was the opposite with a high proportion of the sample scoring very highly. These data were transformed using the reflect and inverse method to achieve a distribution closer to normal. The reflection involves finding the highest value and adding one, then every value is subtracted from this to form the new variable. The inversion involves dividing 1 by the new variable. No transformation was required for the SWAN scale scores.

All model fitting was performed using the Mx package (Neale, 1997). First univariate modelling was performed on each subtype, and the total scores of each scale. The model fitting was performed using the logic as laid out in Neale and Cardon (1992) whereby first a model containing variance components for additive genetics (A), common environment (C) and non-shared environment (E) is examined then nested sub-models are trialled and compared for best fit. If two models have a similar fit to the data then the simpler one (involving fewer variables and degrees of freedom in the model fitting) is accepted for parsimony. For example, first the ACE model is fitted and the $\chi^2$ and $p$-values noted. The AE model is then tested and if the $\chi^2$ for the AE model does not appreciably reduce the fit, given the extra degree of freedom, then the AE is accepted to be the better fitting model. This would be repeated with the CE model and the values again compared to see if it fitted better than the AE or ACE model. The results of the model fitting give the heritability with respect to the components being tested in the model, e.g., a heritability of 60% comprises an additive genetic component of 60% and 40% common plus non-shared environment. The non-shared environment includes any error of measurement and is thus in the absence of any real environmental effects, an estimate of reliability.

Next bivariate model fitting was performed on the DCDQ data against the ADHD data of the other scales, using the relation of twin 1’s ADHD score to twin 2’s DCDQ score and vice versa. This was performed to explore any shared and exclusive inheritance between DCD and ADHD symptoms as measured by the different scales. The same logic was followed for the fitting of bivariate models as for the univariate but with the added complexity of examining variance components between DCD and ADHD in addition to within each disorder. Finally bivariate modelling was performed on the two ADHD scales to explore any similarities and differences in the data collected by the scales. The interpretation of bivariate modelling is described in Levy and Hay (2001) and Fig. 1 described below gives a graphical example with these data.

3. Results

3.1. Prevalence

The prevalence of DCD using the published Canadian cut-off (Wilson et al., 2000) was 2% which was rather low compared with previous estimates of between 5% and 19% (APA, 1994; Henderson & Sugden, 1992; Kadesjö & Gillberg, 1999; Wright & Sugden,
Using the mean ± 1.65sd method as used for the SWAN scale it was 8% which more closely approximates the prevalence given in DSM-IV of 6% (APA, 1994) and the recent Australian national survey (Graetz, Sawyer, Hazell, Arney, & Baghurst, 2001). Of this 8%, 145 were boys and 111 were girls (i.e., 56.6% were boys).

The prevalence of ADHD was split into its subtypes for both the ATBRS and SWAN data as shown in Tables 1a and 1b below. For the ATBRS data, responding “Pretty much/
Often” or “Very much/Often” to six or more items of inattentive and/or hyperactive–impulsive symptoms followed the DSM-IV classification (APA, 1994). The tables indicate there is an approximately 3:1 ratio of boys:girls affected across the scales and subtypes.

3.2. Age, sex and zygosity

Regression was performed by age for each scale in order to determine any age effects. The tests were performed separately for each sex and zygosity in addition to the whole sample. For all the tests $r^2$ ranged from −.001 to .000 and the ANOVA $p$ from .343 to .769 indicated no detectable age effects.

Mann–Whitney tests were performed for zygosity and sex effects. Again the tests were performed separately on the subgroups and the whole sample. For zygosity there were no effects with $p$-values ranging from .147 to .969. For sex all scales apart from gross motor planning showed a significant effect (for gross motor planning $p = .883$, for DCD full scale $p = .011$, for all SWAN and ATBRS scales – ADHD-PI, ADHD-HI, ADHD-C – and DCDQ control during movement, fine motor control, and general coordination $p < .001$). These tests showed that males had significantly higher mean scores on all of the ATBRS scales, and also on all of the SWAN scales. For the DCDQ scales males scored significantly higher only on the control during movement. For fine motor/handwriting, general coordination, and the full scale (including all subscales) females scored higher. All scores were then standardised by sex and the test performed again to check that the sex effects had been removed prior to modelling.

3.3. Univariate modelling

The results of the univariate modelling on ADHD symptoms are shown in Tables 2 and 3. For the ATBRS data (Table 2), it can be seen that for inattentive (ADHD-I) and hyperactive–impulsive (ADHD-HI) subtypes, the AE model which is the best fit but for combined (ADHD-C) type the ACE model is best, although common environment makes a relatively small contribution compared to additive genetics (for the ACE model, the $\chi^2$ is zero as the fitting of three parameters saturates the model). A similar pattern is observed for the data measured using the SWAN (Table 3) except that the magnitude of the additive

Table 2
Univariate best-fitting models for the three ADHD subtypes using the ATBRS

<table>
<thead>
<tr>
<th>ATBRS scale</th>
<th>A</th>
<th>C</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD-I, $p = 1.00$, $\chi^2 = 0$</td>
<td>.88</td>
<td></td>
<td>.11</td>
</tr>
<tr>
<td>ADHD-HI, $p = .95$, $\chi^2 = 0$</td>
<td>.85</td>
<td></td>
<td>.14</td>
</tr>
<tr>
<td>ADHD-C, $p = 1.00$, $\chi^2 = 0$</td>
<td>.76</td>
<td>.13</td>
<td>.11</td>
</tr>
</tbody>
</table>

Table 3
Univariate best-fitting models for the three ADHD subtypes using the SWAN rating scale

<table>
<thead>
<tr>
<th>SWAN scale</th>
<th>A</th>
<th>C</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADHD-I, $p = .90$, $\chi^2 = 1.10$</td>
<td>.92</td>
<td></td>
<td>.09</td>
</tr>
<tr>
<td>ADHD-HI, $p = .89$, $\chi^2 = 1.14$</td>
<td>.98</td>
<td></td>
<td>.02</td>
</tr>
<tr>
<td>ADHD-C, $p = 1.00$, $\chi^2 = 0$</td>
<td>.74</td>
<td>.21</td>
<td>.05</td>
</tr>
</tbody>
</table>
genetic component is larger for all but combined type. Also, for the combined subtype the common environment component is larger for the SWAN than the ATBRS data.

The results for the univariate modelling on the DCD data are shown in Table 4. All of the subscales show a strong additive genetic component in either an AE or ACE model. Interestingly all models for which an ACE model was the best fit had a common environment component of 20%. The general coordination is the only subscale for which a common environmental component was not found.

3.4. Bivariate modelling

Each of the subscales of the DCDQ was modelled separately against each of the ADHD subtypes for both the ATBRS and SWAN scales.

3.4.1. Control during movement

Table 5 shows that the results for the two different ADHD measures are very different. The SWAN detects a shared genetic factor between all ADHD subtypes and control during movement, whereas the ATBRS only shows one for the inattentive subtype. Fig. 1 illustrates the results of the bivariate model fitting, using the extreme example of the SWAN Inattention scale and the DCD control during movement. While the control during movement had a relatively high univariate heritability of .72 (Table 4), in fact all of this was shared with the SWAN scale and there were no genetic effects specific to the DCD scale. To summarise the data in the table, the shared aetiology of control during movement and ADHD is predominantly due to shared common environmental effects when ADHD is measured by the ATBRS or principally due to shared additive genetic effects when measured by the SWAN.

| Table 4 |
| Univariate best-fitting models for each of the components of the DCDQ |

<table>
<thead>
<tr>
<th>DCD scale</th>
<th>A</th>
<th>C</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control during movement, $p = 1.00$, $\chi^2 = 0$</td>
<td>.72</td>
<td>.20</td>
<td>.07</td>
</tr>
<tr>
<td>Fine motor/handwriting, $p = 1.00$, $\chi^2 = 0$</td>
<td>.64</td>
<td>.20</td>
<td>.14</td>
</tr>
<tr>
<td>Gross motor/planning, $p = 1.00$, $\chi^2 = 0$</td>
<td>.71</td>
<td>.20</td>
<td>.08</td>
</tr>
<tr>
<td>General coordination, $p = .95$, $\chi^2 = .712$</td>
<td>.85</td>
<td>.20</td>
<td>.16</td>
</tr>
<tr>
<td>Full scale, $p = 1.00$, $\chi^2 = 0$</td>
<td>.69</td>
<td>.20</td>
<td>.11</td>
</tr>
</tbody>
</table>

| Table 5 |
| Best-fitting bivariate models for ADHD and DCD control during movement |

<table>
<thead>
<tr>
<th>Control during movement</th>
<th>Shared DCD only</th>
<th>ADHD only</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>C</td>
</tr>
<tr>
<td>ATBRS ADHD-I, $p = .04$, $\chi^2 = 21.89$</td>
<td>.12</td>
<td>.22</td>
</tr>
<tr>
<td>ATBRS ADHD-HI, $p = .86$, $\chi^2 = 7.82$</td>
<td>–</td>
<td>.20</td>
</tr>
<tr>
<td>ATBRS ADHD-C, $p = .52$, $\chi^2 = 12.09$</td>
<td>–</td>
<td>.22</td>
</tr>
<tr>
<td>SWAN ADHD-I, $p = .75$, $\chi^2 = 9.36$</td>
<td>.72</td>
<td>–</td>
</tr>
<tr>
<td>SWAN ADHD-HI, $p = .64$, $\chi^2 = 8.79$</td>
<td>.26</td>
<td>.20</td>
</tr>
<tr>
<td>SWAN ADHD-C, $p = .49$, $\chi^2 = 10.50$</td>
<td>.36</td>
<td>–</td>
</tr>
</tbody>
</table>
3.4.2. Fine motor/handwriting

Table 6 demonstrates that both ADHD measures show a strong additive genetic component to the shared aetiology between ADHD and fine motor/handwriting although it is generally larger for the ATBRS. In addition, most of the heritability for the DCD subtype is included in the shared component on ATBRS whereas both have sizeable components of their own on the SWAN scale.

3.4.3. Gross motor/planning subscale

As can be seen in Table 7, significant correlations were only found between gross motor planning and inattention and combined type ADHD measured on the ATBRS. In both models, the shared aetiology was due to shared common environmental effects with both ADHD and DCD having substantial separate additive genetic components.

3.4.4. General coordination

As the shared components are fairly small (Table 8), this implies that general coordination and ADHD are predominantly separate disorders.

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Table 6
Best-fitting bivariate models for ADHD and DCD fine motor/handwriting

<table>
<thead>
<tr>
<th>Fine motor/handwriting</th>
<th>Shared</th>
<th>DCD only</th>
<th>ADHD only</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>C</td>
<td>E</td>
</tr>
<tr>
<td>ATBRS ADHD-I, ( p = .94, \chi^2 = 6.16 )</td>
<td>.66</td>
<td>.20</td>
<td>.06</td>
</tr>
<tr>
<td>ATBRS ADHD-HI, ( p = 1.00, \chi^2 = 2.51 )</td>
<td>.31</td>
<td>–</td>
<td>.01</td>
</tr>
<tr>
<td>ATBRS ADHD-C, ( p = .92, \chi^2 = 5.91 )</td>
<td>.64</td>
<td>.21</td>
<td>.06</td>
</tr>
<tr>
<td>SWAN ADHD-I, ( p = .94, \chi^2 = 5.42 )</td>
<td>.25</td>
<td>–</td>
<td>.01</td>
</tr>
<tr>
<td>SWAN ADHD-HI, ( p = .72, \chi^2 = 7.93 )</td>
<td>.35</td>
<td>.20</td>
<td>.01</td>
</tr>
<tr>
<td>SWAN ADHD-C, ( p = .98, \chi^2 = 3.08 )</td>
<td>.40</td>
<td>.05</td>
<td>.02</td>
</tr>
</tbody>
</table>

Table 7
ADHD and DCD gross motor/planning bivariate best-fitting models

<table>
<thead>
<tr>
<th>Gross motor/planning</th>
<th>Shared</th>
<th>DCD only</th>
<th>ADHD only</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>C</td>
<td>E</td>
</tr>
<tr>
<td>ATBRS ADHD-I, ( p = .99, \chi^2 = 4.38 )</td>
<td>–</td>
<td>.21</td>
<td>.03</td>
</tr>
<tr>
<td>ATBRS ADHD-C, ( p = .95, \chi^2 = 5.11 )</td>
<td>–</td>
<td>.20</td>
<td>.03</td>
</tr>
</tbody>
</table>

Table 8
ADHD and DCD general coordination bivariate best-fitting models

<table>
<thead>
<tr>
<th>General coordination</th>
<th>Shared</th>
<th>DCD only</th>
<th>ADHD only</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>C</td>
<td>E</td>
</tr>
<tr>
<td>ATBRS ADHD-I, ( p = .56, \chi^2 = 10.69 )</td>
<td>.23</td>
<td>.04</td>
<td>.03</td>
</tr>
<tr>
<td>ATBRS ADHD-HI, ( p = .43, \chi^2 = 12.15 )</td>
<td>.16</td>
<td>.06</td>
<td>.00</td>
</tr>
<tr>
<td>ATBRS ADHD-C, ( p = .79, \chi^2 = 7.97 )</td>
<td>.16</td>
<td>.05</td>
<td>.02</td>
</tr>
<tr>
<td>SWAN ADHD-I, ( p = .92, \chi^2 = 6.70 )</td>
<td>.42</td>
<td>–</td>
<td>.07</td>
</tr>
<tr>
<td>SWAN ADHD-HI, ( p = 1.00, \chi^2 = 2.63 )</td>
<td>.04</td>
<td>.02</td>
<td>.00</td>
</tr>
<tr>
<td>SWAN ADHD-C, ( p = .95, \chi^2 = 4.61 )</td>
<td>.10</td>
<td>.03</td>
<td>.01</td>
</tr>
</tbody>
</table>
The full DCD scale and ADHD were modelled. The results are shown in Table 9. For both ADHD measures there is a substantial shared aetiology due to both additive genetic and common environmental factors. Both disorders also have their own substantial additive genetic factors.

Finally the ATBRS and SWAN scales were modelled against each other. The best-fitting model was AE ($p = .59, \chi^2 = 10.30$) with shared $A$ and $E$ being .44 and .04 respectively. The ATBRS scale then had $A$, $C$, and $E$ of .29, .20, and .04 respectively and the SWAN scale had .28, .24, and .01. This shows that there is a substantial overlap between the scales (44%) of the additive genetic component to the two scales. Both scales then have their own additive genetic and common environmental factors. Although there is an overlap between the scales, there are also differences in what each one detects.

4. Discussion

The prevalence of both disorders was found to be comparable to those of previous studies. We found a higher rate of ADHD symptoms in boys than girls with a ratio of approximately 3:1 which is comparable to that found in previous studies.

The univariate analyses demonstrated that all of the subscales of ADHD and DCD have a substantial genetic component. For the ADHD data, both the SWAN and the ATBRS showed similar patterns with the inattentive and hyperactive/impulsive subtypes having substantial additive genetic factors while the combined subtype also has a common environment component. Of the DCD subtypes, only general coordination did not have a common environment component with all the subscales having a substantial additive genetic component.

The bivariate analyses showed that for the SWAN data, the inattention subscale is most closely associated with the control during movement scale of DCD (shared heritability of 72%) than any other subscales for either disorder. For ATBRS data it is inattention and fine motor/handwriting which are most closely associated with a joint heritability of 66%. This close relationship between fine motor ability and inattention has been identified in previous research (McGee et al., 1992; Piek et al., 1999; Pitcher et al., 2003) but never at the genetic level. For most of the other subtypes of DCD and ADHD on either scale the shared heritabilities were modest with no contribution found at all for gross motor/planning and the inattention and combined scales of ADHD, despite very substantial univariate genetic contributions to both this DCD and these ADHD scales.

It is obvious the pattern of the relationships between ADHD depend both upon the measure of ADHD and the specific subscale of the DCDQ. The results cannot be
dismissed as merely the consequence of the use of inaccurate rating scales, given the high Cronbach’s alpha for the DCDQ subscales and the small non-shared environmental component (e) for all the measures, which by definition includes any error of measurement.

Although it would be ideal to determine diagnoses through clinical interview in the case of ADHD and performance testing for DCD, this was not possible given the large sample of children needed for genetic analysis. Indeed, even this sample was too small to take the analysis the next step and to examine for example the structure of the four DCD subscales and the causes of their interrelationship. This sample is planned to increase fourfold to close to 6000 families, by which stage much more comprehensive analysis will be possible. However, the screening tools used in this study produced prevalence estimates approximately equivalent to those found in previous studies, suggesting that these screening tools are appropriate for identification of these disorders.

The Canadian DCDQ cut-off scores produced a much lower DCD rate than that based on the standard deviation cut-off score, and the reason for this is unknown. It may be a real difference between the countries, or an artefact of the reduction from a five to a four-point classification here to make the DCDQ consistent with the other items in the Australian ATAP battery. Alternatively it could be a consequence of recruitment or response bias. In the present study the families were asked to participate, partly to update the information on school-age multiples in our large website on this topic (www.twinsandmultiples.org) and the DCDQ items were embedded among many other questions on child development. There was no focus on DCD that could have led to families of children with motor control problems being more likely to complete the questionnaire.

The difference may also be due to real differences in the severity of DCD in the two countries. Our initial 2% using the published cut-offs may have only included the severely affected, whereas the 8% may have included children with only mild symptoms. Future work will explore this in addition to modelling extreme high scorers separately to those lesser affected to examine aetiological differences.

Of more importance than any difference in prevalence between the two countries and the two questionnaire formats is whether the DCDQ is measuring the same thing in the two samples. One method of doing this is by latent class analysis but the sample was too small to yield reliable estimates, though the potential is there as the sample size increases. In a previous study using an older but larger ATAP cohort, we were able to show the same latent class structure applied as in a Missouri twin cohort (Rasmussen et al., 2002) and even that very similar patterns of genetic estimates were found (Rasmussen et al., 2004), despite the twin samples being recruited in very different ways and the ADHD data being collected in Missouri by phone interview rather than by mailed questionnaire.

Although this would suggest the construct of ADHD may be robust, of concern was the finding that although the comparison of the two ADHD scales showed an overlap between them, there was also a substantial difference in what each scale was detecting. The difference may be due to the questionnaires measuring different underlying factors which on their own show similar characteristics but the differences are amplified in the bivariate analyses. This could be in part due to the structure of the questionnaires in that the ATBRS has responses from “Not at all” to “Very much/Very often” which are unidirectional whereas the Swan has responses from “Far below average” to “Far above average” and so can measure both positive and negative aspects of behaviour. In doing so it seems to have changed the nature of what was being measured away from inattention and hyper-
activity/impulsivity to attention and activity, which may be more relevant concepts for the general population. It is interesting to speculate what would happen if the DCDQ were changed similarly and children identified who were better than average on aspects of motor control. Which measurement technique is more clinically correct will be examined in future work where a subset of our full sample will be assessed, with a formal clinical interview for ADHD and objective measures of DCD. The sample sizes needed for either univariate or bivariate genetic modelling mean that studies such as this one have to be based on questionnaires, with all their possible limitations, rather than the “gold standards” for both ADHD and DCD.

This study represents the first genetic study of DCD, both on its own and in terms of the overlap with ADHD. Two interesting issues arise. What is the C (common family environment) contribution to the determinants of DCD? It could be shared perinatal experiences such as preterm birth, remembering this sample is of twins who have to be born at the same gestation. It could well be to do with nutrition or shared patterns of exercise. This complicates the interpretation of the recent Dutch study (Slaats-Willemse, De Sonnevile, Swabb-Barneveld, & Buitelaar, 2005), which claimed fine motor problems may be a genetic marker for susceptibility to ADHD on the basis that non-ADHD children shared some of the motor control problems of their ADHD sibling. The C does mean these children are sharing relevant family environment factors as well as genes.

The second issue concerns the degree of genetic overlap between ADHD and DCD. If one were looking for common biological bases to some of the connections between these conditions, it would be much more appropriate to look, say, at that between DCD control during movement and SWAN inattention (Fig. 1) where there is no genetic variance unique to the DCD measure, compared with gross motor control and inattentive ADHD where there is no genetic overlap (Table 7). This latter finding is consistent with recent evidence of different cognitive processes being disrupted in the different disorders. For example, there is evidence that children with DCD do not have difficulties with response inhibition although this appears to be a major deficit in children with ADHD (Piek et al., 2004). Furthermore, children with DCD have been identified with difficulties in visual-spatial organisation although children with ADHD do not appear to have difficulties with these (Piek & Pitcher, 2004).

While this is the first genetic study of DCD, the significant heritabilities for all the scales would suggest that in a few years there may be the same body of genetic evidence as exists for ADHD (Levy & Hay, 2001) and the same growing excitement over the identification of specific genes (Langley et al., 2004).

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References


