Depressive Symptomatology in Child and Adolescent Twins With Attention-Deficit Hyperactivity Disorder and/or Developmental Coordination Disorder

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Previous research has demonstrated a link between attention-deficit/hyperactivity disorder (ADHD), developmental coordination disorder (DCD), and depression. The present study utilized a monozygotic (MZ) differences design to investigate differences in depressive symptomatology between MZ twins discordant for ADHD or DCD. This extends previous research as it controls for genetic effects and shared environmental influences and enables the investigation of nonshared environmental influences. In addition, children and adolescents with comorbid ADHD and DCD were compared on their level of depressive symptomatology to those with ADHD only, DCD only, and no ADHD or DCD. The parent-rated Strengths and Weaknesses of ADHD Symptoms and Normal Behavior, Developmental Coordination Disorder Questionnaire, and Sad Affect Scale were used to assess ADHD, DCD, and depressive symptomatology respectively. The results revealed higher levels of depressive symptomatology in MZ twins with ADHD or DCD compared to their nonaffected co-twins. In addition, children and adolescents with comorbid ADHD and DCD demonstrated higher levels of depressive symptomatology compared to those with ADHD only, DCD only, and no ADHD or DCD. The implications of these findings are discussed with emphasis on understanding and recognizing the relationship between ADHD, DCD, and depression in the assessment and intervention for children and adolescents with these disorders.

It is well recognized that depressive symptoms and depressive disorders represent significant mental health problems during childhood and adolescence (e.g., Compas et al., 2004). In a recent Australia wide survey, 3% of children and adolescents aged 4 to 17 years met the criteria for depressive disorder (Sawyer et al., 2001).

Depression in children and adolescents has been associated with a number of risk factors, including genetic influences (Rice et al., 2002; Thapar & McGuffin, 1994), low self-esteem (Reinherz et al., 1989), cognitive factors (Cole & Jordan, 1995), and deficits in social skills (Altman & Gotlib, 1988). Genetic studies have demonstrated the importance of both genetic and environmental influences, particularly nonshared intra- and extra-familial environmental experiences (Birmaher et al., 1996; Cytryn & McKnew, 1996). For example, the Nonshared Environment and Adolescent Development (NEAD) Project found that adolescents who experienced more maternal negativity than their sibling were more likely to be depressed, independent from genetic factors and shared family environment (Reiss et al., 1994, as cited in Pike & Plomin, 1996). Clinical and epidemiological studies have shown that depression in childhood and adolescence is highly comorbid with other disorders such as anxiety disorder, substance abuse, attention-deficit/hyperactivity disorder (ADHD), and conduct disorder (Angold & Costello, 1993; Costello et al., 2002; Kovacs & Devlin, 1998; Lewinsohn et al., 1993). It has been suggested that comorbid ADHD and depression complicates symptoms and increases the risk of psychiatric and educational problems (Kewley, 2001), and may result in higher risk for suicide compared with children without such comorbid disorders (Biederman et al., 1991).

ADHD is one of the most commonly diagnosed childhood psychiatric disorders with approximately 3% to 5% of school aged children affected (American

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Psychiatric Association, 1994). Research using an Australian sample of 3597 children and adolescents (aged 6–17 years) reported a prevalence rate of approximately 7.5% (Graetz et al., 2001). ADHD is characterized by symptoms of inattention, impulsivity, and hyperactivity which must be persistent, developmentally inappropriate, and maladaptive (American Psychiatric Association, 1994). Studies employing clinical based samples have shown that children and adolescents with ADHD are more likely to be diagnosed with mood disorders such as depression and anxiety than comparison children (Angold & Costello, 1993; Biederman et al., 1991). Studies involving community samples have also linked ADHD with increased levels of depressive symptoms if not a diagnosis of depression per se (Jensen et al., 1993; Kitchens et al., 1999; LeBlanc & Morin, 2004). A recent twin study examining separation anxiety and generalized anxiety in DZ twins discordant for ADHD also identified the twin with ADHD at greater risk of these disorders than the nonaffected twin (McDougall et al., 2006).

A strong link between ADHD and motor problems has been identified. We found that approximately 50% of children with ADHD also have motor deficits severe enough to be diagnosed as developmental coordination disorder (DCD; Pitcher et al., 2003). We have also identified a genetic link between the two disorders (Martin et al., 2006). The *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; DSM-IV; American Psychiatric Association, 1994) defines DCD as a significant impairment in the development of motor coordination which is diagnosed when a child's motor coordination is markedly inappropriate given the child's age and intellectual ability. These movement difficulties must significantly interfere with the individual's daily life or academic achievement, and are not due to physical or neurological defects (American Psychiatric Association, 1994). According to the DSM-IV, approximately 6% of children aged 5 to 11 years experience motor problems that meet the criteria for DCD.

In addition to movement difficulties, children with DCD exhibit behavioral, conduct, and attentional problems (Gillberg & Gillberg, 1989; Piek et al., 1999). Children and adolescents with DCD have been found to experience feelings of lower self-worth, perceived lower levels of social support, and higher levels of anxiety (Sigurdsson et al., 2002; Skinner & Piek, 2001). However, little research has examined the relationship between DCD and depression. In a recent study, young school age children with DCD perceived significantly higher levels of depressive symptomatology compared to children without poor coordination (Francis & Piek, 2003). Perceived athletic competence was shown to have a direct impact on depression, which emphasises the importance of motor ability on emotional functioning.

The first aim of the present study was to understand the nature of the relationship between ADHD, DCD, and depression by examining the levels of depressive symptomatology in monozygotic (MZ) twins discordant for ADHD or DCD. This is useful given that research on childhood and adolescent depression has identified the influences of both genetic and environmental factors (Rice et al., 2002). The MZ differences design (co-twin control method) is based on the idea that although within-pair similarities between MZ twins can be the result of genetics or postzygotic events (pre-, peri- and postnatal environment), differences between them are generally due to postzygotic events, although in some instances epigenetic factors such as patterns of methylation (Machin, 1996), demethylation and hypermethylation (Reik et al., 2001) may play a role. Consequently, one twin provides a control for examining prenatal and postnatal development, physiology, and life experiences of the co-twin (Phelps et al., 1997). In addition, MZ twins who are reared together also experience the same common or familial environment (Bulik et al., 2001). Therefore, differences between MZ twins, who are reared together, may be attributed to the influence of unique environmental factors (Bulik et al., 2001). Although there have been several family studies examining the influence of common familial vulnerabilities in the relationship between ADHD and mood disorders such as depression (e.g., Faraone & Bierderman, 1997), twin research in this area has been very limited. Based on previous evidence it is hypothesized that the ADHD-only twins will demonstrate significantly higher levels of depressive symptomatology compared to their non-ADHD twins, and the DCD-only twins will demonstrate significantly higher levels of depressive symptomatology compared to their non-DCD twins.

A further aim of the current study was to investigate the relationship between comorbid 'ADHD+DCD' and depression. Studies involving community samples have found that diagnoses of ADHD and DCD often co-occur (Kadesjo & Gillberg, 1999; Piek et al., 1999), with approximately 50% of individuals with ADHD meeting the criteria for DCD and vice versa (Kadesjo & Gillberg, 1999; Pitcher et al., 2003). Gillberg (1995) categorized this overlap between coordination and attention problems as Deficits in Attention, Motor Control and Perception (DAMP). Children with comorbid ADHD and DCD are often at higher risk for psychiatric and personality disorders than controls without ADHD or DCD. For example, Hellgren et al. (1994) reported that more than half of the adolescents with DAMP also had personality or psychiatric disorders (particularly depression), whereas only one tenth of the control group met these diagnoses. A subsequent study was conducted investigating the outcome of these individuals at the age of 22, and results revealed that 58% of the comorbid group had a poorer outcome, with higher rates of psychiatric disorders, drug or alcohol abuse, and low rates of independence compared to those without ADHD or DCD (Rasmussen & Gillberg, 2000).

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These studies have demonstrated a poorer psychosocial outcome (including higher rates of depression) in the DAMP group compared to a non-DAMP control group (i.e., without ADHD or DCD; Hellgren et al., 1994). Some studies have also revealed a worse outcome in the comorbid group compared to an ADHD-only or DCD-only group (e.g., greater school dysfunction; Kadesjo & Gillberg, 1999, 2001). However, these studies have not investigated differences in rates of depression or levels of depressive symptoms. Therefore, we compared the levels of depressive symptomatology in children and adolescents with both ADHD and DCD to those with DCD only, ADHD only, and no ADHD or DCD in a large community sample. As the number of twins who were discordant for comorbid ADHD and DCD was very small, this comparison was carried out on the entire sample. To ensure the samples were independent, first-born and second-born twins were examined in separate analyses. This study extends previous research as it specifically focuses on depressive symptomatology and examines differences between a comorbid group and ADHD-only and DCD-only groups, as well as a control group without ADHD or DCD. It was predicted that the ADHD + DCD group will demonstrate higher levels of depressive symptomatology compared to the DCD-only, ADHD-only group, and no ADHD or DCD group.

Method

Participants

Co-Twin Comparisons

Sixteen pairs of MZ twins discordant for ADHD only were identified using the SWAN (Swanson et al., 2001). Their mean age was 13.12 years with a *SD* of 3.43 (range = 6.44–16.67). There were 12 male pairs and 4 female pairs. Twins who met the cut-offs on the inattentive and/or hyperactivity/impulsivity subscales were classified as having ADHD (11 twins were classified as inattentive type, two were classified as hyperactive/impulsive, and three met criteria for combined type). All those who scored below the cut-offs were assigned to the control twin group. The DCD-Q (Wilson et al., 2000) was used to ensure that the twins did not meet the criteria for DCD.

Twenty-four pairs of MZ twins discordant for DCD-only were identified using the DCD-Q (Wilson et al., 2000). The mean age was 11.91 years with a *SD* of 3.57 (range = 6.45–16.99 years). They consisted of 11 male and 13 female pairs. MZ twins who scored 63 and below (using calculated cut-offs from the distribution of scores) on the DCD-Q were classified as having DCD. The unaffected co-twins who obtained a DCD-Q score of 64 and above were assigned to the control group. The twins were assessed for ADHD symptoms by the SWAN scale in order to ensure they did not meet criteria for ADHD.

There were no significant differences between the MZ twin pairs for either co-twin comparison on birth weight or apgar scores at 1 or 5 minutes.

Full Sample Comparisons

The full sample of 2040 twin pairs was separated into Twin A (first-born) and Twin B (second-born) groups in order to ensure independence of groups. The twins were then classified into ADHD-only, DCD-only, ADHD + DCD, and control groups. The DCD-only and ADHD-only groups were identified using the same calculated cut-offs described above. Twins had to meet the cut-offs for both DCD and ADHD in order to be classified as having comorbid ADHD and DCD (i.e., ADHD + DCD). Twin A sample comprised of 42 inattentive, 10 hyperactive/impulsive, and 19 combined type in the ADHD-only group. The ADHD + DCD group consisted of 20 inattentive and 13 combined. Twin B sample comprised of 47 inattentive, 15 hyperactive/impulsive, and 19 combined type in the ADHD-only group. The ADHD + DCD group consisted of 18 inattentive and 18 combined.

The cut-off scores for both scales was calculated using the mean and standard deviations (Hay et al.,

Table 1

Sample Size, Age, and Sex of the ADHD-only, DCD-only, ADHD+DCD, and Control Groups

		Age			Sex	
Group	*n*	*M*	*SD*	Range	Males	Females
Twin A						
ADHD only	71	14.08	2.78	6.44–17.49	52	19
DCD only	92	12.26	3.65	6.45–18.02	46	46
ADHD + DCD	33	12.85	3.45	6.52–18.09	23	10
Control	145	14.68	2.71	6.64–18.51	47	98
Twin B						
ADHD only	81	13.95	2.73	6.41–17.49	64	17
DCD only	100	12.12	3.55	6.44–18.29	52	48
ADHD + DCD	36	13.28	3.76	6.52–18.09	27	9
Control	134	14.67	2.45	7.10–18.70	53	81

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2007; Swanson et al., 2001). For the SWAN scale the cut-off was defined as, mean + 1.65 *SD* (as a low score indicates unaffected status) while for the DCD-Q it was defined as mean – 1.65 *SD* (as a high score indicates unaffected status). The control group was defined by those who scored equal to or below –2 ('above average' to 'far above average') on all items of the hyperactive/impulsive and inattentive scales of the SWAN, and scored equal to or above 80 on the DCD-Q.

These cut-offs were chosen in order to ensure that the control group demonstrated minimal DCD and ADHD symptoms. The characteristics of the groups are presented in Table 1.

A one-way independent group analysis of variance (ANOVA) was conducted to determine whether the groups differed in terms of age and sex. There was a statistically significant difference in age between groups in the Twin A, $F(3, 335) = 12.72$, $p < .001$, and Twin B, $F(3, 343) = 14.02$, $p < .001$, samples. Furthermore, there was a statistically significant difference in sex between groups for the Twin A, $F(3, 336) = 14.57$, $p < .001$, and Twin B, $F(3, 346) = 14.33$, $p < .001$, samples.

Measures

Zygosity

Parents were asked whether zygosity had been previously determined by a DNA or blood test. If the twins had not been tested, parents were asked to complete a twin similarity questionnaire (Cohen et al., 1975). This scale had six questions on similarity of features and six on frequency of confusion by the mother. A description of this scale can be found in Hay et al. (2001). Such questionnaires have demonstrated validity and have shown to have good agreement with results from blood or DNA tests (McGuffin et al., 1994).

Strengths and Weaknesses of ADHD Symptoms and Normal Behavior (SWAN)

ADHD symptoms were assessed using the parent-rated SWAN scale (Swanson et al., 2001) which is based on the 18 ADHD symptoms listed in the DSM-IV manual and involves observations based on the last month with reference to other children of the same age. Scoring for each item ranges from 'Far below average' (scored as +3) to 'Far above average' (scored as –3) in order to reflect both strengths and weaknesses. The scores are totalled and then divided by 9 for the inattention and hyperactivity/impulsivity scale, or by 18 for the combined subscale (resulting in an average score for each subtype). The cut-offs between affected and unaffected for inattention and hyperactivity/impulsivity are calculated from the distribution of scores using:

Cut-off score = Mean + (1.65 × standard deviation; Martin et al., 2006)

The calculated cut-offs for the inattention and hyperactivity/impulsivity subtypes were 1.11 and 1.03 respectively. In order the meet a diagnosis for the combined subtype, the cut-offs for both the inattention and hyperactivity/impulsivity had to be met.

Martin et al. (2006) found the prevalence rate of ADHD, as assessed by the SWAN, to be comparable to those reported in previous studies. Hay et al. (2007) found the SWAN to be a more accurate reflection of the ADHD phenotype than DSM-IV based scales. In the present study, Cronbach's alpha was .95 for both the inattention and hyperactive/impulsive scales, demonstrating good internal reliability.

Developmental Coordination Disorder Questionnaire (DCD-Q)

The DCD-Q (Wilson et al., 2000) is a 17-item parent-rated questionnaire, designed to differentiate between children with and without motor problems. The questionnaire includes four subtypes, namely, general coordination, control during movement, gross motor/planning, and fine motor/handwriting as revealed by previous factor analysis using clinical and community samples. This study utilized a similar cut-off calculation to the SWAN (Martin et al., 2006). The cut-off between affected and unaffected is calculated from the distribution of scores using:

Cut-off score = Mean – (1.65 × standard deviation)

As a result, a cut-off score of 63 and below indicates affectedness, and a cut-off score of 64 and above indicates no DCD. Martin et al. (2006) found that this method produced a more reliable prevalence estimate of 8% (as opposed to 2% when using the Canadian cut-offs) which is comparable to the prevalence rate reported in the DSM-IV of 6%.

The DCD-Q has sound reliability and validity with studies reporting good sensitivity and specificity (Crawford et al., 2001; Green et al., 2005). The DCD-Q has demonstrated high internal consistency of the items with reliabilities of .87 to .88 as measured by Cronbach's alpha (Wilson et al., 2000). These reliabilities are comparable to those reported by Martin et al. (2006). The DCD-Q has acceptable concurrent validity as it has been shown to significantly correlate with scores on the Movement Assessment Battery for Children ($r = -.59$), a standardized test designed to identify motor difficulties in children (Wilson et al., 2000). The present study also identified good internal reliability, with a Cronbach's alpha of .84.

Depressive Symptomatology ('Sad Affect')

The Twin and Sibling Questionnaire also includes 12 items relating to 'sad affect' which assess depressive symptomatology (Hartman et al., 2001). These items were taken from a larger questionnaire which includes the 'sad affect' construct among other childhood internalizing and externalizing problems (Hartman et al., 2001). The responses for the 12 items are rated on a 4-point scale and are totalled to produce a 'sad affect' score with a total possible score of 36, with higher scores indicating greater depressive symptoms. Research involving a twin sample from an earlier wave of the Australian Twin ADHD Project reported acceptable internal reliability for the 12-item 'sad

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Table 2

Mean Scores and Standard Deviations of Depressive Symptomatology for the ADHD-Only, DCD-Only, ADHD + DCD, and Control groups

Twin Samples	Group	*n*	*M*	*SD*
A	ADHD only	71	5.87	6.08
	DCD only	92	4.47	4.01
	ADHD + DCD	33	7.00	5.09
	Control	145	2.07	3.01
B	ADHD only	81	5.22	4.25
	DCD only	100	4.92	4.17
	ADHD + DCD	36	8.97	4.81
	Control	134	1.86	2.09

affect' scale with a Cronbach's alpha of .72 and an alpha for each item if deleted, ranging between .68 and .72 (Levy, Bennett, et al., 2005). In the current study, Cronbach's alpha was .77 which demonstrates acceptable internal reliability for the 'sad affect' scale.

Procedure

The current research was carried out as a part of a larger study (The Fourth Wave of the Australian Twin ADHD Project) investigating child and adolescent twins aged between 6 and 17 years and was approved by both the Curtin Human Research Ethics Committee and the Australian Twin Registry. The recruitment procedure used by the Australian Twin ADHD Project (ATAP) is described in Levy and Hay (2001) and Bennett et al. (2006). Following written consent, families were sent the Twin and Sibling Questionnaire which consisted of questions on ADHD, DCD and sad affect. Parents were also requested to provide information on current medication use which revealed that some of the participants comprising the groups were on medication at the time the questionnaires were completed. In this case, parents were asked to rate behavior when the child is off medication. Additionally, the questionnaire was used to ensure that the participants did not meet the exclusion criteria, namely a physical disability, chronic illness, or a medical condition affecting development (e.g., Down Syndrome). Those who met the exclusion criteria were not included in the final sample.

Data Analysis

To determine whether there was a statistically significant difference between the MZ co-twins, a one-tailed Wilcoxon signed rank test was conducted. This nonparametric test was a more suitable analysis as the skewed nature of depressive symptoms (Hankin et al., 2005) violated the stringent assumptions of the related samples *t* test.

To determine whether there was a statistically significant difference in depressive symptomatology between the ADHD + DCD, DCD-only, ADHD-only, and control groups, a Kruskal-Wallis test was used, as opposed to ANOVA. This nonparametric test was used due to the skewed nature of depressive symptoms (Hankin et al., 2005). Planned comparisons (three Mann-Whitney U tests) were carried out in order to determine which specific groups were statistically significantly different.

Results

Co-Twin Comparisons

For the 16 ADHD-only twins, the mean score for depressive symptomatology was 6.75 (*SD* = 6.18), and for their co-twins, the mean was 4.31 (*SD* = 5.12). For the 24 DCD-only twins, the mean score for depressive symptomatology was 5.21 (*SD* = 4.44), and for the co-twins the mean was 3.75 (*SD* = 3.73). A statistically significant difference was found between the ADHD-only and co-twins, $Z = -2.16$, $p = .016$, and between the DCD-only and control twins, $Z = -2.83$, $p = .003$, indicating that the twin with ADHD or DCD demonstrated higher levels of depressive symptomatology compared to their non-ADHD or non-DCD co-twin.

Full Sample comparisons

The mean scores and standard deviations of depressive symptomatology for the ADHD-only, DCD-only, ADHD + DCD, and control groups are presented in Table 2.

The relationship between group membership and depressive symptomatology was found to be statistically significant for both the Twin A sample, χ^2 (3, N = 341) = 65.28, $p < .001$, and Twin B sample, χ^2 (3, N = 351) = 94.26, $p < .001$. Planned comparisons (Mann-Whitney) between the DCD-only and ADHD + DCD groups were found to be significant for both the Twin A sample, $z = -2.70$, $p = .004$, and Twin B sample, $z = -4.53$, $p < .001$, indicating that the ADHD + DCD groups had higher levels of depressive symptomatology than the DCD-only groups. The comparisons between the ADHD-only and ADHD + DCD groups were found to be statistically significant for Twin A sample, $z = -1.65$, $p = .05$, and Twin B sample, $z = -3.98$, $p < .001$. This indicates that the ADHD + DCD group demonstrated higher levels of depressive symptomatology compared to the ADHD-only group. Finally, the comparisons between the ADHD + DCD and control groups were found to be statistically significant for both the Twin A sample, $z = -5.85$, $p < .001$, and Twin B sample, $z = -7.76$, $p < .001$, indicating that the ADHD +DCD group demonstrated higher levels of depressive symptomatology than the control group.

Pearson's correlations revealed that the variables of sex and age did not significantly correlate with the 'sad affect' scores for any of the groups in the Twin A sample. In the Twin B sample there was a weak relationship between age and sad affect for the Twin B DCD-only group ($r = .22$, $p < .05$) , and a moderate relationship between age and sad affect for the Twin B ADHD + DCD group ($r = .59$, $p < .01$). However, given that the results were identical for both Twin A and Twin

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B samples, these relationships for the Twin B sample did not appear to have influenced the findings.

Discussion

The MZ-differences design provides a powerful tool to identify nongenetic risk factors for depression. In the current study, twins with ADHD demonstrated higher levels of depressive symptomatology compared to their non-ADHD co-twins. These findings concur with previous studies which have also indicated that children and adolescents with ADHD are more likely to experience increased levels of depressive symptoms compared to controls without ADHD (Kitchens et al., 1999; LeBlanc & Morin, 2004). However, this study extends from these findings as the significant differences between the twins can be attributed to the effects of unique environmental factors (Phelps et al., 1997).

Previous research suggests that children and adolescents with ADHD experience a number of social, emotional, and behavioral difficulties such as relationship difficulties, school failure, and low self-esteem (Slomkowski et al., 1995). Authors have argued that depression could represent a secondary disorder to ADHD due to the difficulties that the children face (Jensen et al., 1993; Schmidt et al., 1998). Consequently, it is conceivable that the twins with ADHD experience higher levels of depressive symptomatology compared to their co-twins without ADHD because of unique environmental experiences such as academic, behavioral, and social difficulties. It has been noted, however, that estimates of nonshared environmental influences also involve measurement error (Plomin et al., 2001). Consequently, this can also make co-twins differ. However, this would affect the error variance as much as the means and thus cannot be the explanation of consistent co-twin differences. Furthermore, the cross-sectional nature of this study cannot ascertain whether the twins manifested higher levels of depressive symptomatology before or after the ADHD.

A difference in depressive symptomatology was also found between the twins discordant for DCD, indicating that the twins with DCD demonstrated significantly higher levels of depressive symptomatology compared to their co-twins without DCD. These results support the findings of Francis and Piek (2003) who also found increased levels of depressive symptomatology in young children with DCD compared to children without poor motor coordination. Furthermore, research has demonstrated a link between DCD and higher levels of anxiety (Sigurdsson et al., 2002; Skinner & Piek, 2001). This study extends from previous research as it involved a MZ differences design which controls for the effects of genes and shared environmental factors. Thus, the finding of a significant difference in the level of depressive symptomatology between the twins can be attributed to the effects of unique environmental factors. Children and adolescents with DCD experience a number of psychosocial problems such as poor self-perceptions, academic underachievement, perceived lower levels of social support, and negative peer relations (Cratty, 1994; Gillberg et al., 1983; Losse et al., 1991; Skinner & Piek, 2001). It has been argued that the motor problems and the associated psychosocial implications experienced by children with DCD, predispose them to many of the risk factors for depression (Francis & Piek, 2003). Consequently, it is plausible that the twins with DCD experience higher levels of depressive symptomatology compared to their non-DCD co-twins due to unique environmental experiences such as negative social feedback and academic difficulties.

The final analysis in this study indicated higher levels of depressive symptomatology in children and adolescents with comorbid ADHD and DCD compared to those with ADHD only, DCD only, and no ADHD or DCD. Research assessing the prognosis of individuals with comorbid ADHD and DCD has suggested a poor outcome. For example, children and adolescents with the comorbid condition are at greater risk for negative long-term outcomes such as psychiatric disorders, school dysfunction, personality disorders, and neurodevelopmental problems compared to individuals without ADHD or DCD (Hellgren et al., 1994). Depression is one of the main psychiatric problems that appears to be more common in children with DAMP compared to children without DAMP (Hellgren et al., 1994). Consequently, the results of this study support research suggesting a poorer emotional functioning in children and adolescents with comorbid ADHD and DCD. Furthermore, the findings of this study extend from those of previous studies as the comorbid group was also compared to an ADHD-only and DCD-only group. Previous studies have compared a comorbid group to an ADHD-only or DCD-only group on outcomes such as school dysfunction (Kadesjo & Gillberg, 1999, 2001). However, these studies have not addressed the emotional functioning of these individuals. Consequently, this study provides further support for the DAMP model and the associated poorer emotional functioning demonstrated in individuals with the comorbid condition. It should be noted that the control groups were older and included more girls than boys, in contrast to the affected groups. Given that research demonstrates that older girls are more likely to have higher levels of depression (e.g., Angold et al., 1998), these demographic differences may result in an underestimation of the depressive symptoms of the affected groups.

Hellgren et al. (1994) suggest that the high rate of associated depression in individuals with DAMP may be the result of 'biological/ genetic factors that predispose to/show as attention problems and motor clumsiness ... and major depression' (p. 1268). Alternatively, individuals with comorbid ADHD and DCD may experience rejection by peers, teachers, and

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relatives, which may ultimately result in feelings of unhappiness, isolation, and consequently, depression (Hellgren et al., 1994). Furthermore, it is possible that the combination of ADHD and DCD complicates the symptoms experienced by the individual, increasing the risk of psychiatric, educational, and other problems. As a result, they may experience increased levels of depression. The findings of increased levels of depressive symptomatology in MZ twins with ADHD or DCD compared to their co-twins without ADHD or DCD provides indirect evidence that the association between higher levels of depressive symptomatology and comorbid ADHD and DCD is not entirely due to common genetic factors.

There are various limitations that should be taken into account when considering the results of this study. Firstly, ADHD and DCD symptoms were assessed using parent-rated questionnaire measures, which do not produce a 'true' DSM-IV clinical diagnosis. However, these screening measures for ADHD and DCD have produced comparable prevalence rates to those reported in the DSM-IV manual (Martin et al., 2006). The use of parent-rated measures also introduces the issue of parental rating biases and contrast effects. The issue of reliability of parental reports when examining trait symptoms in MZ and DZ twins introduces a bias referred to as 'rater contrast'. This occurs when parents try to make their twins appear more similar (as is the case for MZ twins) or more different (for DZ twins) than they really are, based on their knowledge of the twin's zygosity rather than on their actual behavior (Levy, Hay et al., 2005; Rice et al., 2002). Consequently, this bias may influence the parental responses which may be less likely to occur with twins using self-report measures. Additionally, research has suggested that parents are less likely to report the presence of internalizing symptoms such as depression compared to externalizing problems (Howells Wrobel & Lachar, 1998). Despite these issues, differences in depressive symptomatology were identified between twin pairs in the current study, suggesting that these factors did not influence the findings. Furthermore, the present study involved a cross-sectional design and is therefore unable to specify whether the twins manifested higher levels of depressive symptomatology before or after the ADHD or DCD. It is also possible that certain environmental experiences or life events, unrelated to DCD, may have contributed to the differences in depressive symptomatology between the twins (e.g., stressful life events). Future research should implement a longitudinal design in order to investigate the direction of the relationship between ADHD, DCD, and depressive symptoms.

Conclusions

The results from the study indicate increased levels of depressive symptomatology in MZ twins with ADHD or DCD compared to their co-twin without ADHD or DCD. Furthermore, the results revealed a higher level of depressive symptomatology in children and adolescents with comorbid ADHD and DCD compared to those with ADHD only, DCD only, or no ADHD or DCD. The MZ differences design enables the differences in depressive symptomatology between the discordant twins to be attributed to unique environmental influences. Consequently, it is plausible that the twins with ADHD or DCD face unique environmental experiences such as negative self-perceptions, poor relationships with peers, behavioral problems, negative social feedback, and academic underachievement, which may consequently predispose them to many of the risk factors for increased levels of depressive symptomatology.

The present findings suggest that children and adolescents with ADHD or DCD are more likely than controls to be experiencing higher levels of depressive symptomatology. Therefore, such research could have important implications for the evaluation and treatment of children and adolescents with ADHD or DCD. While the study did not specifically identify the source of the increased levels of depressive symptoms, it highlights the importance of not overlooking aspects of emotional functioning in the evaluation process for children and adolescents with ADHD or DCD. This is crucial as it may point to the need for addressing issues such as depressive symptoms in the treatment of these children and adolescents, in addition to managing the primary ADHD (i.e., inattention and hyperactivity/impulsivity) or DCD (i.e., motor coordination problems) symptoms.

The findings of increased levels of depressive symptoms in children and adolescents with comorbid ADHD and DCD compared to those with ADHD only, DCD only, or no ADHD or DCD also have important clinical implications. Given that ADHD and DCD co-occur at a rate of approximately 50% (Piek et al., 1999), these findings emphasise the importance of addressing psychosocial issues such as emotional problems in the evaluation and treatment of these children and adolescents. In addition to the poorer prognosis associated with comorbid ADHD and DCD, children and adolescents with the comorbid condition may respond differently to treatment compared to individuals with ADHD only or DCD only. This highlights the need for the assessment of motor coordination as a standard practice for children and adolescents with ADHD and vice versa. It is important to acknowledge and explore the various symptoms of each overlapping disorder, as well as associated emotional problems, in the assessment and intervention of these individuals.

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